

A case of refractory chronic spontaneous urticaria treated with omalizumab

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Abstract Chronic spontaneous urticaria (CSU) is a chronic and refractory dermatosis. Omalizumab is a relatively new addition to CSU therapeutic armamentarium. We present a 52-year-old male having symptoms of chronic spontaneous urticaria (CSU) of moderate severity for last 15 years. He was on regular H1 antagonist and mast cell stabilizers was started with 300 mg omalizumab on monthly basis, Urticaria activity score started improving after second month. Patient is still using H1 antagonist but not regularly but with a liberal lifestyle as compared to previously.

Key words

Chronic spontaneous urticaria, omalizumab.

Introduction

Chronic spontaneous urticaria (CSU) is more common in adults, especially middle-aged women. The condition resolves spontaneously within 6 months in 30 to 55% of patients but can persist for years in others. Besides its physical symptoms, CSU has a devastating effect on the quality of life of patients. According to the according to the latest EAACI/GA2LEN/EDF and WAO guidelines, second-generation non-sedating antihistamines are the first-line therapy of CSU. However, 50% of patients of CSU do not respond to antihistamines. The latest EAACI/GA2LEN/EDF and WAO guidelines recommend the use of omalizumab in the management of refractory CSU. Omalizumab is a recombinant humanized anti-IgE monoclonal IgG antibody. We here report a case of CSU not responding to conventional treatment including antihistamines and oral prednisolone.

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Case Report

A 52-year-old male retired from military services, currently working in administrative services at a fertilizer plant, developed generalized wheals and angioedema 15 years back after having some seafood. Symptoms settled with injectable steroids and anti-histamines. Symptoms reappeared with similar severity after an interval of 1-2 months without specific association to food. Later, the patient started having regular complaint of itching and appearance of hives on face, head and arms apparently unrelated to any triggering factor. He required regular medication including multiple antihistamines, mast cell stabilizers and oral steroids occasionally. Skipping medication for 2-3 days resulted in recurrence of symptoms.

The patient had allergic rhinitis for past 3-4 years for which he was using regular nasal decongestants. There was no history of any other allergic manifestation. Patient was also using statin, aspirin, beta-2 antagonist and alpha-1 antagonist. There was no family history of urticaria. He regularly smoked 20-25 cigarettes a day.

On examination of skin, there were urticarial wheals of variable sizes and shapes on trunk and limbs. However, angioedema was not seen. No abnormality was detected on general and systemic physical examination. On clinical grounds, the diagnosis of CSU was made.

Considering the chronicity and poor therapeutic response of disease, omalizumab treatment was planned. Before starting omalizumab, investigations revealed serum IgE level of 1800 IU/dl. Omalizumab, 300 mg subcutaneously was started and repeated after every four weeks. His previous medications oral cetirizine 10 mg/day in morning and hydroxyzine 25 mg/day in evening along with montelukast sodium 10 mg/day along with xylometazoline nasal drops were continued. UAS7 and CU-Q2 oL were used to assess the efficacy of of omalizumab.

At the beginning of the treatment the patient's CU-Q2oL index was 67 and the UAS7 for the preceding 7 days was 30. Symptoms started settling within second week of giving omalizumab treatment. Patient started skipping his regular medication. After 2nd dose, patient stopped all his medication at his own. Symptoms started recurring but with less severity. He was restarted cetirizine and montelukast.

After 4 weeks the CU-Q2oL was 42 and UAS7 was 18. At 8 weeks the CU-Q2oL was 41 and UAS7 was 17. At 10 Weeks of starting treatment CU-Q2oL was 38 and UAS7 was 13 (**Figure 1**). At 14 weeks after starting Omalizumab and giving 3 shots of 300 mg each, CU-Q2 oL was 30 and UAS7 was 4. Montelukast was stopped at 18th week. He received 6 injections of omalizumab 300 mg every 4 weeks. Further 6 injections of omalizumab 150 mg were given at an interval of 4 weeks. Patient was without any other medication and enjoyed a normal lifestyle. He required cetirizine occasionally (1-2 times a week). Symptoms of

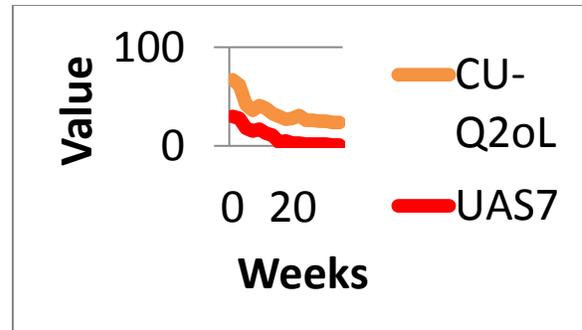


Figure 1 Effect of omalizumab on UAS7 and CU-Q2 oL.

allergic rhinitis also improved and he was less dependent on xylometazoline nasal drops. During the treatment period, no clinical or laboratory adverse effects were seen.

Discussion

Omalizumab, a humanized IgG1 κ anti-IgE antibody has been successfully used in CSU and angioedema.^{1,2,3} It is now well established that this anti-IgE antibody may be useful in patients with chronic urticaria.

According to EAACI/GA2LEN/ EDF and WAO guidelines, the first-line drugs for the treatment of chronic urticaria are antihistamines. If the standard dose is not effective in two weeks, the dose can be increased four-fold.⁴ However, a few cases may still not respond to therapy. It has been speculated that the reduction in IgE levels due to treatment with anti-IgE-IgG-antibodies may lead to a secondary downregulation of the Fc ϵ RI on mast cells.³

In our patient of CSU, the disease was there for last 15 years and not being controlled by regular use of antihistamines, montelukast and periodic intake of oral steroids. Omalizumab was effective in this difficult to treat patient. Response started after two weeks and progressive improvement in terms of UAS7 and CU-Q2 oL continued and after 48 weeks the disease severity was well under control. No

drug-related adverse effects were noticed during this period.

Omalizumab may be used in the treatment of severely affected patients with refractory CSU. Initially a higher dose is needed to control the symptoms and for sustenance of control lower dose may be continued. The drug is also well tolerated.

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