Letter To Editor

Aspirin desensitization in samter's triad

Introduction

Samter's triad (ST) is a syndrome characterized with asthma, nasal polyposis (NP) and rhinosinusitis, in which the patients are unable to tolerate aspirin and aspirin-like drugs. NP often accompanies the existing disease in patients with aspirin intolerance (AI).[1] Topical and systemic steroids are used for the treatment of rhinitis and polyps. Antihistamines and chromons do not seem to have a significant effect in treatment. Cases not responding to this treatment then undergo polypectomy, although it has a recurrence rate of 40% after the surgery.[1,2] Significant clinical improvement has been observed at lower and upper respiratory tract inflammation following the daily determined amount of aspirin intake desensitization in a few of the available studies.[2] With this study, we aimed to prove the clinical benefits of aspirin desensitization treatment for two cases with ST in concordance with the current literature.

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Case 1

A 34-year-old female patient diagnosed with asthma was receiving regular treatment. Past medical history of the patient includes AI with nasal polyposis for which she has undergone various medical treatments and polypectomy four times. Since the patient had recurrence and often exacerbation of her nasal and asthma symptoms, and since she had limited response to applied medical treatments, the patient had undergone aspirin desensitization treatment according to the determined protocol, following the informed consent obtained from the patient. 20 minutes after every dose, the patient was evaluated by clinical examination findings and pulmonary function tests (PFT), and the findings were recorded. After the final aspirin dose, the patient presented significant improvements of nasal symptoms and stabilization of asthma symptoms. The followed aspirin desensitization protocol, PFT and clinical findings are presented in (Table 1).

Table 1 Desensitization protocol, symptoms, and reactions of the cases (Case 1 / 2)

Day	Time	Administered ASA (mg)	Nasal symptom and reaction	Asthma Symptoms	% of decline in FEV1
1	8:00	Placebo	+/+	0/+	0/0
	11:00	Placebo	+/+	0/+	0/0
	14:00	Placebo	+/+	0/+	0/0
2	8:00	30	++ / +	+/+	0/6
	11:00	60	++++ / ++	++ / ++	14 / 18
3	8:00	60	+++ / +++	++ / ++	22 / 8
	11:00	100	++++ / +++	++ / +++	13 / 17
4	8:00	100	++ / +	+/+	16 / 18
	11:00	150	0/+	0/0	0 / 11
	14:00	325	0/0	0/0	0/4
5	8:00	650	0/0	0/0	0/0

ASA, Acetylsalicylic acid; FEV 1, Forced vital capacity in 1 second

Case 2

A 32-year-old female patient was admitted with a history of three polypectomy operations for nasal polyposis and asthma attack associated with aspirin and NSAID use. The patient, who did not benefit from local and systemic treatments for her nasal complaints, gave informed consent and underwent aspirin desensitization treatment according to the determined protocol. The patient was evaluated during and after the treatment, and significant improvements were observed with her nasal symptoms. The followed aspirin desensitization protocol, PFT and clinical findings are presented in (Table 1).

Airway inflammation is observed with nasal polyposis and rhinosinusitis in aspirin-sensitive asthma. The asthma of these patients is prone to cause treatment problems and requires seldom systemic steroid intake. Aspirin inhibits the cyclooxygenase pathway and the arachidonic acid methabolism shifts toward lipoxygenase.

This may cause local inflammation by activation of eosinophils and mast cells.[3] Kowalskive et al. reports that mechanisms of apoptosis are deteriorated in NP and inflammatory cells of respiratory mucosa in patients with ST; and because of that, local inflammatory mechanisms have differentiated.[4]

AI causes the NP to be more common, both in a clinical and radiologic way. Many articles state that AI is considered as a risk factor for recurrence and many patients require repetitive revision surgeries. The presence of asthma negatively affects the severity of NP. Also, asthma is considered as a risk factor in the recurrence of NP.[5] Significant clinical improvements have been observed at lower and upper respiratory tract inflammation in these cases following the daily-determined amount of aspirin intake desensitization in a few studies available in the literature.[2] The relationship between aspirin desensitization and a decline in mediator release was also supported with the biochemical data. Aspirin-sensitive asthma patients could be desensitized against aspirin with a careful oral administration of progressive doses. While there is not an absolute determined dose for desensitization, various researchers used various doses, protocols and times.[6] Following a reaction against a particular dose, recovery is observed within 2 to 24 hours. In conclusion, as a result of aspirin desensitization treatment, we achieved clinical recovery and significant improvements in the asthma and nasal symptoms of two cases with ST; also we suggest that aspirin desensitization should be considered alongside with other medical treatment options for ST patients with NP.

Acknowledgments

None.

Conflicts of interest

Author declares that there is no conflict of interest.

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