ASSESS THE EFFECTIVENESS OF NURSING CARE ON PATIENTS WITH PRESSURE URTICARIA

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ABSTRACT

The study was conducted to evaluate the effectiveness of Nursing care on patients with pressure urticaria. Delayed Pressure Urticaria is considered a rare disease, with clinical diagnosis different from classical urticaria, with possible systemic manifestations. Therefore, it is frequently underdiagnosed, even by specialists. In this article, the case of a patient with a typical history of pressure-induced lesions is presented. Because the patient had fever and leukocytosis, she was admitted to a hospital for investigation of infection. Chronic urticaria is defined as episodic or daily hives lasting for at least 6 weeks and impairs quality of life. Two main subtypes include chronic idiopathic (spontaneous) urticaria and inducible (physical) urticaria, but some patients have urticarial vasculitis. “Autoimmune chronic urticaria” implies the presence of histamine releasing or mast cell activating autoantibodies to Ige or fceri, the high affinity receptor on mast cells and basophils. In patients not readily controlled with labeled dosages of second generation H₁ receptor antagonists (antihistamines), there is evidence for reduction of urticaria using up to 4 fold increases in labeled dosages. The biologic modifier, omalizumab, helps to reduce lesions of chronic urticaria within 1–2 weeks. Chronic urticaria has a wide spectrum of clinical presentations and causes. Still, despite our best efforts no cause may be found in the majority of cases. The treatment options are: Primary prevention in the form of avoidance of aggravating factors; counseling; antihistamines; leukotriene receptor antagonists; prednisolone; sulfasalazine and a host of immunosuppressives like methotrexate, cyclosporine, omalizumab etc..

KEYWORDS: Chronic pressure urticaria, Chronic idiopathic urticaria, eosinophil cationic protein and factor, Urticaria, autoimmune urticaria, autoantibodies.
INTRODUCTION

Pressure urticaria is an uncommon form of physical urticaria. Patients who have had urticaria for more than 6 weeks are given the diagnosis of chronic urticaria. This distinction is important because an inciting event or etiology is not identified for the majority of patients with chronic urticaria—hence the often-used term chronic idiopathic urticaria (CIU). Baxi.S A proportion of patients with chronic urticaria have physical urticaria, which is urticaria incited by a physical stimulus, such as cold, vibration, or pressure.1 Brunet. L Pressure urticaria may occur immediately (within minutes) after a pressure stimulus. More commonly, however, it develops 4-6 hours after a pressure stimulus.2 Champion’ RH, or this reason, the term delayed pressure urticaria (DPU) is commonly used. The wheals may last for 8-72 hours. The hands, feet, trunk, buttocks, legs, and face are the area’s most commonly affected. Lesions can be induced by a variety of stimuli, including standing, walking, wearing of tight clothes, or sitting on a hard surface. For further information on urticaria, see Contact Syndrome Urticaria, Dermographism Urticaria, and Solar Urticaria. For patient education resources, see the Allergy Center and the Skin, Hair, and Nails Center, as well as Hives and Angioedema. Other possible mediators include eosinophils (as suggested by the presence of eosinophilia), eosinophil cationic protein (ECP), and eosinophil cationic factor (ECF) found in biopsy specimens from some patients with DPU, particularly bullous DPU. Elevated concentrations of interleukin (IL)-5 and IL-6 and of leukotrienes have also been found in lesional skin of pressure urticaria patients. Abnormalities in platelets and fibrin or fibrinolysis are also being investigated.

ETIOLOGY

Pressure stimuli may include the following: Standing, walking, or sitting on a hard surface, Using tools (eg; a screwdriver or a hammer), Hand clapping, Carrying a handbag, Wearing tight-fitting clothes (eg, bra straps, belts, shoes, cuffs, or watches), Dental work, Kissing, Sexual intercourse.

PATHOPHYSIOLOGY

The pathogenesis of DPU is unknown. In most cases, no allergen can be identified. Mast cells and histamine release are believed to play roles because the injection of compound 48/80%, which causes depletion of mast cell mediators, prevents the induction of lesions in the injected area. Histamine levels are increased in lesional skin, and intracellular histamine levels are decreased in peripheral white blood cells. Despite these findings and the finding of increased stimulated histamine release, histamine is not likely to be the sole mediator in pressure urticaria, given the relative unresponsiveness of the condition to antihistamine treatment.3 Diepgen.TL Other possible mediators include eosinophils (as suggested by the presence of eosinophilia), eosinophil cationic protein (ECP), and eosinophil cationic factor (ECF) found in biopsy specimens from some patients with DPU, particularly bullous DPU. Elevated concentrations of interleukin IL-5 and IL-6 and of leukotrienes have also been found in lesional skin of pressure urticaria patients. Abnormalities in platelets and fibrin or fibrinolysis are also being investigated.

CLINICAL MANIFESTATION

The clinical manifestations of delayed pressure urticaria (DPU) differ from those typical of most types of urticaria. Onset is typically delayed, most commonly occurring 4 hours after the pressure stimulus.4 Greaves.MW Less commonly, wheals due to pressure develop within minutes, in which case they may be confused with dermatographism. The lesions of DPU can persist for several hours and sometimes for as long as 48 hours, unlike those in typical urticaria, which resolve within 24 hours. The lesions may be pruritic, painful, or burning. They can occur on any cutaneous surface and may mimic angioedema. With severe episodes, patients may experience fever, malaise, fatigue, chills, headache, and generalized arthralgias.5 Hennino. A Affected areas can be refractory to the development of new lesions for 1-2 days. As many as 60% of individuals with DPU have concomitant chronic idiopathic urticaria (CIU), immediate or delayed dermographism (dermatographism), or angioedema. In some reports, the incidence of DPU in patients with CIU varies considerably, ranging from 2% to 40%; other reports estimate the rate to be 2-4%. The physical findings in DPU include wheals, typically involving the palms, soles, legs, and waist.6 Hide.M DPU lesions may also involve the genitals. The wheals, which appear as deep dermal and subcutaneous swellings, often resemble angioedema more than they do typical urticaria. Typical urticaria may also be present as a result of coexisting CIU or some other chronic physical urticaria.

DIAGNOSTIC EVALUATION

An elevated white blood cell (WBC) count or neutrophilia may be present. Complement levels are normal. Some patients with delayed pressure urticaria (DPU) also have concomitant chronic idiopathic urticaria (CIU). The following ancillary testing can be helpful in patients with CIU. The erythrocyte sedimentation rate (ESR), the C-reactive protein (CRP) level, or both may be elevated. Thyroid function tests and antibody testing for autoimmune thyroid disease may return positive results.7 Kalpan. A p Antibody testing for Helicobacter pylori can be performed; if results are positive, the infection should be treated. Testing for autoantibody to immunoglobulin can be performed.

PRESSURE CHALLENGE TESTING

Multiple methods of applying measured amounts of pressure can be used to test for the development of DPU. A consensus Pressure challenge testing (with the dermographometer or the suspended-weight method) may be performed for DPU. Because therapy may influence test results, it is recommended that testing be performed at a time when therapy has been interrupted or stopped; however, some patients may have more

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severe disease that does not allow this. Repetitive testing can be used to assess response to therapy.\textsuperscript{3}

**MANAGEMENT**

Patients should attempt to limit pressure stimuli. A simple intervention is to broaden the handles on heavy items or straps on clothing to disperse the pressure over a larger area. However, avoidance is not easy and may not be helpful in patients with moderate-to-severe disease. The results of pharmacologic treatment of delayed pressure urticaria (DPU) are somewhat disappointing. Besides antihistamines, agents used have included systemic corticosteroids, nonsteroidal anti-inflammatory drugs (NSAIDs), colchicine, dapsone, sulfasalazine, montelukast, chloroquine, cyclosporine, intravenous immunoglobulin and omalizumab. Restrictions in activity depend on the severity of the disease. Consult a dermatologist or allergist for evaluation for other causes of urticaria.\textsuperscript{4}

**PHARMACOLOGIC THERAPY**

Antihistamines can reduce the severity of symptoms and are helpful in controlling associated chronic idiopathic urticaria (CIU), but they may not control the symptoms completely. Some authors have used up to 4 times the recommended dose of non-sedating antihistamines to achieve control. NSAIDs produce variable responses. As recommended dose of nonsedating antihistamines to completely. Some authors have used up to 4 times the recommended dose of nonsedating antihistamines to achieve control.

The adverse effects of steroids must also be considered. Prednisone has some clinical efficacy, but long-term therapy is problematic because of its many adverse effects. The adverse effects of steroids must also be considered and managed. Patients may already be taking H2 blockers in an attempt to manage their urticaria; however, these are also used to treat gastritis from systemic steroid therapy. Long-term steroid use also affects the bones, and patients are often advised to take calcium with vitamin D daily, with additional consideration given to adding a bisphosphonate such as alendronate. Other therapeutic agents that have been tried include colchicine, dapsone, sulfasalazine, and montelukast. Colchicine has been largely ineffective as a therapy. Reports from small studies have found leukotriene antagonists, alone or in combination, to be efficacious for treatment of DPU; other forms of chronic urticaria have not demonstrated similar responses to this treatment. Case reports have demonstrated successful treatment with chloroquine. Cyclosporine and omalizumab have each been used in a small number of patients with severe and refractory disease. Combination therapy may decrease disease activity. Adjunctive agents that reportedly have been successfully employed in this context include leukotriene antagonists (eg, montelukast and zafirlukast) and H2-receptor antagonists (eg, famotidine and ranitidine). Agents used in the management of pressure urticaria include antihistamines, leukotriene antagonists, corticosteroids, and nonsteroidal anti-inflammatory drugs.

**LEVOCETIRIZINE (XYZAL)**

Levocetirizine is an H1-receptor antagonist and an active enantiomer of cetirizine. Peak plasma levels are reached within 1 hour, and the half-life is approximately 8 hours. Levocetirizine is available as a 5-mg breakable scored tablet and a 0.5 mg/mL oral solution. It is indicated for uncomplicated skin manifestations of chronic idiopathic urticaria.

**FEXOFENADINE (ALLEGRA)**

Fexofenadine is a non-sedating second-generation medication that has fewer adverse effects than first-generation medications. It competes with histamine for H1 receptors in the gastrointestinal (GI) tract, blood vessels, and the respiratory tract, reducing hypersensitivity reactions. Fexofenadine does not sedate. It is available in once-daily and twice-daily preparations.

**DESLORATADINE (CLARINEX)**

Desloratadine is a long-acting tricyclic histamine antagonist that is selective for H1 receptors. It is a major metabolite of loratadine, which, after ingestion, is extensively metabolized to the active metabolite 3-hydroxydesloratadine.\textsuperscript{10}

**CETIRIZINE (ZYRTEC)**

Cetirizine selectively inhibits H1 receptor sites in blood vessels, the GI tract, and the respiratory tract, thereby inhibiting the physiologic effects that histamine normally induces at H1 receptor sites. Once-daily dosing is convenient; bedtime dosing may be useful if sedation is a problem.

**ANTIHISTAMINES**

First-generation antihistamines compete with histamine at the tissue-receptor level, preventing it from carrying out its mediator functions in urticaria.\textsuperscript{11}

**DIPHENHYDRAMINE (BENADRYL, DIPHENHIST, ALLERDRYL)**

Diphenhydramine is a first-generation antihistamine with anticholinergic effects. It binds to H1 receptors in the central nervous system (CNS) and the body, competitively blocking histamine from binding to these receptors. Diphenhydramine is employed for symptomatic relief of pruritus caused by release of histamine in inflammatory reactions. It has significant antimuscarinic activity and penetrates the CNS and thus has a pronounced tendency to induce sedation. Approximately half of those treated with Non steroidal Anti-inflammatory Drugs.
IBUPROFEN (ADVIL, MOTRIN IB, NEOPROFEN, ADDAPRIN)

Ibuprofen is an NSAID with analgesic, anti-inflammatory, and antipyretic properties. It inhibits inflammatory reactions and pain by decreasing prostaglandin synthesis. 12

NAPROXEN (ALEVE, NAPRELAN, ANAPROX, NAPROSYN)

Naproxen inhibits inflammatory reactions and pain by decreasing the activity of cyclooxygenase, which results in a decrease of prostaglandin synthesis.

CORTICOSTEROIDS

Prednisone: Prednisone may decrease inflammation by reversing increased capillary permeability and suppressing polymorphonuclear leukocyte (PMN) activity. It stabilizes lysosomal membranes and suppresses lymphocyte and antibody production. 13

PREDNISOLONE (PEDIAPRED, ORAPRED, FLO-PRED)

This glucocorticoid occurs naturally and synthetically. It is used for both acute and chronic asthma. It may decrease inflammation by reversing increased capillary permeability and suppressing polymorphonuclear leukocyte activity.

ANTIHISTAMINES, H2 BLOCKERS

Famotidine is an H2 antagonist that, when combined with an H1 type, may be useful in treating allergic reactions that do not respond to H1 antagonists alone.

RANITIDINE (ZANTAC)

Ranitidine is a second-generation agent that is effective for the treatment of urticaria. It is tolerated very well, with a rate of sedation that is not significantly different from that seen with placebo.

NIZATIDINE (AXID, AXID AR)

This agent competitively inhibits histamine at the H2 receptor of the gastric parietal cells, resulting in reduced gastric acid secretion, gastric volume, and reduced hydrogen concentrations. 14

CONCLUSION

The goals of pharmacotherapy are to reduce morbidity and to prevent complications. Agents used in the management of pressure urticaria include antihistamines, leukotriene antagonists, corticosteroids, and non steroidal anti-inflammatory drugs.

REFERENCES