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Sinusitis as a cause of acute urticaria in a 14-year-old boy: a case report

Zapalenie zatok obocznych nosa jako przyczyna ostrej pokrzywki u 14-letniego chłopca: opis przypadku

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Abstract

Urticaria is a disease characterised by lesions, including wheals and angioedema. It can be divided into acute and chronic. Determining the aetiology of urticaria is often challenging; however, according to current guidelines, additional tests are not required in the acute form of the disease. This paper presents the case of a 14-year-old boy with urticaria, where treatment with second-generation antihistamines and systemic glucocorticosteroids did not alleviate the symptoms. Following further diagnostics, including a computed tomography scan of the sinuses, chronic sinusitis was considered a potential cause of the symptoms. This case exemplifies a situation where targeted diagnostics focusing on potential infection led to effective treatment of the underlying cause. Therefore, in clinically justified cases – especially those with atypical presentations of urticaria – searching for the underlying cause seems warranted. The article can contribute valuably to the discussion on current recommendations and foster a more individualised diagnostic and therapeutic approach to urticaria.

Keywords: children, allergy, sinusitis, acute urticaria, treatment of acute urticaria

Streszczenie

Pokrzywka to choroba charakteryzująca się występowaniem bąbli pokrzywkowych i obrzęku naczynioruchowego. Wyróżniamy postać ostrą (≤6 tygodni) i postać przewlekłą (>6 tygodni). Pokrzywka przewlekła może być idiopatyczna lub związana z określonymi czynnikami wyzwalającymi. Ustalenie jej etiologii jest często trudne, jednak, zgodnie z aktualnymi wytycznymi, w ostrej postaci choroby nie są wymagane badania dodatkowe. W poniższej pracy przedstawiono opis przypadku 14-letniego chłopca z pokrzywką, u którego leczenie z zastosowaniem leków przeciwhistaminowych II generacji oraz glikokortykosteroidów systemowych nie spowodowało ustąpienia objawów. Po pogłębieniu diagnostyki i wykonaniu tomografii komputerowej zatok za potencjalną przyczynę dolegliwości uznano przewlekłe zapalenie zatok, którego wyleczenie skutkowało remisją pokrzywki. Prezentowany przypadek dowodzi, że w przypadku nietypowego przebiegu pokrzywki zasadne wydaje się poszukiwanie przyczyny choroby i wykluczenie ogniska infekcji. Artykuł może stanowić wkład w dyskusję nad aktualnymi rekomendacjami i przyczynić się do bardziej zindywidualizowanego podejścia diagnostyczno-terapeutycznego w pokrzywce.

Słowa kluczowe: dzieci, alergia, zapalenie zatok, ostra pokrzywka, leczenie ostrej pokrzywki

INTRODUCTION

Urticaria, also known as hives, is a cutaneous disorder characterised by the presence of wheals and/or angioedema. Wheals are defined as elevated skin lesions with well-demarcated borders, superficial localisation, variable shapes, and surrounding erythema. These lesions are transient, typically resolving within 24 hours, and are associated with pruritus, sometimes accompanied by a burning sensation. In contrast, angioedema involves oedema of the deeper dermal layers. It may persist for up to 72 hours and is characterised by sensations of burning and tingling[1]. Urticarial lesions exhibit considerable variability, ranging from a few millimetres to several centimetres in diameter, with the number of lesions varying from a few to hundreds[1–3].

Urticaria is classified according to the duration of symptoms into acute (≤6 weeks) and chronic (>6 weeks) forms, as well as by the role of triggering factors as inducible or spontaneous. In cases of intermittent skin lesions, recurrent acute urticaria should be considered. The aetiopathogenesis of acute urticaria is most often linked to non-specific immune responses to viral infections, hypersensitivity reactions to medications, foods, or aeroallergens. In the majority of patients, a specific clinical cause cannot be identified, leading to the disease course being classified as idiopathic and self-limiting[1–3].

The first step in diagnosing urticaria is a detailed patient interview, followed by a physical examination. Because urticarial lesions are transient, they may not be present during the examination. Therefore, one should always review the photographic documentation provided by the patient, whenever possible. The next step involves performing laboratory tests. According to current guidelines for acute urticaria in children, if the clinical course is typical and the response to treatment is satisfactory, it is not recommended to routinely perform additional tests or search for the cause of the disease[4].

This article presents the case of a 14-year-old boy with acute urticaria in whom therapy with second-generation antihistamines and systemic glucocorticosteroids proved ineffective. Significant clinical improvement was achieved only after the identification and targeted antibiotic therapy of sinusitis.

CASE REPORT

A 14-year-old male patient was admitted to the Department of Paediatrics, Paediatric Nephrology and Allergology at the Military Institute of Medicine – National Research Institute due to urticaria refractory to outpatient treatment. The patient reported a 10-day history of urticarial wheals accompanied by angioedema. Initially, the skin lesions were localised to the trunk and scalp, but subsequently became generalised (Figs. 1 and 2). The patient was evaluated on three separate occasions at an urgent care clinic. At each visit, he received intramuscular dexamethasone (8 mg). During the first visit, desloratadine (5 mg twice daily) was also initiated; however, this treatment failed to provide the anticipated relief, with no observed resolution of the skin lesions.

On admission, the patient reported discomfort and intense pruritus. During the physical examination, the following significant deviations from the normal condition were found: severe urticarial lesions located on the trunk

Fig. 1. Skin lesions present at the time of admission

Fig. 2. Skin lesions present upon admission

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Laboratory parameter	Value on admission	Follow-up value
WBC [$\times 10^9$/L]	13.56	19.35
Neutrophils [$\times 10^2$/μL]	11.70	16,69
Neutrophils [%]	86.3	86.2
Lymphocytes [$\times 10^3$/μL]	1.05	1.88
Lymphocytes [%]	7.7	9.7
CRP [mg/dL, $N < 0.5$]	1.4	7.0
ESR [mm/h]	10	–
CRP – C-reactive protein; **ESR** – erythrocyte sedimentation rate; **WBC** – white blood cells.		

Tab. 1. Laboratory results on admission and follow-up

and limbs with accompanying swelling of the feet, hands, and facial area, reddened throat, mucous discharge in the nasal vestibule, and bilaterally opaque tympanic membrane on otoscopic examination.

Initial laboratory investigations revealed low levels of inflammatory markers. Liver and kidney function tests were within normal limits. Polymerase chain reaction (PCR) testing of a nasopharyngeal swab was positive for human coronavirus NL63. A diagnosis of acute urticaria secondary to an upper respiratory viral infection was established, and treatment with systemic glucocorticosteroids (prednisone 50 mg) and antihistamines (cetirizine 40 mg/day divided into four doses) was initiated.

Despite the initiation of treatment, there was no regression of skin lesions or oedema. Consequently, the decision was made to increase the glucocorticosteroid dose (prednisone 60 mg). Still, no alleviation of symptoms was observed. The patient also developed a fever reaching 39°C, which responded poorly to antipyretic medications. Follow-up laboratory tests revealed an upward trend in inflammatory markers (C-reactive protein, CRP: 7 mg/dL, $N < 0.5$ mg/dL) (Tab. 1). To further investigate potential sources of infection, radiological examination of the chest, routine urinalysis, and abdominal ultrasound were performed; however, these investigations were unremarkable.

Given the clinical findings, a computed tomography scan of the head and paranasal sinuses was conducted. The scan revealed significant thickening of the mucosal lining of all walls of the left maxillary sinus, as along with complete filling with frothy secretions (Fig. 3).

Fig. 3. Computed tomography of the paranasal sinuses

Based on the overall clinical presentation and the results of ancillary investigations, a diagnosis of urticaria secondary to paranasal sinusitis was established. Treatment was initiated with antibiotic therapy consisting of amoxicillin with clavulanate (1,200 mg three times daily) and metronidazole (500 mg twice daily), alongside symptomatic treatment. Following the initiation of this treatment regimen, rapid and complete resolution of the skin lesions and oedema was observed, along with improvement in the patient's overall condition.

DISCUSSION

Urticaria is a dermatological condition characterised by the presence of wheals (hives)[1]. Angioedema frequently accompanies urticaria, occurring in approximately 40% of cases. It affects the deeper layers of the skin or mucous membranes and may cause sensations of pressure or pain[5,6]. Angioedema typically presents as larger, well-defined areas of skin with normal or slightly pinkish discoloration. It can persist for 2–3 days. Angioedema results from mast cell activation in the skin, leading to the release of histamine and other inflammatory mediators, such as platelet-activating factor (PAF) and cytokines. This process causes stimulation of sensory nerves, vasodilation, and plasma extravasation[1,2,6,7].

Acute urticaria occurs in approximately 25% of children and adolescents, while chronic urticaria affects approximately 1.8% of the paediatric population[8,9].

Chronic urticaria can be idiopathic (spontaneous) (chronic spontaneous urticaria, CSU) or associated with specific triggering factors (chronic inducible urticaria, CIndU)[3,10,11] (Fig. 4).

Causes of spontaneous urticaria include inflammatory diseases of the gastrointestinal tract, parasitic infections, and infections of the nasopharynx and other inflammatory conditions in the head (teeth, gums, sinuses, ears). All of these account for approximately 90% of idiopathic urticaria cases[12].

In addition to infectious and inflammatory factors, stress and nonsteroidal anti-inflammatory drugs (NSAIDs), such as ibuprofen, play a significant role in the pathogenesis of

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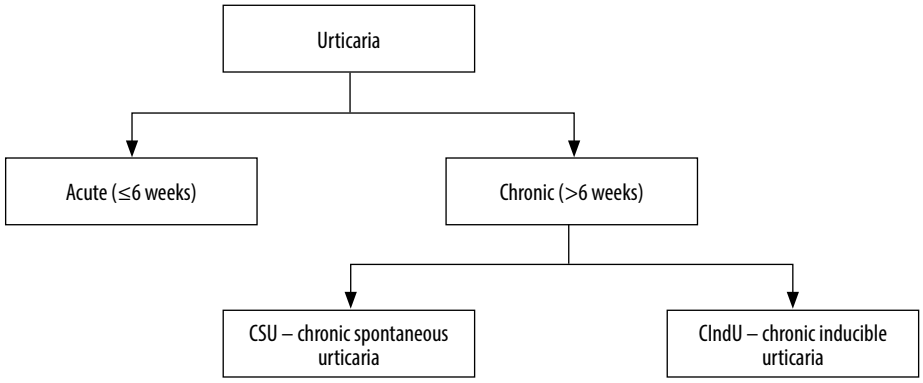

Fig. 4. Classification of urticaria

CSU (drug-induced urticaria accounts for 1–17% of CSU cases)[12]. IgE-mediated food allergy is an extremely rare cause of CSU. In such cases, it is essential to identify and eliminate the specific food allergens as quickly as possible, which usually leads to remission of urticarial lesions within less than 24 hours[3,10,11].

CIndU can be triggered by factors such as low or high temperatures, pressure from clothing, sunlight, water, and vibration[3,10,11].

International guidelines from the European Academy of Allergy and Clinical Immunology, the Global Allergy and Asthma European Network, the European dermatological guidelines, and the Asia Pacific Association of Allergology, Asthma, and Clinical Immunology (EAACI/GA²LEN/EuroGuiDerm/APAAACI) state that expanding the diagnostic workup is not recommended in cases of acute urticaria[4,13].

A detailed medical history is a crucial element of the diagnostic evaluation, enabling identification of potential triggering factors such as allergies, infections, and drug hypersensitivities. The most common drugs that trigger urticarial reactions include NSAIDs, antibiotics (penicillins, cephalosporins), iodinated contrast agents, angiotensin-converting enzyme inhibitors, opioids, heparins (especially low molecular weight heparins), biologic agents such as dupilumab, and antiepileptic drugs[11,14]. Expanding the diagnostic workup may be particularly warranted if type I allergy is suspected. In such cases, prick skin tests, determination of serum levels of specific IgE antibodies to allergens, diagnostic elimination diets lasting 3–4 weeks, and avoidance of potential allergens and histamine may be useful, representing additional, individualised diagnostic parameters.

Chronic urticaria requires a more complex approach. The diagnostic process begins with a thorough medical history, which helps identify potential triggering factors such as stress, medications, and comorbidities (e.g. autoimmune diseases, depression)[15]. A careful physical examination to assess skin changes (wheals, angioedema)[4] is another step. The next step involves laboratory tests, including complete blood counts with differential and markers of inflammation (CRP, erythrocyte sedimentation rate – ESR). Further diagnostic tests should be performed based on the medical history and results of the basic tests. These may include tests for infections (e.g. *Helicobacter pylori*), autoimmune diseases, or allergies[4]. Autoimmune diseases may play an important role in the pathogenesis of urticarial skin changes. Statistical data indicate that they are the cause of 30–50% of urticaria cases. This is particularly common in autoimmune hypothyroidism[12]. Urticaria may also coexist with rheumatoid arthritis, systemic lupus erythematosus, Sjögren's syndrome, coeliac disease, and type 1 diabetes[16]. It is also worth mentioning the significant role of psychiatric disorders in the aetiopathogenesis of acute urticaria; as a psychosomatic symptom, urticaria often precedes the diagnosis of anxiety disorders or depression[17]. Furthermore, cases of urticaria occurring in the course of infections such as sinusitis, tonsillitis, periapical abscesses, urinary tract infections, hepatitis, infectious mononucleosis, parasitic infestations, and *Mycoplasma pneumoniae* infections have been described in the literature[18,19].

In patients with exacerbations of urticarial lesions, a short course (maximum 10 days) of oral steroid therapy is permissible[4,17,20,21]. Treatment of bacterial infections and eradication of *Helicobacter pylori* are recommended. Psychotherapy is also helpful in many cases[4,17,21].

In the presented case, the patient's extremely persistent urticarial symptoms and lack of response to maximum doses of systemic glucocorticosteroids and antihistamines prompted a search for an infectious focus. The diagnosis of acute sinusitis, along with significant and immediate improvement following the implementation of antibiotic therapy, support a potential causal relationship between the chronic infection and the skin symptoms.

The association between infections, especially bacterial infections, and the occurrence of urticaria is well-recognised, although insufficiently documented in the paediatric population. Studies have shown that streptococcal infections, *Helicobacter pylori* infections, *Yersinia enterocolitica* infections, and sinus infections can act as triggers for urticarial reactions[22,23]. Pathophysiological mechanisms may include both direct activation of mast cells by bacterial toxins and indirect activation of the immune system via pro-inflammatory cytokines.

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However, it should be noted that the approach presented in this case of acute urticaria does not suggest the need for changes to the guidelines and should not lead to overuse of diagnostic tests. It is crucial to maintain a balance between adhering to guidelines that limit unnecessary interventions and individualising management in clinical situations that deviate from the typical course of the disease. The present case demonstrates that the presence of symptoms indicative of infection and lack of response to symptomatic treatment may justify targeted diagnostics and causal therapy, resulting in rapid clinical improvement.

CONCLUSIONS

In summary, despite the existing recommendations discouraging expanded diagnostics in the course of acute urticaria, each patient should be approached individually. In the described case, only expanding the diagnostic work-up to include imaging enabled the diagnosis of paranasal sinusitis, which manifested as urticaria. This, in turn, allowed for the implementation of appropriate treatment. This case demonstrates that urticaria may also serve as an indicator of an underlying infection and may be a non-specific symptom prompting further diagnostic evaluation.

Conflict of interest

The authors do not report any financial or personal connections with other persons or organisations which might negatively affect the content of this publication and/or claim authorship rights to this publication.

Author contribution

Original concept of study: AZ, AT. Collection, recording and/or compilation of data: AZ. Analysis and interpretation of data: AZ, NG. Writing of manuscript: AZ, NG, AM. Critical review of manuscript; final approval of manuscript: AT, BK.

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