SIR,

The term alopecia, used by physicians dating back to Hippocrates, originates from the Greek word for fox, “alopex,” and was so-named due to fur loss seen in fox mange. “Areata” is derived from the Latin word, “area,” meaning a vacant space or patch.

The first use of the phrase alopecia areata is attributed to Polish physician John Jonston (1603–1675) in his book “Medicina Practica,” written in 1664. The term alopecia areata (AA) was introduced by French physician Sauvages de Lacroix (1706–1767) in “Nosologia Methodica,” published in 1763.[1] Sauvages classified alopecia into areata, simplex, syphilitica, and volatilium. Rather than a specific disease, AA was used as a descriptive term for patchy hair loss caused by various conditions including ringworm and leprosy.

The original clinical description of AA comes from “A Practical Synopsis Of Cutaneous Disease” written in 1817, by Thomas Bateman (1778–1821), apprentice to renowned dermatologist, Robert Willan.[2] Bateman described “bald patches, mainly circular,” with hair regrowth that “is softer and lighter in color than before.” However, rather than AA, he named it “porrigo decalvans,” meaning depilating scalp disease. He recommended treatment with oil of mace.

With the popularity of nosology in the 18th and 19th centuries, numerous terms to describe patchy scalp alopecia arose including area Celsi, area Jonston, AA, alopecia circumscripta, and Cazenave’s vitiligo. Confusingly, the terms were used interchangeably and often AA was not distinguished from tinea capitis. By the mid-1800s, these diseases had been teased apart and the term AA came to describe the condition familiar to us today.

Toward the end of the 19th century, debate moved on to etiology. Many physicians favored a parasitic cause based on reports of AA outbreaks among families, schoolchildren, and firemen.[3] This theory was eventually discarded after failed attempts to inoculate the disease. Another popular hypothesis considered AA to be a “neurotrophic” disorder, evidenced by a connection with emotional stress and experiments showing alopecia after nerve damage in cats. Other physicians pointed to the sudden hair loss and spontaneous recovery as signs of a transient causative toxin or nutritional deficiency in the blood. Eye strain and dental disease were also implicated as causative.

In 1929, French dermatologist and mycologist, Raymond Sabouraud (1864–1938), collated information from over 200 cases, noting positive family histories in 20% and strong associations with diseases, later understood to be autoimmune.[4] Subsequent histological studies, identifying peribulbar infiltrates, combined with the known positive effect of steroids, led to the proposal of an autoimmune cause in 1958. While AA was found to be associated with a higher incidence of antithyroid antibodies and antigastic parietal cell antibodies, identification of an autoantibody specific to AA remained elusive.

In recent years, genome-wide studies have identified genes that may underpin the immunologic changes seen in AA, such as the gene for UL16-binding protein, which attracts cytotoxic cells to the hair follicles.[5] These findings signify exciting steps forward in our understanding of this common but distressing condition, the classification and etiology of which has puzzled physicians for generations.

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