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Refractory Chronic Urticaria Associated with Widal Syndrome: A Case Report

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Abstract **Case Report**

Widal syndrome, also known as aspirin-exacerbated respiratory disease (AERD), is characterized by the association of asthma, chronic rhinosinusitis with nasal polyps, and hypersensitivity to non-steroidal anti-inflammatory drugs (NSAIDs). Cutaneous manifestations may occur following NSAID exposure. We report the case of a 50-year-old woman with asthma and a history of surgery for nasosinusal polyposis who presented with recurrent chronic urticaria associated with angioedema and exacerbated by NSAID intake. The association of asthma, nasal polyposis, and NSAID hypersensitivity led to the diagnosis of Widal syndrome. Treatment with montelukast combined with up-dosed antihistamine therapy and NSAID avoidance resulted in sustained clinical improvement at 6 months.

Keywords: Chronic urticaria; Widal syndrome; asthma; nasal polyposis; NSAID hypersensitivity; montelukast.

INTRODUCTION

Chronic spontaneous urticaria (CSU) is characterized by recurrent wheals, angioedema, or both for more than six weeks. It may represent a therapeutic challenge when symptoms persist despite antihistamine therapy.

Widal syndrome, also known as Samter's triad or aspirin-exacerbated respiratory disease (AERD), is characterized by asthma, chronic rhinosinusitis with nasal polyps, and hypersensitivity to NSAIDs [1,2]. We report a case of refractory chronic urticaria exacerbated by NSAID intake in a patient with asthma and a history of nasal polyposis, leading to the diagnosis of Widal syndrome.

CASE REPORT

A 50-year-old woman with a medical history of bronchial asthma treated with inhaled corticosteroids combined with long-acting beta-2 agonists and short-acting beta-2 agonists as rescue therapy was admitted to our department for recurrent chronic urticaria.

She had previously undergone surgery for nasosinusal polyposis and was also followed in the rheumatology department for fibromyalgia, for which she frequently used NSAIDs.

The patient presented with recurrent urticarial lesions associated with episodes of angioedema and anaphylactic shock. Physical examination showed a patient in fair general condition who was hemodynamically, neurologically, and respiratorily stable. Her body mass index was 29 kg/m², and her skin phototype was III.

Dermatological examination revealed multiple non-fixed urticarial papules, predominantly involving the lower abdomen and lower limbs, without dermographism (Figure 1). The remainder of the physical examination was unremarkable.

Investigations for physical and inducible urticaria were negative. Laboratory investigations revealed a mild inflammatory response. Autoimmune screening, including thyroid and antinuclear antibody testing, was unremarkable. Screening for gastrointestinal parasitic infections and *Helicobacter pylori*, complement and C1 esterase inhibitor levels, serum protein electrophoresis, viral serologies, and total and specific IgE testing did not reveal significant abnormalities.

A detailed medical history revealed that the urticarial exacerbations occurred in association with NSAID intake, particularly during painful episodes related to fibromyalgia. Given the history of bronchial asthma, previous surgery for nasosinusal polyposis, and

clinical exacerbations associated with NSAID exposure, a diagnosis of Widal syndrome was made.

NSAID avoidance was recommended. Treatment with montelukast 10 mg/day combined with up-dosed antihistamine therapy was initiated. The clinical course was favorable, with sustained improvement at the 6-month follow-up.

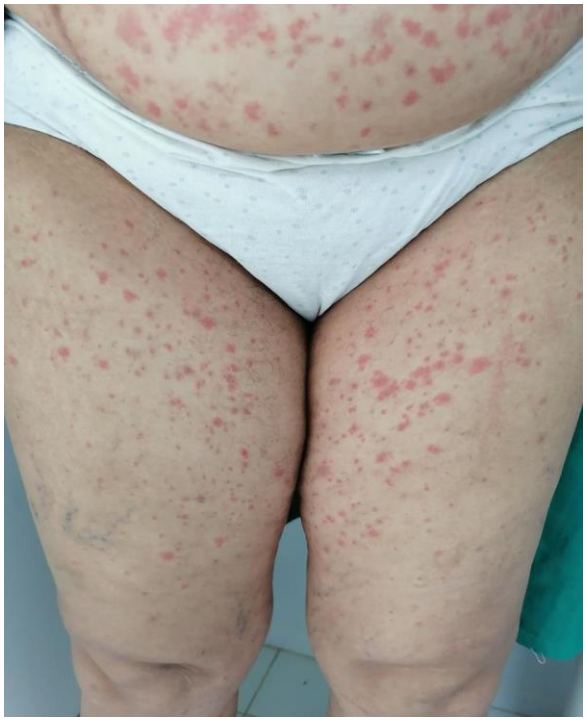

Figure 1: Multiple erythematous, round or polycyclic urticarial papules, some confluent, with a bilateral distribution on the lower limbs

DISCUSSION

Widal syndrome, also known as Samter's triad or aspirin-exacerbated respiratory disease (AERD), is characterized by the association of asthma, chronic rhinosinusitis with nasal polyps, and hypersensitivity to NSAIDs [1,2,5]. It was first described by Fernand Widal and colleagues in 1922 and mainly affects adults [1].

The pathophysiology of AERD is complex and not fully understood. It involves dysregulation of arachidonic acid metabolism, particularly an increased production of cysteinyl leukotrienes [1,2]. In susceptible patients, inhibition of cyclooxygenase-1 by aspirin or other NSAIDs may trigger clinical manifestations [1,4].

The clinical presentation of AERD is predominantly respiratory, including asthma exacerbations and upper airway symptoms related to chronic rhinosinusitis and nasal polyposis [1,2,5]. However, other manifestations, including cutaneous symptoms, may occur following NSAID exposure [1,3].

The diagnosis is primarily based on the clinical association of asthma, chronic rhinosinusitis with nasal polyps, and a history of hypersensitivity reactions to NSAIDs [1,5]. When the clinical history is uncertain, an aspirin challenge may be considered in an appropriate medical setting [1].

In our patient, the history of bronchial asthma and previous surgery for nasal polyposis, combined with recurrent exacerbations temporally associated with NSAID intake, supported the diagnosis of Widal syndrome. The association between NSAID exposure and urticarial exacerbations was particularly important in identifying the underlying drug hypersensitivity.

Management of AERD includes appropriate treatment of asthma and chronic rhinosinusitis with nasal polyps, as well as avoidance of the NSAIDs responsible for hypersensitivity reactions [1,4,5]. Aspirin desensitization may be considered in selected patients [1,4]. Leukotriene-modifying agents, including montelukast, may also be useful because of the important role of cysteinyl leukotrienes in the pathophysiology of the disease [1,2]. Biologic therapies may be considered in selected patients with severe respiratory disease [1,2].

Management of associated chronic urticaria should follow the usual therapeutic approach, with second-generation H1-antihistamines as first-line therapy and dose escalation when necessary. In our patient, the combination of NSAID avoidance, montelukast, and up-dosed antihistamine therapy was

associated with sustained clinical improvement at 6 months.

This case emphasizes the importance of obtaining a detailed drug history in patients with chronic urticaria, particularly when exacerbations occur following NSAID intake. The coexistence of asthma and a history of nasal polyposis should raise suspicion of an associated NSAID hypersensitivity syndrome.

CONCLUSION

In patients with chronic urticaria exacerbated by NSAIDs, a detailed history of NSAID hypersensitivity and associated respiratory disease is essential. The coexistence of asthma and chronic rhinosinusitis with nasal polyps should raise suspicion of Widal syndrome/AERD. Recognition of this association may allow appropriate NSAID avoidance and optimization of treatment.

Conflicts of Interest

The authors declare no conflicts of interest regarding this work.

Author Contributions

Abdourahman Moussa, M. El Amraoui, H. Hassan, and F. Mohamed Sidi contributed to the preparation of the case report and discussion. R. Frikh and N. Hjira critically revised the manuscript and approved the final version.

Patient Consent

Written informed consent was obtained from the patient for publication of this case report and the accompanying clinical image.

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