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Learn about our options for remote access Volume 16, Issue 5 First published: 11 May 2018 Section Editor Professor Dr. D. Nasan, Dortmund Chronic Urticaria is determined by the presence of itchy serum, angioedema or both over a period lasting more than six weeks. The goal of urticaria treatment is to achieve complete freedom from symptoms. Based on what we know today, the cytano degranulation of mast cells in patients with chronic hives involves autoimmune mechanisms. In type 1 of autoimmune CSS, IGE autoantibodies are associated with a high affinity (kare) receptor of mast cells. In type 2b autoimmune CSU, degranulation of mast cells is caused by IgG autoantibody, directed against the IGE receptor of high affinity. The current version of the EAACI/GA2LEN/EDF/WAO urticaria guide recommends the following diagnostic approach in patients with CSU: Correcting differential diagnosesBreaking from severe inflammatory diseasesMea disease activity and control. If CIndU is suspected, ... Diagnosis should be confirmed by provocative tests. Tests. be measured by determining the trigger threshold.disease burden and control must be measured. Second-generation H1 antihistamines remain the first choice. If continuous treatment for 2-4 weeks does not lead to adequate control of symptoms, the guide recommends uposing (up to four times the standard dose). If there is no improvement with high doses of antihistamines, it is recommended to add omalizumab to the regimen in patients with chronic spontaneous hives. If there is no success after six months of omalizumab therapy, offlabel treatment with cyclosporine is recommended. Patients with chronic hives experience significant disorders and require effective treatment. This treatment is preceded by careful diagnosis and measurement of disease activity, disease burdens and disease control using well-ed tools. Treatment is subsequently adjusted according to the needs of patients and the therapeutic response, based on the principles as much as possible, as little as possible (in this order). Once disease control has been achieved, periodic attempts to discontinue drugs to detect spontaneous remission of the disease are recommended. Chronic hives should be treated before spontaneous remission occurs. An updated version of the EAACI/GA2LEN/EDF/WAO urticaria manual, published in early 2018, provides new aspects of the diagnosis and treatment of chronic hives patients. Chronic hives is a common disease, with a prevalence of at least 1% 1. It is determined by the presence of itchy wheezes, angioedema or both 2 over a period of more than six weeks. In chronic hives, a distinction is made between chronic spontaneous hives (CSU) and chronic irrefutable hives (CIndU). The first is characterized by spontaneous symptoms that are not associated with a specific trigger. The latter, on the other hand, requires specific triggers for diuretic symptoms such as sunlight, pressure, friction, or exposure to heat or cold (table 1). Although CSU is two to three times more common than CIndU 3, many patients have more than one type of chronic hives. Chronic hives can have a significant impact on quality of life; it is associated with depression and anxiety disorders and interferes with the patient's work in daily life, school and workplace 4-6. It is therefore imperative to provide chronic urticaria patients with the best treatment possible, that is to try to achieve full control of symptoms. Table 1. Classification of Chronic Urticaria Chronic Urticaria Spontaneous Urticaria (CSU) Induced hives (CIndU) is known cause of unknown cause Physical Hives Symptomatic Dermography Cold Urticaria Solar Urticaria Delaying pressure of hives Vibrations Angioedemaneer Hologgic Contact urticaria Aquagene hives The vast majority of patients, this goal can be achieved with treatment options and tools to measure disease activity and control available today. Serum and angioedem associated with chronic hives are caused by active and degranulation of mast cells 7. Mediators of mast cells, such as histamine, lead to vasodilation and increased permeability, followed by swelling of the upper dermis (hustks) and/or deep dermis and subcutaneous (angioedema). More than half of all CSU patients develop wheezing and angioedem; up to one third of CSU patients show wheezing, but not angioedem; and about one in ten CSU patients only experiences angioedem, but not wheals 3. Based on what we know today, the cyano degranulation of mast cells in patients with chronic hives involves autoimmune mechanisms. Two different mechanisms are believed to be involved in the CSU. In type 1 of autoimmune CSS, IGE autoantibodies are associated with a high affinity (kare) receptor of mast cells. Degranulation occurs after automatic allergen cross-references (endogenous allergens recognized and associated with IGE) 8, 9. Common CSU-associated auto-allergens identified to date include thyroid peroxidase 10 and interleukin 24.11. In type 1b autoimmune CSU, degranulation of mast cells is caused by IgG autoantibody, directed against the IGE receptor of high affinity 8, 12. The final diagnosis of type 1b autoimmune hives is based on a positive autologous serum test, a positive test for the release of basophil histamine, and evidence of these autoantibodies, for example, using ELISA. Mechanisms involved in the degranulation of mast cells in CIndU have been less well researched. It also postulates that autoimmune mechanisms are responsible for cutaneous degranulation of mast cells 13. The reaction of mast cells to degranulators is modulated by many different signals. The most important obese cell modulators in CSU include neuropeptides, complex factors, pathogenic factors, and food and medicine in patients intolerant of the last two. The presence and exposure of one or more of these modulators, for example in the case of mental stress or chronic infections, may explain their role in pathogenesis and disease activity in patients with CSU. This also applies to improvements in some patients after avoiding certain foods 14, 15 or treating chronic infections. The current version of the EAACI/GA2LEN/EDF/WAO urticaria guide recommends the following diagnostic approach in patients with CSU: (1) Exclude differential diagnoses, (2) Exclude severe inflammatory diseases and (3) measure disease activity and control 2. Important differential CSU diagnoses include auto-inflammatory syndromes such as Schnitzler syndrome and hives vasculitis 16 (Figure 1). Although these two disorders are also wheezing, unlike the CSU, they are mediated interleukin 11 not histamine 17, 18. It is therefore important to identify these differential diagnoses in order to initiate effective treatment. In addition to chronic urticaria, recurrent angioedem without wheezing is also found in patients with bradyonin-mediated forms of angioedema, such as angiotensin-converting enzyme (APF), inhibitor and hereditary angioedema (Figure 1). In these cases, swelling is caused by bradykinin and does not respond to the classic drugs used for hives. Diagnostic algorithm for patients with hives, angioedema or both. Abbrev.: AAE, acquired angioedema with a deficiency of C1 inhibitor; ACE inhibitor, an angiotensin enzyme inhibitor; AIS, auto-splavable syndrome; HAE, hereditary angioedema. Once the diagnosis of chronic hives has been confirmed, it is important to determine which forms or forms of chronic hives in the patient. To this end, it is helpful to ask patients whether they can intentionally cause wheezing or angioedem. While patients affected by various forms of CIndU are able to do so, individuals with CSU are not. The guide recommends a two-step approach to laboratory tests for patients with CSU. In all CSU patients, initial tests should include levels of reactive protein and/or red blood cell deposition, as well as a differential blood test. While the purpose of these tests is to rule out systemic inflammatory events, hives alone can lead to elevated inflammatory, signs, too. Depending on the history, as well as the duration and severity of chronic hives, patients must subsequently undergo further diagnostic work for reasons and related disorders, including autoimmune diseases, infections and intolerances. For patients with CSU, the guide recommends measuring disease activity, disease burdens, and disease control using standardized tools. This allows the doctor to accurately assess the severity of the disease and the course, including the success of the treatment. Given that most CSU patients have significant variability in daily symptoms, it is recommended to use pro-reported results. Current guidance approves an assessment of hives activity (UAS) and/or angioedema activity (AAS) activity assessment to assess CSU activity in patients with CSU. The current guidance approves the evaluation of urticaria activity (UAS) 19-22 and/or an angioedema activity assessment (AAS) 23 to assess disease activity in CSU patients. In order to calculate UAS, patients record and quantify their symptoms (serum and indol) on a daily basis in the diary; the same applies to angioedem using angioedem activity assessment. In addition, the Urticaria Control Test (UCT) should be used in all CSU. UCT is a retrospective tool used to fast and reliable four simple questions (Figure 2). Patients answer each of the four UCT questions, and the corresponding scores (0-4 per answer) amount to give an overall score of 0-16. The cut for controlled hives is 12 points. In other words: an estimate of 11 or less indicates insufficient disease control, while an estimate of 12 or more offers adequate disease control of 25. Urticaria Test (UCT). In addition to diagnosis and disease control, it is also helpful and recommended for assessing quality of life. This should be done on a regular basis, ideally with tools specifically designed for this purpose, such as CU-2ol. (Chronic quality and life questionnaire Of Urticaria) 26 and Ae'ol. (Angioedema's quality and life questionnaire) 27, 28. In everyday clinical practice, assessing disease activity, disease burdens and control, facilitates treatment decisions, especially before systemic therapy is used. If you suspect CIND (1) differential diagnoses should be excluded, (2) the diagnosis should be confirmed by provocative tests, (3) the activity of the disease should be measured by determining the trigger threshold, and (4) the burden of disease and control should be measured 2. Differential diagnoses are excluded through a thorough medical history and possibly subsequent diagnostic tests (Figure 1). Provocative tests are carried out in all forms of CINDU to confirm the diagnosis. In this context it should be noted that the patient may have several forms of CIndU and therefore needs to be tested appropriately as suggested by his/her story. When performing provocative tests, keep in mind that these tests suffer from symptomatic therapy such as antihistamines. Therefore, treatment should be interrupted at least three - ideally seven - days before testing. If this is not possible, the test result should be evaluated with caution 29. Provocative tests, as well as the definition of the trigger threshold, should be carried out before treatment and during subsequent treatment. Standardized provocative testing procedures should be used to make test results more comparable. Like CSU, disease control in CIndU patients is assessed by UCT. Unlike CSU, there are currently no disease-specific tools for determining the burden of disease in patients with CIndU 30. Only for cholelithic hives there is such a tool, Cholu'ol. (Cholelithic Craiprat quality of life questionnaire) 31. Treatment of chronic hives follows a standard approach in order to achieve complete freedom from symptoms. All patients should avoid known triggers. In particular, this includes some medications such as non-steroidal anti-inflammatory drugs and appropriate trigger stimuli in the case of CIndU. However, given that this approach leads to freedom from symptoms only in a very very few cases, symptomatic treatment is recommended for almost all patients, and should be based on a new treatment algorithm (Figure 3) 2. Recommended algorithm for the treatment of chronic hives. nsAH: unseeded antihistamine. Second-generation H1 antihistamines remain the treatment of choice. Unlike first-generation antihistamines, which should no longer be used, they have almost no or only mild sedative effects. Initially, antihistamines are administered at an approved dose. It is important to instruct patients that antihistamines should 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 guide recommends uposing (up to four times the standard dose). Although the use of a higher than approved dose is a non-labeled treatment, it is much more effective than the standard dose of therapy and has a similar side effects profile of 32. If after 2-4 weeks of second-generation antihistamine therapy in a higher than the standard dose, it is necessary to add sufficient improvement to the regimen. Recombinant humanized antibodies against IgE, omalizumab is administered subcutaneous injection at a recommended dose of 300 mg every four weeks. It is approved for the treatment of chronic spontaneous hives in patients aged 12 years and older. Most patients show - usually a complete - response before the second dose is 33-35. A small percentage of patients require multiple doses to succeed treatment of 36. Omalizumab has also been shown to be effective and safe in treating CIndU patients. This, however, is not approved for this indication, at least not in patients with a single CIndU, but without CSU 13, 37-46. If there is no success after six months of omalizumab therapy, offlabel treatment with cyclosporine is recommended 2. The effectiveness of cyclosporine was confirmed in placebo-controlled trials 47-49. An important aspect that should usually be taken into account in the approach to treatment and management of patients is the individual severity of the disease. Given the prevalence of chronic hives, general practitioners or emergency services are often the first to consult. For patients with mild illness, a standard dose of antihistamine therapy is usually sufficient. However, if this is not the case, patients should be referred to a specialist. Professionals not only tend to have more diagnostic options at their disposal, but they can also collaborate with UCARes (urticaria centers and best practices). Certified by the European GA2LEN Network (Global Allergy and Asthma Network), these centers are highly specialized in terms of research and clinical patient care. 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In the case of relapse of critical discomfort for more than six weeks, it is called acute hives. Spontaneous hives do not lead to spontaneous remission. In chronically crop urticaria, symptoms are caused by specific triggers. Which of the following forms of hives is not chronically induced by the form of hives? Urticaria pigmentosa cold hives Pressure Urticaria Lichturticaria Cholelithic Urticaria What is the claim about the diagnosis of chronic hives is correct? In all patients, an extensive search of the cause should be conducted through laboratory diagnostic tests. In most patients with chronic hives, infections are essential, which can be identified through targeted stool diagnosis. Provocative tests are especially relevant for diagnostic procedure in patients with chronic spontaneous hives. Family history is at the center of the diagnosis of chronic non-cognitive hives. In patients with chronic non-cognitive hives, provocative tests and stimulation threshold determination should be performed. Which tool is not used to examine patients with chronic hives? Asthma Control Test (ACT) Urticaria Control Test (UCT) Urticaria Activity Assessment (UAS) Angioedema Activity Assessment (AAS) Chronic Urticaria Quality of Life Questionnaire (CU-Ash) What is the statement about the clinical picture of chronic hives is wrong? There are patients with chronic hives who have only angioding and no quad bikes. There are patients with chronic hives who have only quads and no angioedema. There are patients with chronic hives who

quads and angioedema. There are patients with chronic hives who do not have quads or angioedema. There are patients with chronic hives, whose quads and angioedema do not meet simultaneously. What is the correct statement about the diagnostic procedure of chronic hives? There should always be an intense search for the root causes. Provocative tests are possible only in exceptional cases in chronic non-cognitive hives. In CSU patients should always determine differential blood test, as well as CRP and/or BSG. A skin prick test for standard allergens should always be conducted. In the presence of symptomatic dermatography, chronic spontaneous hives are excluded. What is the wrong claim about the diagnosis and differential diagnosis of chronic hives? A skin biopsy should be performed in any case for diagnosis. Long-term quads (24 hours) may indicate hives vasculitis. Accompanying recurrent fevers should make you think about auto-inflammatory diseases. Angioedema can be histamine or bradyconin-mediated. Antihistamines are ineffective in an ACE inhibitor caused by angioedema. What therapy is not suitable for long-term treatment of chronic hives? Second-generation antihistamines, standard dose of second-generation antihistamines, 4-fold standard dose of cykloporin omalizumab topical /systemic glucocorticoids What is wrong with the treatment of chronic hives? First-generation antihistamines should no longer be used. Omalizumab is only effective in chronic spontaneous hives. A higher dose of antihistamines is recommended for all forms of chronic hives. Cyclosporine can be used to treat chronic hives. Glucocorticoids are recommended only for short-term urticaria treatment. What is the claim about chronic hives therapy correct? First-generation antihistamines should be preferred. Continuous use of antihistamines is not recommended. A higher dose of second-generation antihistamines is not recommended due to side effects. Omalizumab is the therapy of choice in antihistamine refracted patients. Treatment of chronic urticaria with cycloprin is outdated. Dear readers, the deadline for filing applications with DDA on this issue is June 29, 2018. Correct solution to the theme of Elastoholise and skin diseases with the loss of elastic fibers in issue 2 (February 2018): (1c, 2d, 3e, 4d, 5a, 6e, 7e, 8a, 9b, 10d). Please use the current form on the next page to send or enter the solution online . A. Birley's Machine, Mark Serota, David A. Norris, Nebula Skin Diseases: Alopecia Areata, Vitiligo, Psoriasis, and Urticaria, Autoimmune Diseases, 10.1016/B978-0-12-812102-3.00061-0, (1211-1234), (2020061-0, (1211-1234). Enrique Lopez Valentin, Alvaro Pedrosa Melendez, Jose Guadeloupe Huerta Lopez, Urticaria cr nica en ni os. Revisi n sistem tica, Alergia, Asma e Inmunologa Pedi tricas, 10.35366/93322, 29, 1, (16-30), (2020).M. Alc n tara Villar, J.C. Armario Hita, S. Chimbolek, M.D. Fernandez Ballos, M. 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