



Submitted: 13.3.2018
Accepted: 6.4.2018
Conflict of interest
None.

DOI: 10.1111/ddg.13531

Chronic urticaria – What does the new guideline tell us?

Marcus Maurer, Torsten Zuberbier, Frank Siebenhaar, Karoline Krause

Department of Dermatology, Venereology, and Allergology, Charité – University Medical Center, Berlin, Germany

Section Editor
Prof. Dr. D. Nashan, Dortmund

Summary

Patients with chronic urticaria experience significant impairment, and require an effective treatment. Such treatment is preceded by a thorough diagnostic workup and measurement of disease activity, disease burden and disease control using well-established tools. Treatment is subsequently adjusted according to patient needs and therapeutic response, based on the tenet “as much as necessary, as little as possible” (in that order). Once disease control has been achieved, it is recommended that intermittent attempts at medication withdrawal be made in order to identify spontaneous disease remission. Chronic urticaria should be treated until spontaneous remission occurs.

Introduction

Chronic urticaria is defined by the presence of pruritic wheals, angioedema or both for a period lasting more than six weeks.

The updated version of the EAACI/GA²LEN/EDF/WAO urticaria guideline published in early 2018 contains new aspects regarding the diagnosis and treatment of patients with chronic urticaria. Chronic urticaria is a common disease, with a prevalence of at least 1 % [1]. It is defined by the presence of pruritic wheals, angioedema or both [2] for a period lasting more than six weeks.

With chronic urticaria, a distinction is made between chronic spontaneous urticaria (CSU) and chronic inducible urticaria (CIndU). The former is characterized by spontaneous symptoms that are not associated with a specific trigger. The latter, on the other hand, requires specific triggers for the urticarial symptoms to occur, such as sunlight, pressure, friction, or exposure to heat or cold (Table 1). While CSU is two to three times more common than CIndU [3], many patients have more than one type of chronic urticaria. Chronic urticaria can have a significant impact on quality of life; it is associated with depression and anxiety disorders and interferes with the patient's performance in everyday life, at school and at the workplace [4–6]. It is therefore crucial to provide chronic urticaria patients with the best treatment possible, that is, to attempt to achieve complete symptom control.

The treatment goal in urticaria is to achieve complete freedom from symptoms.

Table 1 Classification of chronic urticaria

Chronic urticaria		
Spontaneous urticaria (CSU)		Inducible urticaria (CIndU)
known cause	unknown cause	Physical urticaria Symptomatic dermographism Cold urticaria Solar urticaria Delayed pressure urticaria Vibratory angioedema Cholinergic urticaria Contact urticaria Aquagenic urticaria

In the vast majority of patients, this goal can be achieved with the treatment options and tools for measuring disease activity and control available today.

Pathogenesis of urticaria and clinical presentation

The wheals and angioedema associated with chronic urticaria are caused by active and degranulating mast cells [7]. Mast cell mediators such as histamine lead to vasodilatation and increased permeability, with subsequent edema in the upper dermis (wheals) and/or deep dermis and subcutis (angioedema). More than half of all CSU patients develop wheals and angioedema; up to one-third of CSU patients show wheals but no angioedema; and about one in ten CSU patients solely experiences angioedema but no wheals [3].

Based on what we know today, cutaneous mast cell degranulation in patients with chronic urticaria involves autoimmune mechanisms. Two different mechanisms are thought to be involved in CSU. In type 1 autoimmune CSU, IgE autoantibodies bind to the high-affinity (cutaneous) mast cell receptor. Degranulation occurs after autoallergen cross-linking (endogenous allergens recognized and bound by IgE) [8, 9]. Common CSU-associated autoallergens identified to date include thyroid peroxidase [10] and interleukin 24 [11].

In type IIb autoimmune CSU, mast cell degranulation is caused by IgG autoantibodies directed against the high-affinity IgE receptor [8, 12]. The definitive diagnosis of type IIb autoimmune urticaria is based on a positive autologous serum test, a positive basophil histamine release test, and evidence of these autoantibodies, for example by means of ELISA. The mechanisms involved in mast cell degranulation in CIndU have been less well investigated. Here, too, it is postulated that autoimmune mechanisms are responsible for cutaneous mast cell degranulation [13].

The reaction of mast cells to degranulators is modulated by many different signals. The most important mast cell modulators in CSU include neuropeptides, complement factors, pathogen-associated factors as well as foods and drugs in patients intolerant to the latter two. The presence and effects of one or more of these modulators, for example in case of mental stress or chronic infections, may explain their role in the pathogenesis and disease activity in patients with CSU. This also applies to the improvement in some patients following the avoidance of certain foods [14, 15] or the treatment of chronic infections.

Based on what we know today, cutaneous mast cell degranulation in patients with chronic urticaria involves autoimmune mechanisms.

In type 1 autoimmune CSU, IgE autoantibodies bind to the high-affinity (cutaneous) mast cell receptor.

In type 2b autoimmune CSU, mast cell degranulation is caused by IgG autoantibodies directed against the high-affinity IgE receptor.

Diagnostic approach to chronic spontaneous urticaria

The current version of the EAACI/GA²LEN/EDF/WAO urticaria guideline recommends the following diagnostic approach in patients with CSU:

1. Ruling out of differential diagnoses
2. Ruling out of severe inflammatory diseases
3. Measurement of disease activity and control.

The current version of the EAACI/GA²LEN/EDF/WAO urticaria guideline recommends the following diagnostic approach in patients with CSU: (1) Rule out differential diagnoses, (2) Rule out severe inflammatory diseases, and (3) measurement of disease activity and control [2].

Important differential diagnoses of CSU include autoinflammatory syndromes such as Schnitzler syndrome and urticarial vasculitis [16] (Figure 1). While these two disorders are likewise characterized by wheals, unlike CSU, these are mediated by interleukin 1 and not by histamine [17, 18]. It is therefore important to identify these differential diagnoses in order to initiate effective treatment. Besides chronic urticaria, recurrent angioedema without wheals also occurs in patients with bradykinin-mediated forms of angioedema such as angiotensin-converting enzyme (ACE) inhibitor-induced angioedema and hereditary angioedema (Figure 1). In these cases, the swelling is caused by bradykinin and does not respond to classic drugs used for urticaria.

Once the diagnosis of chronic urticaria has been confirmed, it is important to determine which form or forms of chronic urticaria the patient has. To this end, it is useful to ask patients whether they can deliberately trigger wheals or angioedema. While patients affected by the various forms of CIndU are able to do so, individuals with CSU are not.

The guideline recommends a two-step approach with respect to laboratory tests for patients with CSU. In all CSU patients, initial tests should include C-reactive protein levels and/or erythrocyte sedimentation rate as well as a differential blood count. While the goal of these tests is to rule out systemic inflammatory events, the urticaria itself may lead to elevated inflammatory marks, too. Depending on the history as well as on the duration and severity of chronic urticaria,

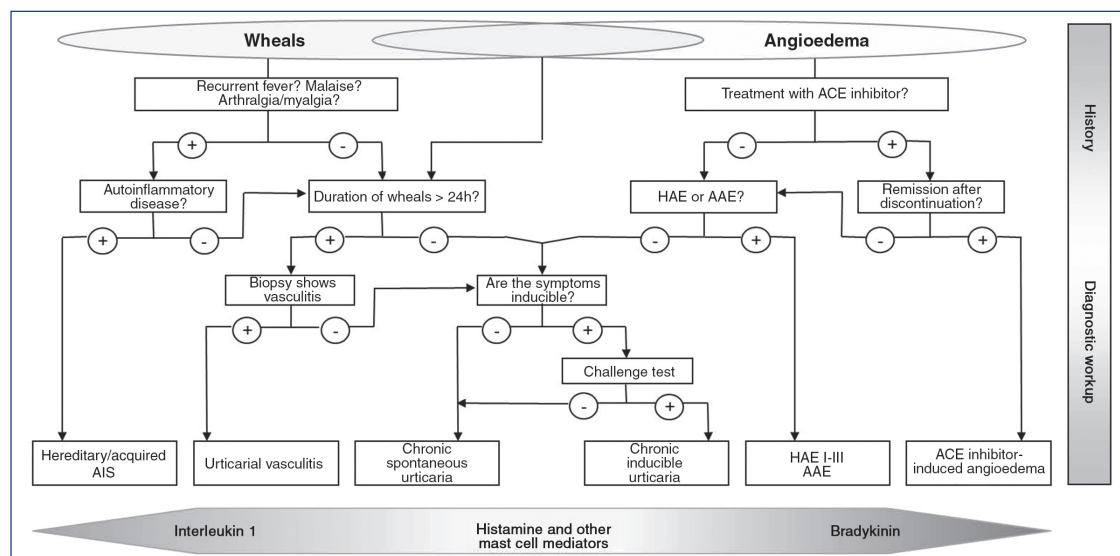


Figure 1 Diagnostic algorithm for patients with urticaria, angioedema or both. *Abbr.:* AAE, acquired angioedema with C1 inhibitor deficiency; ACE inhibitor, angiotensin-converting enzyme inhibitor; AIS, autoinflammatory syndrome; HAE, hereditary angioedema.

The current guideline endorses the urticaria activity score (UAS) and/or the angioedema activity score (AAS) to assess disease activity in CSU patients.

In addition, the urticaria control test (UCT) should be used in all CSU patients.

patients should subsequently undergo further diagnostic workup for causes and associated disorders, including autoimmune diseases, infections and intolerances.

For patients with CSU, the guideline recommends measurement of disease activity, disease burden and disease control using standardized tools. This enables the physician to accurately assess disease severity and course, including treatment success. Given that the majority of CSU patients exhibit considerable variability in daily symptoms, it is recommended to employ patient-reported outcome (PRO) tools. The current guideline endorses the urticaria activity score (UAS) [19–22] and/or the angioedema activity score (AAS) [23] to assess disease activity in CSU patients. In order to calculate the UAS, patients record and quantify their symptoms (wheals and pruritus) on a daily basis in a diary; the same applies to angioedema using the angioedema activity score. In addition, the urticaria control test (UCT) should be used in all CSU patients [24, 25].

The UCT is a retrospective tool used to rapidly and reliably assess disease control with four simple questions (Figure 2). Patients answer each of the four UCT questions, and the corresponding points (0–4 per answer) are added up to yield a total score of 0–16. The cut-off for controlled urticaria is 12 points. In other words: a score of 11 or less indicates insufficient disease control, whereas a score of 12 or more suggests adequate disease control [25].

Besides disease activity and control, it is also useful and recommended to assess quality-of-life impairment. This should be done on a regular basis, ideally with tools specifically designed for this purpose, such as the CU-Q2oL (Chronic Urticaria Quality-of-Life Questionnaire) [26] and the AE-QoL (Angioedema Quality-of-Life Questionnaire) [27, 28]. In everyday clinical practice, assessment of disease activity as well as of disease burden and control facilitates treatment decision making, especially prior to employing systemic therapies.

Urticaria control test

Name: _____ Date: ____ . ____ . ____

Date of birth: ____ . ____ . ____

Instructions: You have hives (urticaria). The following questions are meant to assess the current disease status. Please read each question carefully and select the one answer that *best describes your situation*. Please refer to the *past 4 weeks*. *Don't ponder for too long and remember to answer all questions* and to select *only one answer* for each question.

- To what extent have you suffered from **physical symptoms of urticaria** (pruritus, wheals and/or swelling) in the past 4 weeks?
☐ very severely ☐ severely ☐ moderately ☐ slightly ☐ not at all
- How much has your **quality of life** been affected by the urticaria in the past 4 weeks?
☐ very severely ☐ severely ☐ moderately ☐ slightly ☐ not at all
- How often has the **treatment** of your urticaria **not been sufficient** to control the urticaria symptoms in the past 4 weeks?
☐ very frequently ☐ frequently ☐ occasionally ☐ rarely ☐ not at all
- How well has your urticaria been **under control overall** in the past 4 weeks?
☐ not at all ☐ hardly ☐ moderately ☐ well ☐ completely

Figure 2 Urticaria Control Test (UCT).

Diagnostic approach to chronic inducible urticaria

If CIndU is suspected, ...

1. differential diagnoses should be ruled out.
2. the diagnosis should be confirmed by provocation tests.
3. disease activity should be measured by determining the trigger threshold.
4. disease burden and control should be measured.

If CIndU is suspected, (1) differential diagnoses should be ruled out, (2) the diagnosis should be confirmed by provocation tests, (3) disease activity should be measured by determining the trigger threshold, and (4) disease burden and control should be measured [2].

Differential diagnoses are ruled out by taking a thorough medical history, and possibly by subsequent diagnostic tests (Figure 1). Provocation tests are conducted to confirm the diagnosis in all forms of CIndU. In this context, it should be noted that a patient may have multiple forms of CIndU and should therefore be tested accordingly as suggested by his/her history. When performing provocation tests, it should be borne in mind that these tests are affected by symptomatic therapies such as antihistamines. Treatment should therefore be interrupted at least three – ideally seven – days prior to testing. If this is not possible, the test result should be evaluated with caution [29]. Provocation tests, just as determination of the trigger threshold, should be conducted prior to treatment initiation and during follow-up. To improve comparability of test results, standardized provocation test procedures should be used.

Similar to CSU, disease control in CIndU patients is assessed using the UCT. Unlike CSU, there are currently no disease-specific tools for determining the disease burden in CIndU patients [30].

Only for cholinergic urticaria is such a tool available, the CholU-QoL (Cholinergic Urticaria Quality-of-Life Questionnaire) [31].

Treatment of chronic urticaria

Treatment of chronic urticaria follows a standard approach with the goal of achieving complete freedom from symptoms. All patients should avoid known triggers. In particular, this includes certain medications such as nonsteroidal antiinflammatory drugs and relevant triggering stimuli in the case of CIndU. However, given that this approach results in freedom from symptoms in only very few cases, symptomatic treatment is recommended for nearly all patients, and should be based on the new treatment algorithm (Figure 3) [2].

Second-generation H₁ antihistamines remain the treatment of first choice.

If continuous treatment for 2–4 weeks does not lead to adequate control of symptoms, the guideline recommends up dosing (up to four times the standard dose).

If there is no improvement with high-dose antihistamines, it is recommended to add omalizumab to the regimen in patients with chronic spontaneous urticaria.

Second-generation H₁ antihistamines remain the treatment of choice. Unlike first-generation antihistamines, which should no longer be used, they have hardly any or only mild sedative effects. Initially, antihistamines shall be administered at the approved dose. It is important to instruct patients that antihistamines must be taken on a daily basis and not on demand. If continuous treatment for 2–4 weeks does not lead to adequate control of symptoms, the guideline recommends up dosing (up to four times the standard dose). Although using a higher-than-approved dose constitutes off-label treatment, it is much more effective than standard-dose therapy and has a similar side effect profile [32].

If sufficient improvement does not occur after 2–4 weeks of second-generation antihistamine therapy at a higher-than-standard dose, omalizumab should be added to the regimen. A recombinant humanized anti-IgE antibody, omalizumab is administered by subcutaneous injection at the recommended dose of 300 mg every four weeks. It is approved for the treatment of chronic spontaneous urticaria in patients 12 years and older. Most patients show a – usually complete – response even before the second dose [33–35]. A small percentage of patients require multiple doses to achieve treatment success [36]. Omalizumab has also been shown to be effective and safe in the treatment of CIndU patients. It is, however, not approved for this indication, at least not in patients with sole CIndU but without CSU [13, 37–46].

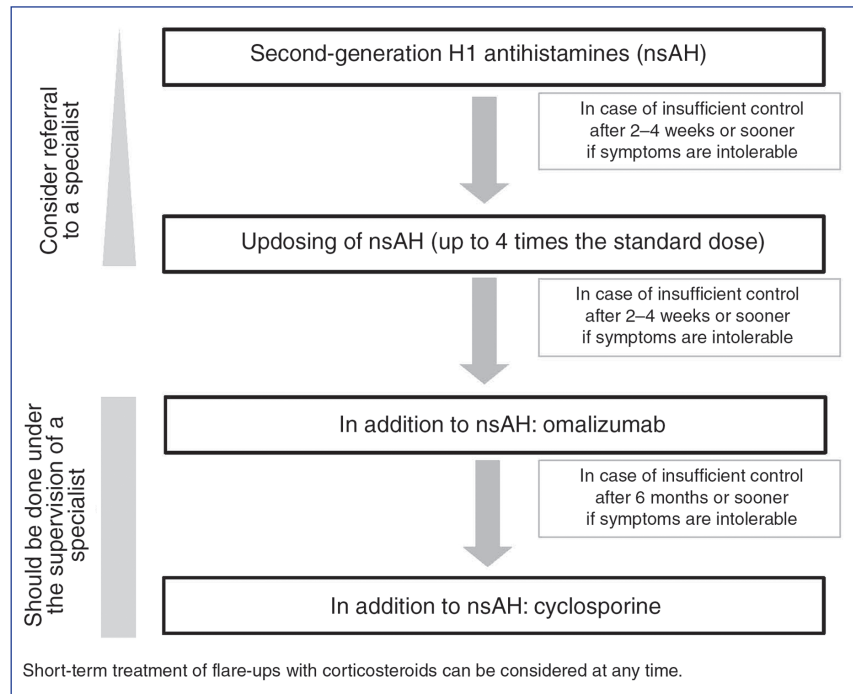


Figure 3 Recommended treatment algorithm for chronic urticaria.
nsAH: non-sedating antihistamine.

If there is no success after six months of omalizumab therapy, off-label treatment with cyclosporine is recommended.

If there is no success after six months of omalizumab therapy, off-label treatment with cyclosporine is recommended [2]. The efficacy of cyclosporine has been confirmed in placebo-controlled trials [47–49].

A crucial aspect to be generally considered in the treatment approach and patient management is the individual disease severity. Given the sheer prevalence of chronic urticaria, general practitioners or emergency medical services are frequently the first to be consulted. For patients with mild disease, standard-dose antihistamine therapy is usually sufficient. However, if this is not the case, patients should be referred to a specialist. Not only do specialists usually have more diagnostic options at their disposal, they can also collaborate with UCAREs (urticaria centers of reference and excellence). Certified by the European GA²LEN network (Global Allergy and Asthma European Network), these centers are characterized by a high degree of specialization in terms of research and clinical patient care. A list of these centers can be found at: <http://www.ga2len-ucare.com/centers.html>.

Correspondence to

Prof. Dr. med. Marcus Maurer
Klinik für Dermatologie, Venerologie und Allergologie
Charité – Universitätsmedizin
Berlin

Charitéplatz 1
10117 Berlin, Germany

E-mail: marcus.maurer@charite.de

References

- 1 Zuberbier T, Balke M, Worm M et al. Epidemiology of urticaria: a representative cross-sectional population survey. *Clin Exp Dermatol* 2010; 35: 869–73.
- 2 Zuberbier T, Aberer W, Asero R et al. The EAACI/GA²LEN/EDF/WAO Guideline for the Definition, Classification, Diagnosis and Management of Urticaria. The 2017 Revision and Update. *Allergy* 2018 [Epub ahead of print].
- 3 Maurer M, Weller K, Bindslev-Jensen C et al. Unmet clinical needs in chronic spontaneous urticaria. A GA²LEN task force report. *Allergy* 2011; 66: 317–30.
- 4 Maurer M, Abuzakouk M, Bérard F et al. The burden of chronic spontaneous urticaria is substantial: Real-world evidence from ASSURE-CSU. *Allergy* 2017; 72(12): 2005–2016.
- 5 Maurer M, Staubach P, Raap U et al. ATTENTUS, a German online survey of patients with chronic urticaria highlighting the burden of disease, unmet needs and real-life clinical practice. *Br J Dermatol* 2016; 174: 892–94.

- 6 Maurer M, Staubach P, Raap U et al. H1-antihistamine-refractory chronic spontaneous urticaria: it's worse than we thought—first results of the multicenter real-life AWARE study. *Clin Exp Allergy* 2017; 47: 684–92.
- 7 Church MK, Kolkhir P, Metz M, Maurer M. The role and relevance of mast cells in urticaria. *Immunol Rev* 2018; 282: 232–47.
- 8 Kolkhir P, Church MK, Weller K et al. Autoimmune chronic spontaneous urticaria: What we know and what we do not know. *J Allergy Clin Immunol* 2017; 139: 1772–81. e1.
- 9 Kolkhir P, Church MK, Weller K et al. Reply. *J Allergy Clin Immunol* 2018; 141(3): 1166–7.
- 10 Altrichter S, Peter H-J, Pisarevskaja D et al. IgE mediated autoallergy against thyroid peroxidase – a novel pathomechanism of chronic spontaneous urticaria? *PloS one* 2011; 6: e14794.
- 11 Schmetzer O, Lakin E, Topal FA et al. IL-24 is a common and specific autoantigen of IgE in patients with chronic spontaneous urticaria. *J Allergy Clin Immunol* 2017; pii: S0091–6749(17)31875-4.
- 12 Fiebiger E, Maurer D, Holub H et al. Serum IgG autoantibodies directed against the alpha chain of Fc epsilon RI: a selective marker and pathogenetic factor for a distinct subset of chronic urticaria patients? *J Clin Invest* 1995; 96: 2606–12.
- 13 Maurer M, Metz M, Brehler R et al. Omalizumab treatment in patients with chronic inducible urticaria: A systematic review of published evidence. *J Allergy Clin Immunol* 2018; 141: 638–49.
- 14 Magerl M, Pisarevskaja D, Scheufele R et al. Effects of a pseudoallergen-free diet on chronic spontaneous urticaria: a prospective trial. *Allergy* 2010; 65: 78–83.
- 15 Siebenhaar F, Melde A, Magerl M et al. Histamine intolerance in patients with chronic spontaneous urticaria. *J Eur Acad Dermatol Venereol* 2016; 30: 1774–7.
- 16 Maurer M, Magerl M, Metz M et al. Practical algorithm for diagnosing patients with recurrent wheals or angioedema. *Allergy* 2013; 68: 816–9.
- 17 Bonnekoh H, Scheffel J, Maurer M, Krause K. The use of skin biomarker profiles to distinguish Schnitzler's syndrome from chronic spontaneous urticaria: results of a pilot study. *Br J Dermatol* 2017; 561–2.
- 18 Krause K, Metz M, Makris M et al. The role of interleukin-1 in allergy-related disorders. *Curr Opin Allergy Clin Immunol* 2012; 12: 477–84.
- 19 Hawro T, Ohanyan T, Schoepke N et al. The Urticaria Activity Score – validity, reliability, and responsiveness. *J Allergy Clin Immunol Pract* 2017 [Epub ahead of print].
- 20 Hawro T, Ohanyan T, Schoepke N et al. Comparison and interpretability of the available urticaria activity scores. *Allergy* 2018; 73: 251–5.
- 21 Mathias SD, Crosby RD, Zazzali JL et al. Evaluating the minimally important difference of the urticaria activity score and other measures of disease activity in patients with chronic idiopathic urticaria. *Ann Allergy Asthma Immunol* 2012; 108: 20–4.
- 22 Młynek A, Zalewska-Janowska A, Martus P et al. How to assess disease activity in patients with chronic urticaria? *Allergy* 2008; 63: 777–80.
- 23 Weller K, Groffik A, Magerl M et al. Development, validation, and initial results of the Angioedema Activity Score. *Allergy* 2013; 68: 1185–92.
- 24 Ohanyan T, Schoepke N, Bolukbasi B et al. Responsiveness and minimal important difference of the urticaria control test. *J Allergy Clin Immunol* 2017; 140: 1710–3. e11.
- 25 Weller K, Groffik A, Church MK et al. Development and validation of the Urticaria Control Test: a patient-reported outcome instrument for assessing urticaria control. *J Allergy Clin Immunol* 2014; 133: 1365–72. e6.
- 26 Młynek A, Magerl M, Hanna M et al. The German version of the Chronic Urticaria Quality-of-Life Questionnaire: factor analysis, validation, and initial clinical findings. *Allergy* 2009; 64: 927–36.
- 27 Weller K, Groffik A, Magerl M et al. Development and construct validation of the angioedema quality of life questionnaire. *Allergy* 2012; 67: 1289–98.
- 28 Weller K, Magerl M, Peveling-Oberhag A et al. The Angioedema Quality of Life Questionnaire (AE-QoL)-assessment of sensitivity to change and minimal clinically important difference. *Allergy* 2016; 71: 1203–9.
- 29 Magerl M, Altrichter S, Borzova E et al. The definition, diagnostic testing, and management of chronic inducible urticarias – The EAACI/GA²LEN/EDF/UNEV consensus recommendations 2016 update and revision. *Allergy* 2016; 71: 780–802.

- 30 Weller K, Siebenhaar F, Hawro T et al. Clinical measures of chronic urticaria. *Immunol Allergy Clin North Am* 2017; 37: 35–49.
- 31 Ruft J, Asady A, Staubach P et al. Development and Validation of the Cholinergic Urticaria Quality of Life Questionnaire: CholU-QoL. *Clin Exp Allergy* 2018; 48(4):433–4.
- 32 Guillén-Aguinaga S, Jáuregui Presa I, Aguinaga-Ontoso E et al. Updosing nonsedating antihistamines in patients with chronic spontaneous urticaria: a systematic review and meta-analysis. *Br J Dermatol* 2016; 175: 1153–65.
- 33 Kaplan A, Ledford D, Ashby M et al. Omalizumab in patients with symptomatic chronic idiopathic/spontaneous urticaria despite standard combination therapy. *J Allergy Clin Immunol* 2013; 132: 101–9.
- 34 Maurer M, Rosén K, Hsieh H-J et al. Omalizumab for the treatment of chronic idiopathic or spontaneous urticaria. *N Engl J Med* 2013; 368: 924–35.
- 35 Saini SS, Bindslev-Jensen C, Maurer M et al. Efficacy and safety of omalizumab in patients with chronic idiopathic/spontaneous urticaria who remain symptomatic on H₁ antihistamines: a randomized, placebo-controlled study. *J Invest Dermatol* 2015; 135: 67–75.
- 36 Gericke J, Metz M, Ohanyan T et al. Serum autoreactivity predicts time to response to omalizumab therapy in chronic spontaneous urticaria. *J Allergy Clin Immunol* 2017; 139: 1059–61. e1.
- 37 Aubin F, Avenel-Audran M, Jeanmougin M et al. Omalizumab in patients with severe and refractory solar urticaria: a phase II multicentric study. *J Am Acad Dermatol* 2016; 74: 574–5.
- 38 Bindslev-Jensen C, Skov PS. Efficacy of omalizumab in delayed pressure urticaria: a case report. *Allergy* 2010; 65: 138–9.
- 39 Boyce JA. Successful treatment of cold-induced urticaria/anaphylaxis with anti-IgE. *J Allergy Clin Immunol* 2006; 117: 1415–8.
- 40 Bullerkotte U, Wiczorek D, Kapp A, Wedi B. Effective treatment of refractory severe heat urticaria with omalizumab. *Allergy* 2010; 65: 931–2.
- 41 Ghazanfar M, Sand C, Thomsen S. Effectiveness and safety of omalizumab in chronic spontaneous or inducible urticaria: evaluation of 154 patients. *Br J Dermatol* 2016; 175: 404–6.
- 42 Güzelbey O, Ardelean E, Magerl M et al. Successful treatment of solar urticaria with anti-immunoglobulin E therapy. *Allergy* 2008; 63: 1563–5.
- 43 Maurer M, Schütz A, Weller K et al. Omalizumab is effective in symptomatic dermographism – results of a randomized placebo-controlled trial. *J Allergy Clin Immunol* 2017; 140: 870–3. e5.
- 44 Metz M, Bergmann P, Zuberbier T, Maurer M. Successful treatment of cholinergic urticaria with anti-immunoglobulin E therapy. *Allergy* 2008; 63: 247–9.
- 45 Metz M, Ohanyan T, Church MK, Maurer M. Retreatment with omalizumab results in rapid remission in chronic spontaneous and inducible urticaria. *JAMA Dermatol* 2014; 150: 288–90.
- 46 Metz M, Schütz A, Weller K et al. Omalizumab is effective in cold urticaria – results of a randomized placebo-controlled trial. *J Allergy Clin Immunol* 2017; 140: 864–7. e5.
- 47 Grattan C, O'Donnell B, Francis D et al. Randomized double-blind study of cyclosporin in chronic 'idiopathic' urticaria. *Br J Dermatol* 2000; 143: 365–72.
- 48 Kulthanan K, Chaweekulrat P, Komoltri C et al. Cyclosporine for chronic spontaneous urticaria: a meta-analysis and systematic review. *J Allergy Clin Immunol Pract* 2018; 6(2): 586–99.
- 49 Vena GA, Cassano N, Colombo D et al. Cyclosporine in chronic idiopathic urticaria: a double-blind, randomized, placebo-controlled trial. *J Am Acad Dermatol* 2006; 55: 705–9.

Fragen zur Zertifizierung durch die DDA

1. Welche Aussage zur Urtikaria ist richtig?

- a) Die Urtikaria ist eine seltene Erkrankung.
- b) Die Urtikaria betrifft meist nicht nur die Haut.
- c) Bei Wiederauftreten von urtikariellen Beschwerden für mehr als sechs Wochen spricht man von einer akuten Urtikaria.
- d) Bei der spontanen Urtikaria kommt es nicht zu einer Spontanremission.
- e) Bei einer chronisch induzierbaren Urtikaria werden die Beschwerden durch spezifische Auslöser hervorgerufen.

2. Welche der folgenden Urtikariaformen ist **keine** chronisch induzierbare Urtikariaform?

- a) Urtikaria pigmentosa
- b) Kälteurtikaria
- c) Druckurtikaria
- d) Lichturtikaria
- e) Cholinergische Urtikaria

3. Welche Aussage zur Diagnostik der chronischen Urtikaria ist richtig?

- a) Bei allen Patienten sollte eine umfangreiche Ursachensuche mit Hilfe labordiagnostischer Tests erfolgen.
- b) Bei den meisten Patienten mit chronischer Urtikaria liegen Infekte zugrunde, die mit Hilfe einer gezielten Stuhl Diagnostik identifiziert werden können.
- c) Provokationstests sind insbesondere für das diagnostische Vorgehen bei Patienten mit chronischer spontaner Urtikaria relevant.
- d) Im Mittelpunkt der Diagnostik der chronischen induzierbaren Urtikaria steht die Familienanamnese.
- e) Bei Patienten mit chronischer induzierbarer Urtikaria sollten Provokationstests und Reizschwellenbestimmungen durchgeführt werden.

4. Welches Instrument dient nicht der Untersuchung von Patienten mit chronischer Urtikaria?

- a) Asthma-Kontroll-Test (ACT)
- b) Urtikaria-Kontroll-Test (UCT)
- c) Urtikaria-Aktivitäts-Score (UAS)
- d) Angioödem-Aktivitäts-Score (AAS)
- e) Chronische Urtikaria Quality of Life Questionnaire (CU-QoL)

5. Welche Aussage zum klinischen Bild der chronischen Urtikaria ist **falsch**?

- a) Es gibt Patienten mit chronischer Urtikaria, die ausschließlich Angioödem aufweisen und keine Quaddeln.
- b) Es gibt Patienten mit chronischer Urtikaria, die ausschließlich Quaddeln und keine Angioödem aufweisen.
- c) Es gibt Patienten mit chronischer Urtikaria, die Quaddeln und Angioödem aufweisen.
- d) Es gibt Patienten mit chronischer Urtikaria, die weder Quaddeln noch Angioödem aufweisen.
- e) Es gibt Patienten mit chronischer Urtikaria, bei denen Quaddeln und Angioödem nicht zeitgleich auftreten.

6. Welche Aussage zum diagnostischen Vorgehen bei chronischer Urtikaria ist richtig?

- a) Es sollte stets intensiv nach zugrundeliegenden Ursachen gefahndet werden.
- b) Provokationstestungen kommen bei chronischer induzierbarer Urtikaria nur in Ausnahmefällen in Betracht.
- c) Bei CSU-Patienten sollte immer ein Differenzialblutbild sowie CRP und/oder BSG bestimmt werden.
- d) Ein Hautpricktest auf Standardallergene sollte stets erfolgen.
- e) Bei Vorliegen eines symptomatischen Dermographismus ist eine chronische spontane Urtikaria ausgeschlossen.

7. Welche Aussage zur Diagnose und Differenzialdiagnose der chronischen Urtikaria ist **falsch**?

- a) Eine Hautbiopsie sollte in jedem Fall zur Diagnosesicherung erfolgen.
- b) Langanhaltende Quaddeln (> 24 h) können auf eine Urtikaria-Vaskulitis hindeuten.
- c) Begleitende rezidivierende Fieberschübe sollten an eine autoinflammatorische Erkrankung denken lassen.
- d) Angioödem können Histamin- oder Bradykinin-vermittelt sein.
- e) Antihistaminika sind bei ACE-Hemmer-induzierten Angioödem unwirksam.

8. Welche Therapie ist **nicht** für eine Langzeitbehandlung der chronischen Urtikaria geeignet?

- a) Antihistaminika der zweiten Generation, Standarddosis
- b) Antihistaminika der zweiten Generation, 4fache Standarddosis
- c) Ciclosporin
- d) Omalizumab
- e) Topische/systemische Glukokortikoide

9. Welche Aussage zur Therapie der chronischen Urtikaria ist **falsch**?

- a) Antihistaminika der ersten Generation sollten nicht mehr eingesetzt werden.
- b) Omalizumab ist nur bei chronischer spontaner Urtikaria wirksam.
- c) Eine Höherdosierung von Antihistaminika ist für alle Formen der chronischen Urtikaria empfohlen.
- d) Ciclosporin kann zur Behandlung der chronischen Urtikaria eingesetzt werden.
- e) Glukokortikoide sind nur zur kurzfristigen Behandlung der Urtikaria empfohlen.

10. Welche Aussage zur Therapie der chronischen Urtikaria ist richtig?

- a) Antihistaminika der ersten Generation sollten bevorzugt eingesetzt werden.
 - b) Eine kontinuierliche Einnahme von Antihistaminika ist nicht zu empfehlen.
 - c) Eine Höherdosierung von Antihistaminika der zweiten Generation ist aufgrund von Nebenwirkungen nicht empfehlenswert.
 - d) Omalizumab ist die Therapie der Wahl bei Antihistaminika-refraktären Patienten.
 - e) Die Behandlung der chronischen Urtikaria mit Ciclosporin ist obsolet.
-

Liebe Leserinnen und Leser,

der Einsendeschluss an die DDA für diese Ausgabe ist der 29. Juni 2018.

Die richtige Lösung zum Thema „Elastolysen und Hauterkrankungen mit Verlust der elastischen Fasern“ in Heft 2 (Februar 2018) ist: (1c, 2d, 3e, 4d, 5a, 6e, 7e, 8a, 9b, 10d).

Bitte verwenden Sie für Ihre Einsendung das aktuelle Formblatt auf der folgenden Seite oder aber geben Sie Ihre Lösung online unter <http://jddg.akademie-dda.de> ein.
