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Review

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Review

The Metabolic Profile of Psoriatic Disease

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Abstract

Psoriatic disease is an autoinflammatory, autoimmune systemic disease characterized by the presence of psoriasis which may be accompanied by psoriatic arthritis (PsA). The disease is characterized by the presence of metabolic syndrome. Obesity, arterial hypertension, diabetes mellitus type 2, hyperlipidemia and fatty liver disease may be observed in patients with psoriatic disease. The pathophysiology of metabolic syndrome in patients with psoriatic disease has not been completely elucidated. However, hyperinsulinemia may be observed and may be implicated in the pathogenesis of systemic inflammation observed in psoriatic disease. All components of the metabolic syndrome in patients with psoriatic disease need treatment. Arterial hypertension should be managed, diabetes mellitus should be managed, and hyperlipidemia should be specifically therapeutically targeted. The presence of obesity may necessitate specific adaptation of the management of PsA such as adjustment of the dose of biologic agents applied for the management of the disease. Weight loss induced either by diet or surgically or via an incretin-based approach improves psoriasis. Weight loss may prevent progression of psoriasis to PsA. Weight loss may improve disease activity in PsA. Modern treatment modalities in the management of obesity hold a great promise in the treatment of psoriatic disease as they appear to have both metabolic as well as immune modulating effects. In this review the pathophysiology, management and therapeutic implications of metabolic syndrome in patients with psoriatic disease will be discussed.

Keywords: psoriatic disease; psoriatic arthritis; psoriasis; metabolic syndrome; obesity; hyperinsulinemia; GLP-1R agonists

1. Introduction

Psoriatic disease presents a disease characterized by the presence of psoriasis and psoriatic arthritis (PsA) [1]. Psoriasis and PsA may occur simultaneously, psoriasis may precede the development of PsA or PsA may present first [2–4]. However, psoriasis or PsA may occur independently. Psoriasis is characterized by the presence of an inflammatory skin eruption which takes various forms and affects various parts of the body [5,6]. PsA is an enthesopathy and belongs to the category of spondyloarthritis. It is characterized by the presence of enthesopathy and arthritis affecting the axial skeleton while in some cases it also affects peripheral joints and induces peripheral arthritis.

Psoriatic disease is frequently accompanied by metabolic syndrome [7,8]. Metabolic syndrome in the context of psoriatic disease is manifested by the presence of central obesity [9–11], diabetes mellitus type 2 (DM 2), arterial hypertension, hyperlipidemia and fatty liver disease. The pathophysiology of metabolic syndrome in the context of psoriatic disease has been extensively investigated. However, it has not been completely elucidated.

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All conditions related to the phenotype of the metabolic syndrome in psoriatic disease need management and successful treatment [12–14]. Obesity needs multifaceted prevention and treatment [9], DM 2 needs management [15], arterial hypertension needs treatment [16], and fatty liver disease needs prevention and treatment [17]. Obesity, in particular abdominal obesity is a central feature in the context of psoriatic disease. It may be involved in disease pathogenesis; it is involved in disease pathophysiology and may induce severe disease by modulating the disease phenotype. In this review the inflammatory pathways induced by obesity will be discussed. The interrelationship between obesity and psoriatic disease, both psoriasis and PsA will be reviewed. Treatment modalities to induce weight loss, hygienic, surgical and medical will be dealt with.

2. Psoriatic Disease

Psoriatic disease is characterized by the presence of PsA and psoriasis [18]. The two entities may occur simultaneously, or the one may precede the other [19]. Psoriasis may occur first and PsA may present later [4]. Approximately one third of patients with psoriasis will proceed to develop PsA.

PsA is an autoinflammatory disease. It is characterized by the presence of enthesitis. The axial skeleton is usually affected but peripheral arthritis may also occur [20–22]. The disease is also characterized by extraarticular manifestations. Enthesitis occurs, the axial and peripheral skeleton is affected with arthritis. However, as inflammation is central in the pathogenesis of psoriatic disease and psoriatic arthritis bone resorption is also observed and may lead to osteopenia or osteoporosis [23,24].

PsA is a chronic inflammatory disease which affects the nails and skin, is characterized by peripheral arthritis, enthesitis, dactylitis and involvement of the axial skeleton. These clinical manifestations may occur isolated or combined with each other. The prevalence of PsA globally ranges from 0.05% to 0.25% [25]. It has been reported that a percentage of up to 30% of patients with psoriasis will proceed to develop PsA [20]. Furthermore, PsA is heterogenous in nature and can have an array of symptoms and clinical manifestations, which poses a challenge in diagnosis and treatment [20]. More than 50% of patients with PsA may have axial involvement, i.e., involvement of the spine and the sacroiliac joints [26]. Only a small involvement of PsA patients have only axial disease, as most exhibit additional peripheral arthritis. Peripheral joint involvement may appear as polyarticular, mono or oligoarticular, distal or arthritis mutilans and includes dactylitis and enthesitis [27]. Although earlier reports described axial involvement mostly in male patients, recent reports describe axial involvement in female patients as well. The occurrence of HLA-B27 gene may be associated with severe phenotypes of PsA [28].

Psoriasis is a skin disease which is accompanied by inflammation, scaling, erythema, plaque formation and itching. Psoriasis is an immune-mediated inflammatory disorder characterized by activation of T helper lymphocytes, Th17 and Th22 lymphocytes and augmented expression and levels of inflammatory cytokines either systemically or locally within the lesions [29,30]. It affects various body areas and may take several forms. The disease manifests as scaling papules and plaques. The lesions are circumscribed, circular, red papules or plaques and have a grey or white dry scale. The lesions may affect the scalp, elbows, knees and body folds. The lesions may develop at sites of injury. If the disease is progressive, it may take the form of generalized exfoliative erythrodermic psoriasis [31]. Nails may be affected especially if PsA is present [32].

3. Metabolic Syndrome

Metabolic syndrome is characterized by the presence of central obesity, impaired glucose metabolism which may take the form impaired glucose tolerance or frank diabetes mellitus type 2, arterial hypertension and hyperlipidemia. Fatty liver disease may accompany the metabolic syndrome [33–35]. Metabolic syndrome was initially described by Reaven and was called syndrome X [36]. It was recognized that it was related to insulin resistance, i.e., to resistance to insulin mediated glucose uptake [37]. Metabolic syndrome is accompanied by an increased risk for the development

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of DM 2 and an enhanced risk for the development of cardiovascular disease. Food affluence as well as the prevalence of a lifestyle compatible with ample food intake and lack of physical activity led to increased prevalence of the metabolic syndrome in the western world, as well as in the developing countries [38]. The Third Report of the National Cholesterol Education Program Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (ATP III), the National Heart, Lung, and Blood Institute, and the American Heart Association have issued definitions of the metabolic syndrome [39–42]. Central or abdominal obesity is a dominant characteristic of the metabolic syndrome [43–45]. Metabolic syndrome is related to inflammation [46,47]. Central or abdominal obesity which is an inherent feature of metabolic syndrome is associated with systemic low-grade inflammation. C-reactive protein (CRP) levels and inflammatory cytokines increase [48–50]. In particular, the pro-inflammatory cytokines TNFα and interleukin-6 levels are known to be increased in obesity related to overnutrition. TNFα is a cytokine with multiple functions and is involved in the regulation of many biological processes such as immune function, proliferation and energy metabolism [51]. TNFα is produced by adipocytes as well as by various stromal cells found in adipose tissue, such as preadipocytes, endothelial cells and macrophages.

Psoriatic disease has been shown to be accompanied by metabolic syndrome [52] (Figure 1). Metabolic syndrome induces the development of an inflammatory milieu which render the organism susceptible to the development of may induce psoriatic disease [53–55]. Additionally, the presence of metabolic syndrome in the context of psoriatic disease may induce a severe phenotype [14] and may necessitate modifications in the treatment of the disease.

PsA may be accompanied by metabolic syndrome in a percentage of 29% to 46%, which is higher than the prevalence of the metabolic syndrome in the general population which is estimated to be between 12.5% to 31.4% [56–58]. Many of the comorbid situations in PsA are related to the metabolic syndrome. These include obesity, hypertension, DM 2 and hyperlipidemia. Gupta et al. [59] reviewed the comorbidities of PsA in a meta-analysis of 39 studies. They observed DM 2 in 12,9%, arterial hypertension in 34.2%, hyperlipidemia in 24.2%, metabolic syndrome in 28.8%, obesity in 27.4% and any cardiovascular disease in 19.4%. The review by Gupta et al. [59] combined data from more than a hundred thousand patients suffering from PsA and indicated that 1 in 3 patients had arterial hypertension, 3 in 10 had metabolic syndrome and 1 in 4 patients had obesity. According to this review all comorbidities were commoner in PsA patients than in controls. These comorbidities were found to be associated with poorer quality of life, impaired function and discontinuation of treatment with TNF inhibitors.

Psoriasis is also related to the presence of metabolic syndrome [60]. Psoriasis is associated with metabolic syndrome and its characteristics, such as abdominal obesity, arterial hypertension, hyperlipidemia, DM 2 as well as non - alcoholic fatty liver disease. Several studies have indicated that the prevalence of metabolic syndrome in psoriasis patients may be between 20% to 50%. The risk of developing metabolic syndrome is double in psoriasis as compared to control subjects. In a large systematic review Armstrong et al. [61] examined the relationship between psoriasis and metabolic syndrome which included 41,853 patients suffering from psoriasis and a large control population. They observed a strong relationship between psoriasis and the metabolic syndrome, the prevalence of metabolic syndrome in psoriasis in the range of 14% to 40%. They hypothesized that elevated free fatty acid levels may lead to adipocyte dysfunction, may inhibit insulin secretion and potentially lead to the development of DM 2 [62]. They hypothesized that elevated free fatty acid levels along with increased cytokines such as TNF-a and IL-6 lead to increased glucose production in the liver and reduced glucose uptake by the muscles leading to impaired glucose tolerance. The authors suggested that shared genetic risk loci, such as CDKAL1 may be associated with both psoriasis and type 2 diabetes [63,64]. The association of metabolic syndrome with psoriasis severity has been examined in the literature. It has been observed that psoriasis severity is related to the presence of the metabolic syndrome [65–67]. Additionally, the presence of the metabolic syndrome correlates with the risk of developing PsA in patients with psoriasis [68].

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In a review it has been shown that there exists an increased risk for cardiovascular diseases at the level of 43% as compared to a control population, the risk referring to myocardial infarction, with an elevated risk at the level of 68%, cerebrovascular disease at the level of 22% and heart failure at the level or 31% [69]. Increased disease activity and severity in PsA, as shown by markers such as polyarthritis, dactylitis and systemic inflammation were linked to a higher risk for cardiovascular events. Psoriasis, in particular severe disease is accompanied by an increased risk for coronary artery disease, stroke and subsequent mortality [70]. It appears that systemic inflammation present in psoriatic disease may contribute to the development of atherosclerosis, endothelial and vascular damage [16].

Fatty liver disease has also been observed in patients with psoriatic disease [17,71–73]. Common inflammatory pathways maybe involved in the pathophysiology of psoriatic disease and liver steatosis and indicate that both diseases should be simultaneously managed therapeutically.

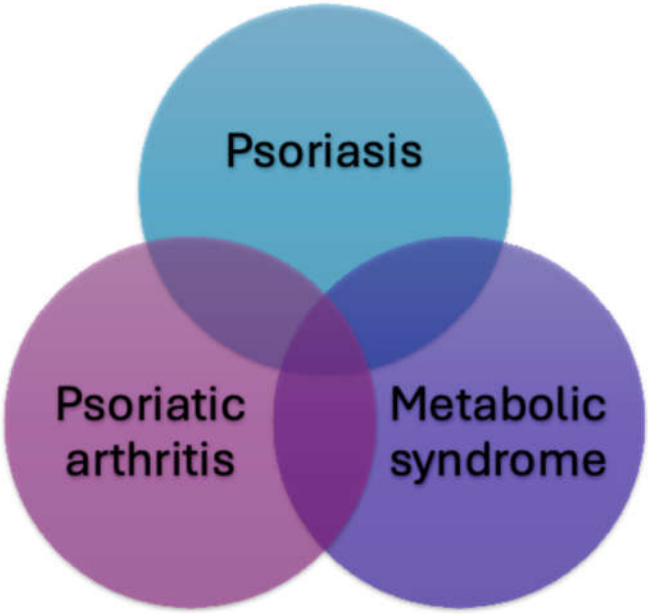

Figure 1. The intimate relationship between psoriasis, psoriatic arthritis and metabolic syndrome.

4. Obesity and Psoriatic Disease

Obesity accompanies psoriatic disease [68]. Obesity may be a risk factor for the development of psoriatic disease and it may induce a state of increased disease activity in patients with the disease.

Obesity is a chronic, progressive and relapsing disease which increases the risk for various disease states, including DM 2 and cardiovascular disease [74]. It represents a state of low inflammatory activity [75]. Low grade inflammation in the context of obesity is brought about by visceral adipose tissue [76–78]. Visceral adipose tissue releases inflammatory adipokines, such as leptin, TNF α, IL-6 and IL-18. Leptin induces monocyte and granulocyte activation and increases the number of M1 macrophages. Leptin also affects T lymphocyte differentiation, favoring Th1 CD4 cells (Treg) cells. Leptin increases the secretion of inflammatory cytokines, such as IL-6, IL-12, IL-17 and TNFα [79]. TNFα is an inflammatory cytokine implicated in the pathophysiology of obesity and psoriatic disease [80,81]. TNFα increases the proportion of M1 macrophages, creating a pro-inflammatory state. Various studies have shown that adipose tissue is an endocrine organ which secretes various adipocytes and stromal cells such as vascular cells and adipocytes. Macrophages, neutrophils, T cells, B cells and mast cells are detected within the adipose tissue [14]. In addition to adipocytes, adipose tissue is composed of stromal-vascular cells and leukocytes. Cytokines secreted by adipose tissue, generally named adipokines, are regulated by a complex network of genetic and

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environmental factors, and obesity is associated with an abundance of macrophages, neutrophils, T cells, B cells, and mast cells in adipose tissue [15].

Various studies clinical and translational have led to the development of the concept of metaflammation, i.e., inflammation related to metabolism derangement, which may be involved in the pathogenesis and clinical expression of PsA [48,82]. Additionally, it has been proposed that a positive feedback loop may exist between obesity induced inflammation and psoriatic disease, which is known as the "psoriatic march" [83,84].

It is also possible that obesity in PsA induces inflammation due to mechanical reasons exerted at the level of the enthesis. Enthesitis is considered as the hallmark of PsA [85]. Biomechanical stress induced by obesity as a mechanical load may be a factor responsible for enthesitis [86].

The role of intra-articular fat pads, which may play a mechanical as well as an immune role should also be noted. In particular, the infrapatellar fat pad in the knee may act as a shock absorber but may also secrete adipokines and cytokines to induce local inflammation, as has been shown in osteoarthritis. Intra-articular fat pads, such as the infrapatellar fat pad in the knee, have dual mechanical and immunological roles. In addition to functioning as shock absorbers in joints, they secrete adipokines and cytokines that modulate local inflammation [87,88] and induce development of a systemic inflammatory disease, such as PsA.

The accumulation of adipose tissue also acts to induce and augment pain. Patients with PsA and obesity experience higher than expected levels of pain and physical disability compared to those without obesity [89,90].

It appears that adipose tissue accumulation in the context of abdominal obesity may lead to the development of an inflammatory environment and may induce the development of psoriatic disease, psoriasis and PsA. Obesity acts via various pathways, it induces an inflammatory phenotype by the augmented production of adipokines and inflammatory cytokines by the adipose tissue, it may induce metaflammation in the context of an autoinflammatory disease, it may act mechanically by inducing enthesitis by the sheer effect of weight on the entheses, it may act locally in the intra-articular fat pad and it may act to induce or augment pain. Thus, it appears that obesity and inflammation in the context of psoriatic disease create a vicious cycle, which may induce a severe phenotype of the psoriatic disease by augmenting both inflammation and pain.

5. Treatment

All aspects of metabolic syndrome need management in the context of psoriatic disease. Obesity needs prevention and treatment [11,91,92]. Diabetes mellitus type 2 needs management, arterial hypertension needs management and hyperlipidaemia needs treatment.

Obesity needs prevention and treatment in the context of psoriatic disease. Hygienic measures such as diet and physical exercise should be recommended to all patients with psoriatic disease. However, compliance with diet and physical exercise can be challenging, it may require time and effort, intensive interventions and only around half of the patients achieve their goals [93,94]. Upala et al. [95] in a meta-analysis of randomized controlled trials which assessed lifestyle interventions by diet and or exercise in obese patients with psoriasis on disease severity and included seven randomized control trials with a total of 878 patients, observed a greater reduction in the Psoriasis Area Severity Index (PASI) score in patients on weight loss intervention than in controls. They also observed that patients receiving lifestyle weight loss interventions were more likely to achieve a PASI75 score. Even only adherence to a diet and exercise plan, without achieving the goal of weight loss leads to a reduction in PASI score in patients with psoriasis [96,97]. Ramadan fasting also improved PASI score in patients suffering from moderate to severe psoriasis [98].

Diet which leads to weight loss may also have beneficial effects in the treatment of PsA. Hypocaloric diet in individuals with obesity and PsA had as an effect weight loss and improvement in disease activity as shown by improvement in active enthesitis and joint counts, reduced inflammatory markers, improved skin lesions, physical function, perception of health and fatigue [99]. These effects were sustained for a period of two years [100]. The effect of diet either hypocaloric

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or free in patients with PsA who were obese and on treatment with TNF inhibitors was assessed in a study and it was found to improve disease activity. The effect of various diet modalities on psoriasis and PsA was evaluated in a study [101]. A total of twenty-six individuals with PsA and psoriasis were assigned either to a Mediterranean diet or to a ketogenic diet. Both diet forms resulted in significant weight loss, decreased waist circumference, BMI, total fat mass and visceral fat. Ketogenic diet led to a significant improvement in PASI and disease activity index in PsA. A study evaluated diet interventions, a Mediterranean diet, a low calorie diet or a general diet in patients with PsA and obesity. Modest weight loss and improvement in disease activity was noted in all groups. A very low-calorie ketogenic diet also improved BMI and disease activity score in patients with PsA [102]. BMI reduction correlated with the improvement in disease activity score. The practice of intermittent fasting in the context of Ramadan also led to improvement in disease outcome in patients with PsA despite lack or limited weight loss [103]. All these studies show that weight loss, regardless of the diet modality itself, lead to improvement in disease activity. It also appears that weight loss in the context of psoriatic disease may improve cardiovascular outcomes [104].

Bariatric surgery is another method applied to induce weight loss in patients with psoriatic disease. Various methods of bariatric surgery exist. In various case studies complete remission of psoriasis has been reported after gastric bypass surgery with the Roux-en-Y method [105–108]. In a study on the effect of weight loss bariatric surgery on psoriasis severity in morbidly obese patients was investigated [109]. The study involved 33 morbidly obese patients with psoriasis. They found improvement in more than 40% of the cohort after bariatric surgery. Improvement was related to the degree of weight loss and the Roux-en-Y procedure. In a small study involving patients with psoriatic disease, outcomes regarding skin and joint disease improved after bariatric surgery. This effect was greater in patients with severe disease. Data from a Swedish study involving obese Swedish patients indicated that bariatric surgery reduced the incidence of psoriasis but did not have an effect on PsA incidence [110]. This study confirmed a role of bariatric surgery in the prevention of psoriasis in obese patients. In a study performed in Denmark the risk of psoriasis was reduced after gastric bypass, but not after gastric banding, whereas both gastric bypass and banding decreased the incidence of PsA [111].

Nowadays methods of drug treatment for obesity exist, including the use of glucagon-like peptide 1 receptor agonists (GLP-1RA) [112,113]. GLP-1RA are a novel and important treatment modality in the management of both obesity and DM 2 [114,115]. GLP-1 RA act both centrally by modulating brain areas which control appetite and hunger [116,117] and peripherally by increasing insulin secretion [118], reducing glucagon secretion and by modulating gastric emptying and the release of gut hormones [119]. GLP-1 RA reduce triglyceride levels and cholesterol, such as low-density lipoprotein. Additionally, they modulate inflammation of adipose tissue and reduce ectopic fat deposits. In recent years the application of GLP-1RA has increased considerably and it has emerged that they are effective in inducing weigh loss [120] and in preventing and managing obesity comorbities such as cardiovascular events and DM 2 [121,122]. GLP-1RA have been applied in the treatment of musculoskeletal diseases such as osteoarthritis [123] and have shown beneficial effects. In particular, the use of GLP-1RA exhibits anti-inflammatory properties. In a mouse model of a neurodenerative disorder, namely Alzheimer's disease liraglutide was shown to have anti-inflammatory and neuroprotective properties [124]. In a prospective trial involving 24 patients exenatide was injected twice daily to the group. Fasting blood glucose, HbA1c, free fatty acid levels decreased [125]. Although weight loss was not observed, reactive oxygen species and the mRNA expression of inflammatory cytokines, such as TNFa, interleukin-1β in mononuclear cells decreased significantly. Exenatide was shown to have a potent anti-inflammatory effect irrespective of weight loss. In a mouse model of arterial hypertension liraglutide was found to reduce immune cellular infiltration of heart tissues by decreasing the secretion of inflammatory cytokines such as TNF and IL-1β [126].

The role of GLP-1 in the physiology of the joint as an organ is currently evaluated. It has been shown that GLP-1 is detected in various tissues within the joints. GLP-1R has been detected in both

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normal and osteoarthritic chondrocytes. The GLP-1R pathway may be involved in the prevention of chondrocyte apoptosis, may have protective properties against inflammation and may be involved in matrix protection against degeneration [127]. In a model of knee osteoarthritis in the rat liraglutide was found to attenuate cartilage degeneration [128]. Liraglutide was found to protect chondrocytes against cytokine induced apoptosis [128]. The protective effect of liraglutide on chondrocytes has also been observed in an experimental rat model [127]. In a rat model of inflammatory osteoarthritis the activation of GLP-1R reduced inflammation in cartilage [129]. GLP-1R seems to be expressed in human and murine macrophages leading to the hypothesis that it may have a modulating effect on macrophage function and differentiation [130] and that incretin or GLP-1 related treatment modalities may modulate macrophage function. Novel data indicate that GLP-1-based treatment modalities may have anti-inflammatory properties with anti-inflammatory effects in various tissues including the vascular system mediated by decreasing levels of inflammatory cytokines as well as by attenuating immune cell tissue infiltration [131,132]. Additionally, it has been proposed that GLP-1R agonists may have analgesic effects [131]. The therapeutic potential of GLP-1R agonists in rheumatoid arthritis has also been explored [133,134].

Weight loss induced by incretin-based treatments has been shown to improve psoriasis. In particular, Ahern et al. [135] examined the effect of the administration of liraglutide, a GLP-1 analogue to a group of 7 patients with psoriasis, diabetes and obesity and they found a beneficial effect on psoriasis, as they observed a significant decrease in PASI and a significant increase in the number of circulating natural killer T cells. Weight and glycemic control also improved. Hogan et al. [136] investigated the effect of GLP-1 on immune pathways and they proposed that metabolic incretin-based drugs may be repurposed and applied as immunomodulatory agents [137]. It has been proposed that GLP-1R agonists may be applied in the treatment of systemic autoimmune and autoinflammatory disorders such as PsA [138–140]. It should be noted that in patients with psoriatic disease the administration of GLP-1R agonists may improve cardiovascular outcomes, as these agents improve cardiovascular health [121,141,142]. Now the application of GLP-1R agonists on PsA is evaluated in combination with ixekizumab and is compared to dietary counseling [138]. This combination of ixekizumab with incretin-based treatment modalities is also evaluated in patients with psoriasis [138]. The application of sodium-glucose cotransporter-2 inhibitors in the context of diabetes as far as the effect on psoriasis incidence has been evaluated with negative results [143]. Other agents, such as orlistat and topiramate in the treatment of obesity have been evaluated [144]. Topiramate was found to induce weight loss in a small group of psoriasis patients with psychiatric morbidity [145]. In this context topiramate was found to improve psoriasis.

It appears, that obesity, in particular abdominal obesity, is a dominant characteristic in patients with psoriatic disease, psoriasis and PsA. In these patients abdominal obesity is related to a severe disease phenotype in psoriasis and augemented disease activity in PsA. Obesity is related to cardiovascular morbidity in psoriatic disease. The inflammatory environment related to obesity may induce the development of psoriasis or it may induce PsA in patients with psoriasis. Weight loss induced by any method, either modification of behavior, exercise, any kind of diet or surgery improves the disease phenotype in psoriasis and PsA (Figure 2). The application of incretin-based treatment modalities may hold plural roles in the treatment of psoriatic disease as it induces weight loss, improves glycemic control, improves cardiovascular risk and may have immune modulating properties.

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Figure 2. Treatment methods for obesity in the context of psoriatic disease.

6. Conclusions

It appears that psoriatic disease, psoriasis and PsA are strongly related to metabolic dysfunction, namely the metabolic syndrome. Patients present with obesity, DM 2, arterial hypertension, dyslipidemia and fatty liver disease. The presence of the metabolic syndrome induces an inflammatory milieu which may predispose to the development of psoriasis and PsA. Obesity is related to a severe disease phenotype in patients with psoriasis and to disease severity in patients with PsA. Additionally, drug treatment with biological agents may need to be modified in patients with PsA and obesity. Patients presenting with psoriatic disease and obesity should be offered advice and treatment for obesity and the other metabolic syndrome parameters on presentation. Weight loss should be advised. Patients should be offered advice on diet and exercise. If diet and other hygienic measures prove ineffective treatment with incretin-based agents should be initiated. Weight loss induced by any method induces improvement in psoriasis and PsA. The application of novel incretin-based treatment modalities in patients with psoriatic disease holds a great promise, as they appear to have both metabolic and immune modifying effects.

Author Contributions: For research articles with several authors, a short paragraph specifying their individual contributions must be provided. The following statements should be used "Conceptualization, L.A and P.A.; methodology, L.A, P.A., G.K., C.S. Y.S.; software, L.A, P.A., G.K., C.S. Y.S.; validation, L.A, P.A., G.K., C.S. Y.S. and I.K.A.; formal analysis, L.A, P.A., G.K., C.S. Y.S. and I.K.A. investigation, L.A, P.A., G.K., C.S. Y.S. and I.K.A.; resources, L.A, P.A., G.K., C.S. Y.S. and I.K.A.; data curation, L.A, P.A., G.K., C.S. Y.S. and I.K.A.; writing—original draft preparation, L.A, P.A., G.K., C.S. Y.S. and I.K.A.; writing—review and editing, L.A, P.A., G.K., C.S. Y.S. and I.K.A.; visualization, L.A., P.A.; supervision, P.A; project administration, L.A. and P.A; funding acquisition, L.A, P.A., G.K., C.S. Y.S. and I.K.A. All authors have read and agreed to the published version of the manuscript.

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