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The Triumph of JAK Inhibitors for the Treatment of Alopecia Areata

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ABSTRACT

Alopecia areata (AA), with a disease incidence of 2% in the USA, is the most common immune-mediated cause of hair loss in the world. There were no FDA approved treatments for AA in the past. All treatment modalities used alone or in combination with each other, were prescribed off label. The JAK inhibitor baricitinib was approved by the FDA in June 2022. While it's a significant leap forward, we are still missing the safety profile data in multiple areas. Topical JAK inhibitors are being tested along with sublingual options including tofacitinib, ruxolitinib, and ritlecitinib. In this review, we discuss the JAK inhibitor baricitinib in detail, along with its dosing regimen, challenges presented in the clinic, missing data, and what the future may hold.

Keywords: alopecia areata, baricitinib, FDA approved, JAK inhibitors

INTRODUCTION

Alopecia areata (AA) is the most common immune mediated cause of hair loss in the world.[1,2] The disease incidence in the USA and United Kingdom is approximately 2%.[3] AA affects both children and adults and does not show a preference for gender, skin type, hair type, or hair color.[2] AA is characterized by a circular well-circumscribed area of hair loss, typically on the scalp or face, but it can affect any hair-bearing region.[1,2] It can range in severity from patchy, diffuse involvement to more severe forms involving the entire scalp or all body hair. AA can be a self-limiting condition that often results in spontaneous regrowth, but as an autoimmune disease, disease extent and activity can vary and patients can experience recurrent episodes of patchy or extensive hair loss.[1,3,4] While the disease mechanisms driving AA have not been fully determined, it is clear that genetic predisposition and autoimmunity are key contributors together with likely environmental factors.[3] Genetic epidemiology has demonstrated an increased risk of AA in first degree relatives, with the incidence being between 10% and 50%.[1,3,5] This genetic predisposition has been linked to the major histocompatibility complex (MHC) gene and more specifically to the human leukocyte antigen (HLA) class II genes.[1,3,5] Many single nucleotide polymorphisms (SNPs) associated with AA led to the identification of candidate susceptibility genes through genome-wide association studies (GWASs).[1,3,5] Previous studies showed that AA largely involves attack of the hair follicle bulb region by autoreactive CD8+ T-cells, which comprise the characteristic "swarm of bees" around the hair follicle bulb in AA. These autoreactive T-cells are initially activated by numerous cytokine pathways that utilize the Janus kinase (JAK)-signal transducer and activator of transcription (STAT) signaling.[1-4] Treatment of AA has been challenging in the past due to an inadequate understanding of the pathogenesis of the disease.[6]

Past and Current Treatments for AA

Current treatment strategies for patients with AA differ based on several factors including disease severity, affected area(s), and patient age. Broadly, these can be categorized into three groups: topical, intra-lesional, and systemic therapies.

Traditional first-line therapies for patients with limited AA include topical (TCs) and intralesional corticosteroids (ICs). For both children and adults, use of higher potency TCs is preferred as they have

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