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Investigation of the Frequency of Angioedema and Cough in Children Using Angiotensin Converting Enzyme Inhibitors

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ABSTRACT

Objective: Although ACE inhibitor-induced angioedema is a well-documented adverse effect in adults, there is a paucity of data on its development and prevalence in children, except case reports. This study aimed to investigate the frequency and clinical course of angioedema and cough in pediatric patients taking ACE inhibitors for hypertension, chronic renal failure, and proteinuria.

Materials and Methods: The study population comprised pediatric patients using ACE inhibitors due to hypertension, chronic kidney disease, and proteinuria, who were treated at the Ankara Bilkent City Hospital Pediatric Nephrology Clinic between 1 January 2022 and 1 April 2022.

Results: The study included 357 patients with hypertension, of whom 157 were pediatric patients on ACE inhibitors. Two patients (1.7%) developed acute angioedema with tongue, lip, and facial involvement after chronic use of ACE inhibitors. No cough was observed in any of the patients on ACE inhibitors.

Conclusion: Angioedema as a result of antihypertensive drugs in pediatric populations is a rare but potentially life-threatening condition. In the event of angioedema developing in patients undergoing treatment with ACE inhibitors, it is of the utmost importance to be mindful of this and to cease the administration of the drug in question immediately.

Keywords: ACE inhibitors, angioedema, children

INTRODUCTION

The prevalence of hypertension in children and adolescents is 3.5% (1). The objective of treatment for hypertension is not only to lower blood pressure but also to prevent the development of cardiovascular diseases in adulthood. End-organ damage can be prevented with appropriate treatments (2). Patients whose hypertension cannot be controlled despite lifestyle changes should be started on antihypertensive medication (3).

It is important for pediatricians to recognise and manage the adverse effects associated with antihypertensive drugs. Angiotensin converting enzyme (ACE) inhibitors are known to cause angioedema in adults. When ACE is inhibited, the breakdown of bradykinin is reduced and the increase in bradykinin causes vasodilation and an increase in capillary permeability in the mucosal or submucosal vessels of various tissues. Therefore, edema occurs in the tissues as intravascular fluids leak locally into the interstitial space (4).

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Angioedema manifests in the deep dermal layer of the skin and submucosal tissues and is characterised by asymmetry, localised distribution, self-limiting nature, absence of pruritus, lack of urticaria, non-inflammatory presentation, and non-marking (5). ACE inhibitor-induced angioedema typically presents with isolated angioedema of the lips, tongue, and face, and rarely the larynx; abdominal attacks are rare, while limb attacks are not expected (6). It usually persists for 2-5 days. Attacks involving the respiratory tract can lead to asphyxia if they are not treated in the right way (7). Patients may not recognise this progression, but clinicians must be vigilant and initiate appropriate treatment as soon as possible. 0.3% of adults using ACE inhibitors have been shown to develop angioedema (8).

It has been reported that between 5 and 20% of patients using ACE inhibitors develop dry cough (9,10). The precise mechanism by which ACE inhibitors cause cough is currently unknown. However, kinins and substance P are metabolised by converting enzymes. The enzyme in question has been demonstrated to increase in activity when inhibited. Consequently, an increase in prostaglandin production results in bronchial irritation and the development of cough (10). It has been established that ACE inhibitor-induced cough only occurs in susceptible individuals, irrespective of the dosage of the drug. Consequently, it is an idiosyncratic reaction. By the revised terminology, it is a non-immune type B hypersensitivity reaction and is one of the well-defined side effects of ACE inhibitors (11,12). The incidence of cough in children receiving ACE inhibitors is 3.2% (13). Reports of cough were lower among children than adults.

Although ACE inhibitor-induced angioedema and cough are well-documented complications in adults, there is a paucity of research examining their development and frequency in children, except case reports. This study aimed to investigate the prevalence and clinical course of angioedema and cough in pediatric patients taking ACE inhibitors for hypertension, chronic renal failure, and proteinuria.

MATERIAL and METHODS

The study population comprised pediatric patients using ACE inhibitors due to hypertension, chronic kidney disease, and proteinuria in the Pediatric Nephrology Clinic of Ankara Bilkent City Hospital between 1 January 2022 and 1 April 2022. The demographic characteristics of the patients, as well as their history of chronic diseases, diagnostic tests, hospitalisation, and other treatments, were recorded. The age, gender, history of angioedema, duration of symptoms, and response to treatment of the patients were monitored. The names of the drugs, duration of use, and complaints at the presentation of patients using ACE inhibitors were emphasised.

Diagnosis of Angioedema

The diagnosis of antihypertensive angioedema is made by excluding other conditions that may cause upper airway obstruction. Allergic reactions, anaphylaxis, drugs, chronic urticaria, hereditary angioedema, bacterial and viral infections, autoimmune diseases, hypereosinophilic syndrome and urticarial vasculitis may cause angioedema. The use of drugs that may cause angioedema (NSAIDs, hormonal drugs, DDP-4 inh, neutral endopeptidase inh, aminopeptidase inh, plasminogen activators, streptokinase and urokinase) was excluded. The presence of urticaria and the degree of angioedema were assessed by skin examination. Angioedema that developed in patients without pruritus and urticaria and an anaphylaxis clinic was considered to be bradykinergic mediated. It was thought that these patients may present as a first episode of hereditary angioedema or may develop angioedema due to medication use.

In patients presenting with angioedema, the function and mass levels of C4 and C1 inhibitors were studied during the attack to exclude hereditary angioedema in patients who were thought to have bradykinin-mediated angioedema based on history and physical examination.

As ACE inhibitor-induced cough is dry and usually develops within the first 2 weeks of starting ACE inhibitors, patients were asked during clinical follow-up if they had cough complaints, and patients with cough before starting ACE inhibitors were excluded. Children who developed a cough while taking ACE inhibitors were asked about conditions that may cause cough more often, such as previous upper respiratory tract infections, asthma, reflux, and postnasal drip. Children with these diagnoses were excluded from the study.

RESULTS

A total of 357 patients with hypertension were included in the study, of whom 157 were pediatric patients on ACE inhibitors. The remaining 200 patients, who used angiotensin receptor blockers, calcium channel blockers, diuret-

Table I: Demographic characteristics of patients using ACE inhibitors.

Demographic features	
Gender (male), n (%)	106 (67.5)
Age (year) median	15.3 (min:2- max:18, SD:4.1)
Obesity, n (%)	59 (37.5)
Duration of ACE inhibitor use	14.5 month (min:1 week- max:108 month SD:16.2 month)
Family history of hereditary angioedema	None
History of angioedema with medications	None

ics, beta-blockers, and alpha agonists but had never used ACE inhibitors, were not included in the study.

Of the total number of patients, 106 were male (67.5%), with a median age of 15.3 years (ranging from 2 to 18 years). A total of 59 patients (37.5%) were identified as obese. The mean duration of ACE inhibitor use was 14.5 months, with a minimum of one week and a maximum of 108 months. No patients had a family history of hereditary angioedema or drug-induced angioedema (Table I).

Among the patients who were taking ACE inhibitors, there were no cases of cough. Two patients were switched over to calcium channel blockers due to renal dysfunction, and at the time of the follow-up, they reported a regression of their symptoms.

Two patients (1.7%) developed acute angioedema with tongue, lip, and facial involvement following chronic use of ACE inhibitors. The potential causes of angioedema, including infection, anaphylaxis, other drug use, and hereditary angioedema, were excluded. Symptoms improved in two patients within approximately two days after changing antihypertensive treatment. There were no further episodes of angioedema during the follow-up period.

A total of 10 patients (6.3%) had been diagnosed with asthma by a physician, 6 patients (3.8%) had been diagnosed with allergic rhinitis, 1 patient (0.6%) had a food allergy, 1 patient (0.6%) had atopic dermatitis, and 1 patient (0.6%) had a drug allergy.

Patient 1

A 16-year-old female was being monitored for several health conditions, including non-atopic asthma, non-allergic rhinitis, obesity, insulin resistance, hypertension, obsessive-compulsive disorder, and attention deficit hyperactivity disorder. The patient sought emergency department care on two occasions within the previous month, citing swelling of the face and tongue as the primary presenting symptom. The patient presented no symptoms compatible with infection and no known food or drug allergies. There was no family history of angioedema or allergy. The patient had been using inhaled ciclesonide for asthma, metformin for insulin resistance, enalapril for hypertension for six years, and sertraline 150 mg for obsessive-compulsive disorder. The last dose of enalapril was taken 10 hours ago.

Upon arrival at the emergency room, the patient's vital signs were found to be stable. She developed angioedema on her face and tongue, but there was no evidence of urticaria or respiratory distress. Lung sounds were normal bilaterally. Immediate administration of methylprednisolone 40 mg and IV diphenhydramine was undertaken in the emergency department. The patient was stabilised and transferred to the observation unit. However, given the persistence of the patient's symptoms, a comprehensive investigation was conducted to exclude the possibility of hereditary angioedema. This entailed the assessment of C4 and C1 inhibitor function and mass. Complement levels were within the normal range. The patient's angioedema regressed within 72 hours. Pediatric Nephrology was consulted, enalapril was discontinued, and amlodipine was initiated. Angioedema did not recur during follow-up. An enalapril provocation test was recommended to the family for diagnosis; however, the family declined the test.

Patient 2

A 15-year-old female patient was being monitored for morbid obesity, non-alcoholic fatty liver disease, gallstones, irritable bowel syndrome, insulin resistance, and hypertension. Facial and lip swelling developed 1.5 hours after the administration of flurbiprofen for the treatment of a toothache and 18 hours after the administration of enalapril. The patient had been using metformin for one year to treat insulin resistance, enalapril for hypertension

for one year, and lansoprazole for gastroesophageal reflux. The patient was administered 50 mg methylprednisolone in the emergency department, yet no response was observed. There were no known food or drug allergies. She did not report any signs of infection in her history. There was no family history of hereditary angioedema or allergy. C4 and C1 inhibitor function and mass sent to the emergency department with the complaint of angioedema resulted in normal values.

The patient underwent a diagnostic oral provocation test. She developed angioedema of the lips 17 hours after being administered enalapril under supervision in an allergy clinic. Vital signs remained stable, and the patient exhibited no additional symptoms. The patient was monitored under observation, and the angioedema regressed. Enalapril was discontinued, and amlodipine was initiated. No further episodes of angioedema occurred during the follow-up period.

Given the patient's history of flurbiprofen intake, an oral provocation test was conducted with paracetamol, meloxicam, and aspirin. No reaction was observed during or after the test. Consequently, the patient's lansoprazole was discontinued. The test was repeated with esomoprozole, which has a distinct side-chain reaction profile.

Both patients continued to take metformin, and no angioedema was observed despite this. The characteristics of the patients are presented in Table II.

DISCUSSION

Angioedema due to ACE inhibitors is a well-documented phenomenon in adults. However, there is a paucity of research in the pediatric population. Only case reports have been published. Of the 157 patients who were prescribed ACE inhibitors, two developed angioedema.

The development of angioedema can be mediated by histamine and bradykinin. As the mechanism of these two types is different, the treatment is also different. Because angioedema can cause sudden collapse of the upper airway, accurate diagnosis and prompt treatment are important. The differential diagnosis with anaphylaxis should be carefully considered. Hereditary angioedema, acquired angioedema, and ACE inhibitor-associated angioedema are bradykinin-mediated angioedemas. In these conditions, an increase in bradykinin causes vasodilation and increased permeability of vessels in the mucosa or submucosa of various tissues. As a result, intravascular fluids leak locally into the interstitial space (4).

In a study of children with angioedema without urticaria, infection was found in 21% of patients, allergy in 14%, thyroid autoimmunity in 8%, NSAID in 6%, and no cause was found in 51% of patients (14). If angioedema in children is associated with itching and/or urticaria, it is most likely histaminergic (allergic) (15). 20% of patients with chronic spontaneous urticaria have angioedema as the only symptom (16). Hereditary angioedema is a rare

Table II: Features of two cases.

	Patient 1	Patient 2
Age	16	15
Gender	F	F
ACE inhibitor	Enalapril	Enalapril
Duration of ACE inhibitor use	6 year	1 year
Frequency of angioedema	Twice in the last month	1 time at home and 1 time after drug provocation test
Recurrence of angioedema after drug withdrawal	Did not happen	Did not happen
Antihistamine and steroid response	No	No
Family history for hereditary angioedema	No	No
C4 level	Normal	Normal
C1q inhibitor function	Normal	Normal
Drug provocation test	Not done	Verified
Other drug use	Metformin	Flurbiprofen, aspirin drug provocation test used
Other diseases	Non-atopic asthma, obesity, insulin resistance	Obesity, insulin resistance

autosomal dominant genetic disorder caused by a mutation in the C1-INH gene (SERPING 1); in both types of the disease, the clinical picture is caused by elevated levels of bradykinin (17). Acquired angioedema is an acquired C1-INH deficiency due to increased consumption of C1-INH, such as in lymphoproliferative diseases. It is extremely rare in children (18). Bacterial and viral infections, hypereosinophilic syndrome, urticarial vasculitis, and autoimmune diseases can also cause angioedema (14,15,19).

The diagnosis of antihypertensive angioedema is made by excluding other conditions that may cause upper airway obstruction. ACE inhibitor-induced angioedema accounts for approximately 30% of acute angioedema cases in the USA (20). In a 10-year retrospective study conducted in France, 88 (77%) patients who met the diagnosis of bradykinin-mediated angioedema developed ACE-induced angioedema (21). No studies have been conducted on children, and case reports have been available (22-24).

This may be overlooked by clinicians because patients have been taking antihypertensive drugs for years. This can lead to delays in stopping the drugs responsible for the patient's problem. Our 15-year-old female patient had been on ACE inhibitors for 1 year and our 16-year-old female patient had been on ACE inhibitors for 6 years. Although most patients develop angioedema within the first week of taking the drug, cases have been reported after prolonged use (25).

When treating angioedema due to ACE inhibitors, the airway should be stabilised and the suspected drug should be stopped. The swelling will begin to resolve within 48–72 hours of stopping the drug. If swelling occurs in the oropharynx, floor of the mouth, or tongue, sudden airway obstruction should be considered. There may be no response to treatment with antihistamines, corticosteroids, and adrenaline. As histamine-mediated angioedema is more common, antihistamine and corticosteroid treatment may be used primarily in patients with angioedema alone (25). If there is no response to antihistamines and corticosteroids, bradykinin-mediated angioedema should be considered. Angioedema has been reported to resolve in 24-48 hours and can last up to 5 days if left untreated (26). In our patients, the angioedema resolved within a median time of 56 hours.

The development of angioedema in a patient should be evaluated for other conditions that may cause angioedema and other medications used before ACE inhibitors are associated. Other possible causes were excluded in our patients. When evaluating the medications used, it was noted that the second patient was using NSAIDs, which are known to cause angioedema (27). The patient had taken flurbiprofen for toothache 1.5 hours before the onset of angioedema. In cases where more than one drug is suspected, oral provocation tests are used to find the drug responsible. As our patient had hypertension and was on enalapril, an enalapril provocation test was performed in our clinic. This was followed for 6 hours and no acute reaction was observed. The patient was then discharged. However, he was admitted to the emergency department again on the 17th hour with complaints of recurrent angioedema. This was evidence of Enalapril-induced angioedema.

Both of our patients who developed angioedema were being treated for obesity. Obesity is an important cause of hypertension, and its increasing prevalence is thought to be associated with an increasing prevalence of hypertension in children (28). Both of our patients were taking metformin for therapeutic purposes, but angioedema did not recur after discontinuing the ACE inhibitor, although they continued their metformin treatment.

A wide variety of drugs are used to treat hypertension in children. ACE inhibitors, angiotensin receptor blockers (ARBs), and long-acting calcium channel blockers are preferred when starting treatment (3). Angioedema develops in a dose-independent manner with ACE inhibitors (19). Calcium channel blockers and angiotensin receptor blockers can also cause angioedema (29,30). If angioedema develops as a result of ACE inhibitor use and antihypertensive medication is required, a switch to a different class of medication should be made. Our patients were switched to calcium channel blockers and did not develop angioedema during follow-up.

None of our patients developed cough associated with ACE inhibitor use. Previous studies have shown that cough occurs in 3.2% of children and 11% of adults (9,13). Baker Smith et al analysed randomised controlled trials and found no significant increase in the incidence of cough associated with ACE inhibitor and ARB use (13).

The most common causes of chronic cough in children are asthma, persistent bacterial bronchitis, and nonspecific cough (31). These causes should be investigated first in children with prolonged cough. Cough due to ACE inhibi-

tor use typically develops within the first two weeks after initiation of treatment, is more frequent in women, may improve within four days after discontinuation of treatment, and recurs when ACE inhibitors are used again (32).

Several risk factors have been identified as potential causes of ACE inhibitor-associated cough in adults, including increasing age, female gender, congestive heart failure, East Asian ethnicity, and smoking (33,34). It is still unclear why some people who take ACE inhibitors develop a cough and others do not. Genetic factors are thought to play a role.

CONCLUSION

The use of ACE inhibitors should be subject to questioning, particularly in cases of tongue, lip, and facial angioedema. Angioedema due to antihypertensive drugs in pediatric age groups is a rare but potentially life-threatening condition. Angioedema due to ACE inhibitors is unpredictable. In some cases, angioedema may progress and cause sudden airway obstruction. As the prevalence of hypertension in children increases, so too will the use of ACE inhibitors. It is therefore crucial that all doctors, particularly those working in emergency services, are aware of this issue. When angioedema develops in patients taking ACE inhibitors, it is of the utmost importance to discontinue the drug.

Conflict of Interest

The authors have no conflicts of interest to declare.

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Authorship Contributions

Concept: **Emine Dibek Misirlioglu,** Design: **Emine Dibek Misirlioglu,** Data collection or processing: **Deniz Yılmaz, Elif Benderlioğlu,** Analysis or Interpretation: **Emine Dibek Misirlioglu,** Literature search: **Deniz Yılmaz, Emine Dibek Misirlioglu,** Writing: **Deniz Yılmaz,** Approval: **Sare Gülfem Özlü, Umut Selda Bayrakçı, Emine Dibek Misirlioglu.**

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