CLINICAL VIGNETTE

Psoriasis: Medications and Other Environmental Factors

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Case Report

The patient is a 64-year-old male with a history of psoriasis, hyperlipidemia, hypertension, and type-2 diabetes, who presented to the office with worsening psoriasis over the past week. His back and knees have been more swollen and painful and his psoriatic rash worsened on the extensor surfaces of his elbows and knees and trunk. He had eaten more seafood lately to reduce his saturated fat intake and also increased his omega-3 fatty acids. Two weeks ago he went to urgent care with an upper respiratory infection and was treated with azithromycin. His cardiologist also increased his dose of carvedilol from 3.125 mg to 6.25 mg twice daily last month.

Past medical history also includes osteoarthritis, GERD, spinal stenosis and asthma. Medications include etanercept, Metformin, Atorvastatin, Pantoprazole, Losartan, Tamsulosin, Ethacrynic acid, Carvedilol, and Naprosyn. Allergies include Penicillin, Sulfa-containing antibiotics, and systemic steroids.

He is a non-smoker who has 1-2 drinks per week. His alcohol intake increased lately due to work stress. He exercises regularly using an elliptical trainer and spends most of his time indoors. Family history is negative for psoriasis, and remarkable for parents with hypertension, and a sister with type-2 diabetes.

Physical examination revealed a height of 5 foot 10 inches, weight 217 pounds, BMI of 31, blood pressure 126/86, pulse 64 and normal temperature. His examination was normal except for an impressive scaly erythematous rash on the back and extensor surfaces of his extremities and swollen, inflamed knees.

Labs included normal CBC, chemistries, ESR, thyroid, creatine kinase, and lipid panel.

Psoriasis is a chronic multisystem autoimmune disease that typically presents with dermatologic and/or rheumatologic manifestations[1]. Psoriasis is very common, affecting 2-4% of the population in the United States[2]. A large percentage of patients with psoriasis have other disorders such as type-2 diabetes, hypertension, and hyperlipidemia and take multiple prescription medications[3]. In some studies, 25% of patients with psoriasis were taking three or more prescription medications[4]. Although no definitive causal relationship has been established, evidence suggests certain medications, smoking, bacterial and viral infections, alcohol consumption, obesity, trauma, stress, and other environmental exposures can contribute to psoriasis[5]. One study reported approximately 83% of psoriatic exacerbations were related to medications[6].

Environmental exposures impact the expression of psoriasis several ways: exacerbating currently active psoriasis; triggering previously unexpressed psoriasis; or causing a drug-eruption mimicking psoriasis[7]. These manifestations can present with all of the various dermatologic forms of psoriasis including the less common pustular psoriasis[8]. In addition to the nails, non-dermatologic systems can also be affected including the joints[7]. Environmental factors may affect psoriasis through both immunologic and non-immunologic mechanisms which are complex in nature and appear to be mediated by t-cells and interleukins in the dermis[8]. In addition, these factors can trigger a new psoriatic reaction or aggravate a pre-existing psoriasis[9]. Medications are the common triggers including beta-blockers, antibiotics, and nonsteroidal anti-inflammatory drugs (NSAIDs) and diet is also a common trigger[10].

Beta blockers are very commonly prescribed and can worsen psoriasis[8]. Psoriatic rashes are the most common dermatologic side-effect of beta blockers and are commonly in patients with no personal or family history of psoriasis[11]. Topical beta blockers, including those for glaucoma, have also been associated[12]. Dermatologic manifestations typically begin as early as 1 month after initial treatment and can arise as late as 2 years into therapy[13]. Skin manifestations tend to resolve or significantly improve within days to weeks after discontinuation of the medication and recur within days of re-

challenge[11]. Some psoriatic rashes associated with beta blockers do not completely resolve[14]. The pathophysiology of beta blocker induced psoriasis is complex and not fully elucidated but appears to involve cyclic adenosine monophosphate (cAMP), lymphocyte transformation, intracellular protein metabolism, intracellular calcium changes, macrophage abnormalities, and abnormal neutrophil function[4]. The management of beta blocker induced psoriatic rashes involves withdrawing the medication if possible and initiating or adjusting conventional psoriatic treatments[15].

Antibiotics are also commonly associated with psoriasis. The most commonly associated class is the tetracycline family although macrolides and penicillin have also been linked[16]. Pencillins have been noted to trigger the pustular variant of psoriasis[17]. Those with a personal or family history as well as those with certain HLA-genotypes such as HLA-B13, B17, and B27 appear to be at the greatest risk of this sort of reaction.[4]

NSAIDs are also commonly associated with dermatologic manifestations of psoriasis[5]. Naproxen has been the most commonly reported and both orally and topically administered NSAID's have been associated[18]. NSAID-associated psoriatic rashes tend to occur fairly quickly after initiating therapy frequently within days to weeks.4 As with other types of drug-induced dermatologic processes, most cases resolve or dramatically improve shortly after discontinuing the offending agent[19].

Several hundred other medications have been associated with exacerbating or causing psoriatic rashes[7]. These include lithium, ace-inhibitors, anti-malarials, interferons, benzodiazepines, gemfibrozil, cimetidine, amiodarone, fluoxetine, and digoxin[18]. A personal or family history of psoriasis and age over 50 both increase the risk of cutaneous reactions[19]. Careful consideration of withdrawal of the potential offending agent is sometimes necessary to investigate a true cause and effect relationship[20].

Dietary factors also can trigger psoriatic reactions[2]. For example, iodine contained in intravenous contrast or foods have both been associated with psoriasis and bullous pemphigoid[21]. Shellfish intake has been suggested as a possible trigger for psoriatic rashes[22]. Additionally, hypovitaminosis D has been associated with vitiligo and psoriasis[23]. Dietary supplementation with omega-3 fatty acids has been associated with clinical improvement in the dermatologic manifestations of psoriasis[24]. Various supplements have been tested to see how they impact psoriasis with mixed results[25]. Hypocalcaemia appears to be significant risk factor for psoriatic exacerbations[26]. and alcohol consumption increases the risk of exacerbations in several studies[27].

A detailed medical history suggested multiple possible triggers for this patient's psoriatic exacerbation. They included his recent use of an antibiotic (azithromycin), the recent increase in his beta blocker dose, increased intake of alcohol, and his recent consumption of iodine-containing seafood. Increased Carvedilol was important so it was not adjusted. We recommended minimizing intake of iodine-containing foods and alcohol. He remained off of antibiotics in the subsequent weeks and within two months his psoriasis had dramatically subsided and was back under optimal control, without adjusting his psoriasis regimen.

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