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Early Recognition And Successful Treatment Of Acquired Idiopathic Generalized Anhidrosis In A Deployed Servicemember

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Abbreviations:
AIGA: Acquired idiopathic generalized anhidrosis
WHEC: Warrior Heat and Exertion Related Collaboration
CHAMP: Consortium for Health and Military Performance

1. Introduction

Acquired Idiopathic Generalized Anhidrosis (AIGA) is a rare autoimmune condition impairing eccrine sweat gland function. Loss of sweating can be life-threatening in military populations exposed to extreme heat. Early recognition is critical, as AIGA is highly responsive to treatment. We present a case of AIGA in a deployed servicemember, underscoring the importance of awareness among military clinicians.

2. Case Report

A 31-year-old active-duty male stationed in Djibouti, Africa, presented with acute generalized anhidrosis. Symptoms began in the proximal upper extremities and progressed to near-complete body involvement, sparing only the axillae. As an avid runner, he immediately noted impaired heat tolerance during physical training. He was without any other neurologic or autonomic abnormalities. His inability to sweat posed immediate risk to mission readiness in a hot, austere environment.

The patient received a comprehensive work-up with Neurology that was unremarkable for any neurogenic, endocrine, metabolic or drug-induced etiologies of anhidrosis. The patient was then referred to Dermatology where 4mm punch biopsies of the skin on the left lower arm and left lower back both demonstrated a peri-eccrine inflammatory infiltrate. Immunostaining identified the inflammatory cells as mast cells and lymphocytes (Figures 1-4). These findings were most consistent with a diagnosis of AIGA. The patient was then treated with a prednisone taper (1mg/kg or 60 mg po daily) for one week followed by a decrease of 20 mg each subsequent week. His heat tolerance quickly improved, the ability to sweat returned by week 2 and he returned to a "fit for full duty" status. He was counseled regarding the possibility of recurrence and to monitor for signs and symptoms of heat related injuries.

3. Discussion

Current understanding of AIGA pathophysiology supports an autoimmune etiology targeting the cholinergic innervation of eccrine sweat glands [2, 3]. Kageyama et al. describe two potential pathological mechanisms: the "sweat allergy" type, involving IgE-mediated reactions to Malassezia fungi secreted in sweat leading to mast cell and basophil degranulation, and the "acetylcholine-mast cell direct interaction" type, where acetylcholine directly triggers mast cell activation, resulting in damage or obstruction of eccrine sweat ducts [2]. The Japanese Dermatological Association proposed a clinical diagnosis criteria that includes 1) the absence of other neurological or autonomic symptoms and 2) anhidrotic or hypohidrotic areas that affect more than 25% of the body [2].

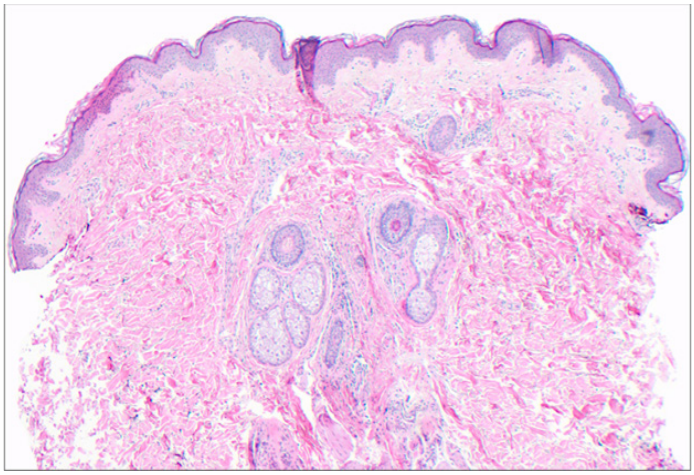

Figure 1: Lower back, punch biopsy, H&E 40x.The epidermis is unremarkable. In the dermis, there is a mild, superficial and deep, perivascular and periadnexal inflammatory infiltrate with lymphocytes and mild superficial edema.

Recent studies increasingly support an autoimmune cholinergic receptoropathy model of AIGA, characterized by autoreactive T-cell infiltration around eccrine glands, mast cell–acetylcholine interactions, and downstream cytokine release. While earlier descriptions emphasized 'sweat allergy' versus direct mast cell activation, newer data suggest these processes may coexist within a broader spectrum of immune dysregulation. Notably, immunohistochemistry has shown both T-cell and mast cell infiltration with increased expression of Th2 cytokines,

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underscoring the immune basis of the disorder. This evolving understanding also informs therapy: in addition to systemic corticosteroids, recent reports describe efficacy of cyclosporine and pilocarpine as adjuncts, particularly in refractory disease [4].

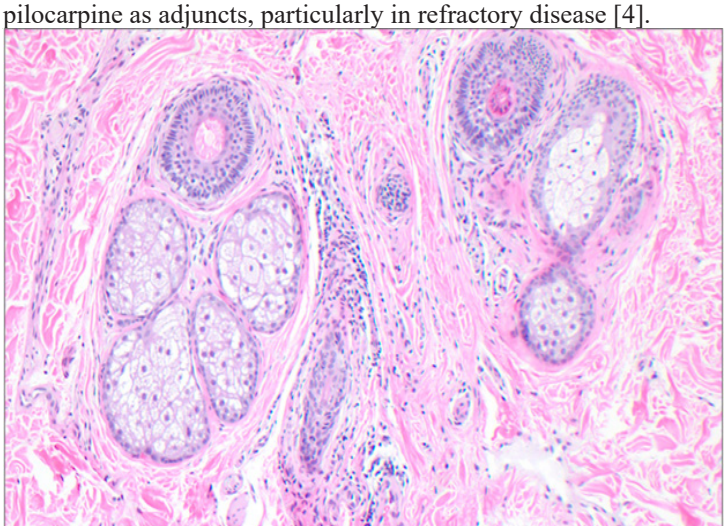

Figure 2: Left lower back, punch biopsy, H&E 100x

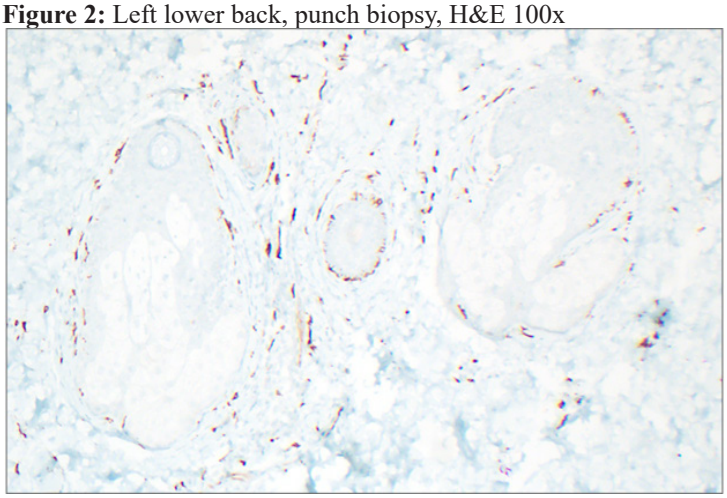

Figure 3: Left lower back, punch biopsy, CD117, Increased mast cells are identified with tryptase and CD117 in a perivascular and periadnexal distribution.

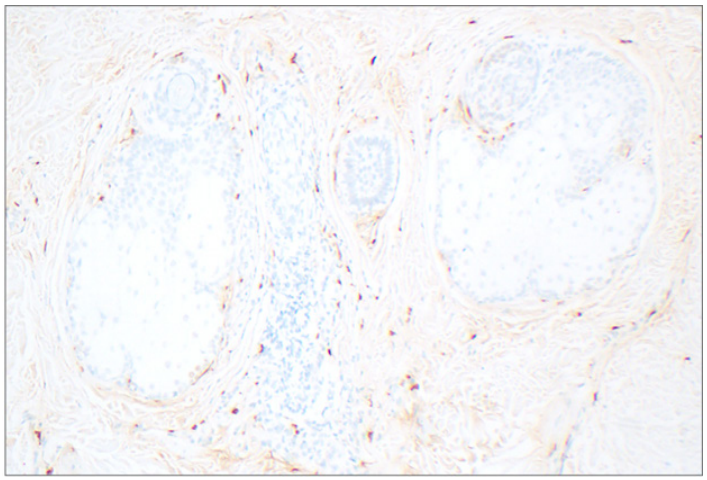

Figure 4: Left lower back, punch biopsy, Tryptase

Histopathological examination of reported cases consistently demonstrates CD3+ lymphocytic infiltration around eccrine sweat glands, supporting the autoimmune hypothesis [2, 5]. Increased mast cell presence and activity, as evidenced by positive CD117 and tryptase staining, further supports the inflammatory nature of the condition [2, 6]. Although not necessary for diagnosis, a skin biopsy demonstrating peri-eccrine inflammation with lymphocytes and mast cells can support the diagnosis of AIGA. The preservation of adrenergically-innervated apocrine gland function (such as axillary sweating) provides additional evidence that the primary pathological process specifically targets cholinergic muscarinic M3 receptors on eccrine sweat glands [2].

Further study of potential treatments is warranted. One such study examined the efficacy of oral pilocarpine, both as a primary treatment, and as adjuvant treatment in combination with pulse steroids. Though the study was quite small, consisting of only 10 patients, the combination of steroid pulse therapy with oral pilocarpine showed modest success, with a sound underlying principle of treatment. Pilocarpine targets the same M3 muscarinic receptors that are most commonly dysfunctional in AIGA patients, and therefore may aid in maximizing sweat gland function in those patients who experience only partial recovery of sweating function. Other studies have seen success with cyclosporine [7,8]. Mok and Tey reported successful treatment of AIGA with cyclosporine in two cases, suggesting that targeted immunosuppression may be effective for patients who do not respond to or cannot tolerate corticosteroids [7]. This approach offers treatment of the underlying autoimmune process through a different mechanism than corticosteroids. However, pulse steroid currently remains the most common and well documented treatment for its efficacy, safety, and low side effect profile.

AIGA is frequently responsive to early treatment as illustrated with our 31-year old active duty male. However, it may also recur, with nearly half of patients experiencing reduced sweat function within a year of treatment [5]. A younger age of onset and delay between the appearance of symptoms and treatment correlate with worse outcomes and increased recurrence [5]. Monitoring, identifying, and intervening in preventable heat injuries is a matter of increasing importance within the DoD. Joint service investments such as the Warrior Heat and Exertion Related Collaboration (WHEC) and the Consortium for Health and Military Performance (CHAMP) underscore an all-services commitment to combating heat injury. An undiagnosed case of AIGA drastically increases the likelihood of heat stroke, and the rarity of the disease is part of the threat it poses, although the condition may be poorly recognized and under reported [9]. In this case the treatment was simple, and as a result of an early diagnosis and prompt treatment, the patient was able to return to a fit for full duty status without limitation.

AIGA, though rare, poses significant risk to military personnel operating in hot climates. Early recognition, exclusion of secondary causes, and prompt corticosteroid therapy can restore sweat function and prevent heat injury. Military physicians should maintain a high index of suspicion for AIGA in servicemembers presenting with unexplained anhidrosis. Multidisciplinary approaches are necessary as patients may benefit from coordination between dermatology, neurology, and occupational medicine

specialists. Finally, long-term monitoring is needed given the high recurrence rate.

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