*Cardiology in the Young*

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# Investigation of the presence of atopy in children visiting the paediatric cardiology department due to chest pain

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## Original Article

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## Abstract

*Background:* The cases of chest pain in children are usually not of cardiac origin. *Objectives:* To investigate asthma and other atopic diseases in children with chest pain not of cardiac origin. *Patients and Methods:* Children aged 6–18 years who were seen for chest pain were included in the study. Haematologic parameters, pulmonary function tests, and skin prick tests were performed. Atopic diseases and environmental factors were investigated. *Results:* The non-cardiac chest pain group (Group 1) included 88 children (female: 53.4%) with a mean age of 11.9 ± 3.4 years; the control group (Group 2) included 29 children (female: 53.8%) with a mean age of 11.4±2 years ($p > 0.05$). A family history of atopy (22.7%) and skin prick test positivity (28.4%) was more common in Group 1 than Group 2 ($p = 0.006$ and $p = 0.017$, respectively). The rate of presence of all environmental factors except stove use and mould was significantly higher in Group 1 (54.5%) than Group 2 (3.4%) ($p < 0.001$). Asthma was diagnosed in 44.3% and allergic rhinitis in 9.1% of patients in Group 1. Idiopathic chest pain, musculoskeletal system disorders, gastroesophageal reflux, and pneumonia were identified in 23.9%, 11.4%, 8%, and 3.4% of patients in Group 1, respectively. *Conclusions:* In this study, the most common cause of non-cardiac chest pain was asthma. The local prevalence of asthma is higher than normal, and this may have affected the results of this study. A detailed history and physical examination will accurately establish the cause of chest pain in most children.

Chest pain is the second most common reason for referral to a paediatric cardiologist after murmur.[1,2] However, these cases of chest pain are generally not of cardiac origin.[1–3] Chest pain due to cardiac disorders was reported to account for less than 4% of the total chest pain cases.[1,4,5] In order to find the main cause of the pain, the patient's complaints should be investigated in detail. It was reported that chest pain in children is most often idiopathic. However, many pathologies, such as musculoskeletal system and respiratory system disorders, also result in chest pain in children.[1,4] Chest pain and chest tightness are self-limiting conditions that can occur with respiratory system diseases as well as during asthma attacks.[6,7] Asthma is one of the most common chronic respiratory diseases in children, occurring with different symptoms.[8] It is characterised by airway obstruction, airway hypersensitivity, shortness of breath, chest tightness, and cough.[9]

The main aim of this study was to examine the presence of asthma and other atopic diseases in patients with chest pain not of cardiac origin together with their clinical findings.

## Patients and methods

This study was approved by the local ethics committee (study identification number: E-46418926-050.01.04—69151) and was conducted between June 2021 and January 2022 in accordance with the ethical standards specified in the 1964 Declaration of Helsinki. All patients or their parents gave informed consent to participate. Patients aged 6–18 years who were examined in the paediatric cardiology department with the complaint of chest pain were included in the study. Patients with chest pain determined to be of cardiac origin after detailed physical examination, and 12-lead electrocardiography and echocardiographic examinations were excluded from the study. Patients with non-cardiac chest pain were referred to the paediatric allergy and immunology department.

After physical examination and a detailed anamnesis in terms of atopic diseases in the paediatric allergy and immunology department, complete blood count, total immunoglobulin E levels, pulmonary function tests, and skin prick tests were evaluated. Haemoglobin, haematocrit, mean corpuscular volume, white blood cell count, eosinophil count, and platelet count were obtained from the complete blood count results.

All patients and their families were questioned about asthma, other atopic diseases, and the presence of environmental factors in the home (smoking exposure, presence of pets, stove use, wool use, and mould). An increase in allergic respiratory diseases such as asthma can be seen in

those who are exposed to wool.[10] Some of the reasons for this are thought to be due to endotoxin, an inflammatory lipopolysaccharide and parasites such as Dermestidae that can be highly accumulated in wool materials, which are asthma triggers.[10,11] Therefore, in this study, the use of wool at home was questioned in terms of the risk of asthma. Asthma was diagnosed and treated with reference to the Global Initiative for Asthma protocols,[8] according to which the diagnosis of asthma is based on variability in respiratory symptoms, hypersensitivity in respiration, and reduced forced expiratory volume in 1 second. Spirometry results may sometimes not be consistent with asthma. In such cases, the diagnosis of asthma was made based on signs of improvement after treatment.[8,12,13]

All patients underwent skin prick tests with inhalant allergens (grass pollens, tree pollens, weed pollens, house dust mites, moulds, and animal dander) and food allergens (egg mix, cow milk, beef, chicken, soy flour, wheat flour, rice flour, and cacao).

Children who did not have chest pain or cardiac pathology, who were examined in the paediatric cardiology department due to participation in sports, and who agreed to participate in the study were included in the control group. The control group underwent electrocardiography and echocardiographic examinations after examination in the paediatric cardiology department. Those with chronic diseases other than atopic diseases and those who had a psychiatric disease at any time were not included in the study.

The Childhood Asthma Control Test was used to assess whether asthma was adequately controlled.[14] The level of asthma control was recorded as "controlled" or "poorly controlled" based on Childhood Asthma Control Test scores.

## Statistical analysis

Descriptive statistics for continuous variables were expressed as mean, standard deviation, and minimum and maximum values. Numbers and percentages were recorded for categorical variables. The Kolmogorov–Smirnov and Shapiro–Wilk normality tests were used to test the normality of the data. Statistical analysis of the data of the normally distributed variables was done with Student's *t* test for two independent groups and with the Mann–Whitney *U* test for those who did not show normal distribution. The Bonferroni correction was used to counteract the multiple comparisons problem. Categorical variables were compared with the chi-square test (Fisher's exact test). Results were given as mean ± standard deviation for normal data and median with minimum–maximum values for non-normal data. Statistical significance was accepted at the level of $p < 0.05$ with a 95% confidence interval. IBM SPSS Statistics 23.0 was used in all data analyses.

## Results

A total of 105 patients who were examined with chest pain in the paediatric cardiology department were initially included in this study. Three patients (2.9%) with mitral valve prolapse were excluded and 102 patients without cardiac problems were referred to the paediatric allergy and immunology department. Among these patients, four patients with chronic diseases, three patients with active systemic infections, and seven patients who did not attend follow-up appointments were also excluded (Fig. 1).

The non-cardiac chest pain group (Group 1) included 88 children (female: 53.4%) with a mean age of 11.9 ± 3.4 years; the control group (Group 2) included 29 children (female: 53.8%) with a mean age of 11.4 ± 2 years. There was no difference between the groups in terms of age or gender distribution ($p > 0.05$) (Table 1).

Haematologic parameters and total immunoglobulin E were not different between the groups ($p > 0.05$). In Group 1, 53 (60.2%) of the children who were able to perform pulmonary function tests had normal forced expiratory volume in 1 second and 18 (20.5%) had low values of forced expiratory volume in 1 second (60–80%). The results were normal for all of the children who were able to perform pulmonary function tests in Group 2. Lower values were significantly more common in Group 1 than Group 2 ($p = 0.014$) (Table 1).

There was a statistically significant difference between groups according to the environmental factors ($p < 0.001$). Post hoc analysis was conducted with a Bonferroni correction applied, resulting in a significance level set at $p < 0.005$, revealing that smoking exposure (36.4% versus 3.5%, $p = 0.0007$; <0.005), wool use (25% versus 0%, $p = 0.0029$; <0.005), and presence of a pet (23.9% versus 0% $p = 0.0038$; <0.005) were significantly higher in Group 1 compared to Group 2. There was no difference between the two groups in terms of the presence of mould and stove use ($p = 0.24$ and $p = 0.036$; >0.005, respectively).

In Group 1, there were 20 patients (22.7%) whose mother, father, or sibling had atopic disease. In Group 2, there was no family history of atopy. There was a significant difference between the groups in this respect ($p = 0.006$) (Table 1).

Skin prick tests were positive for 28.4% of patients in Group 1, which was a significantly higher rate compared to Group 2 (6.9%) ($p = 0.017$). Of the 25 positive patients, seven were sensitised to tree pollens, five to house dust mites, five to soy flour, three to grass pollens, three to cats, two to dogs, one to milk, and one to beef (Table 1).

When the patients in Group 1 were evaluated in terms of additional complaints in the paediatric allergy and immunology department, shortness of breath (40.9%) and cough (11.4%) were most common. Nasal congestion (9.1%), sneezing (8%), gastroesophageal reflux symptoms (8%), effort dyspnoea (5.7%), palpitation (5.7%), itchy nose (3.4%), and snoring (2.3%) were also seen to lesser extents (Table 2). When the histories of the patients were investigated, it was learned that 12 (13.6%) had asthma, 9 (10.2%) had allergic rhinitis, 7 (8%) had gastroesophageal reflux, and 17 (19.3%) had both allergic rhinitis and adenoidal hypertrophy at some point in their lives (Table 2). According to the detailed clinical histories and physical examinations of the patients, asthma and allergic rhinitis were diagnosed in 39 (44.3%) and 8 (9.1%) cases, respectively. Besides atopic diseases, idiopathic chest pain, musculoskeletal system disorders, gastroesophageal reflux, and pneumonia were identified in 23.9%, 11.4%, 8%, and 3.4% cases, respectively (Table 2). Among patients diagnosed with asthma and allergic rhinitis, while skin prick test positivity was found to be significantly higher in those with allergic rhinitis (5 of 8 patients with allergic rhinitis, $p = 0.027$, and 8 of 39 patients with asthma, $p = 0.68$), no difference was observed between them in terms of immunoglobulin E levels ($p > 0.05$).

Appropriate treatments were given to those with asthma, allergic rhinitis, gastroesophageal reflux, and pneumonia. In follow-up appointments after 1 month, it was observed that the complaints of all but five of the patients were completely resolved. Although the complaints of five patients (three with asthma, two with gastroesophageal reflux) were not resolved, they had decreased. Thirty-six patients given asthma treatment had "controlled asthma" and three patients were "poorly controlled" according to Childhood Asthma Control Test scores.

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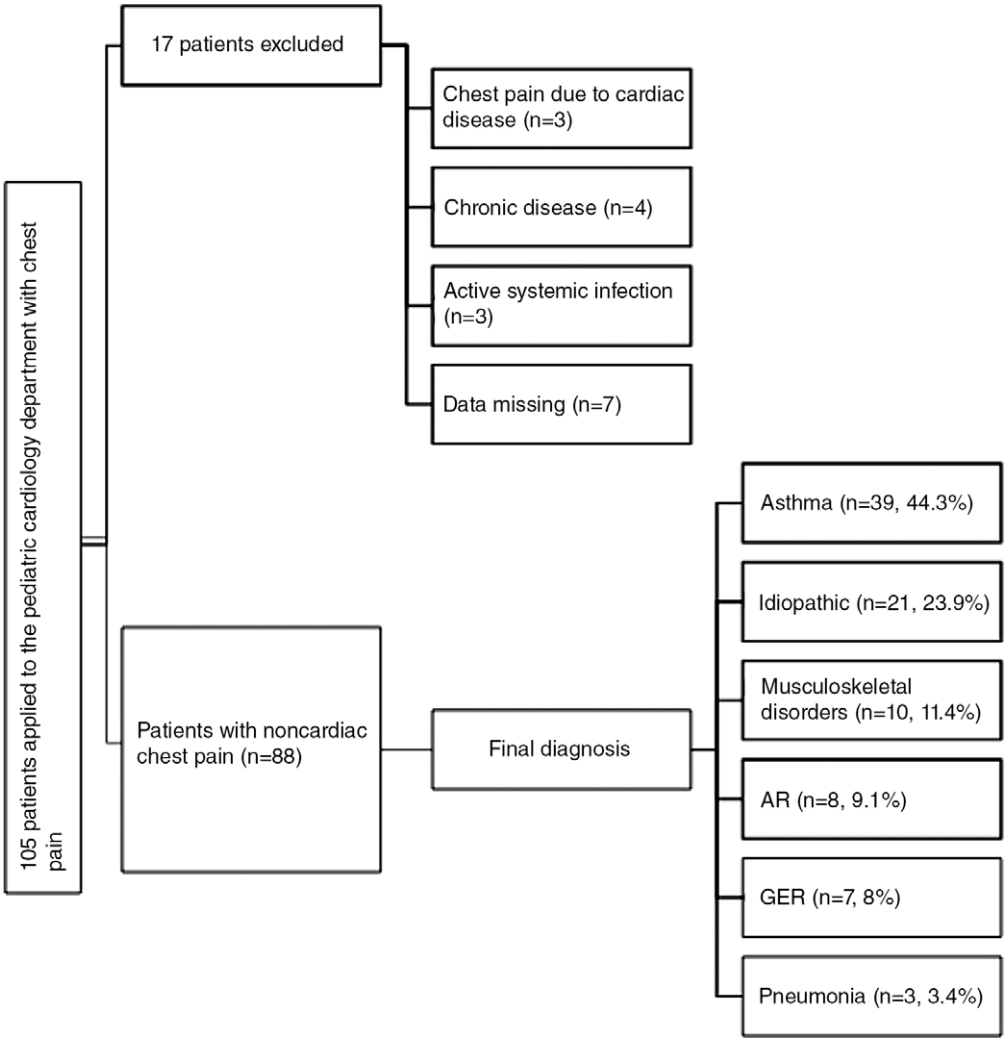


**Figure 1.** Flow chart regarding selection of patients.

## Discussion

In this study, non-cardiac chest pain in children was evaluated in terms of atopic diseases. It was found that approximately half of the patients had asthma and nearly 10% had allergic rhinitis. These findings have important implications because children may present to emergency departments and paediatric cardiology departments with complaints of chest pain.[1,4,5,15] The majority of patients and their families assume that the child's chest pain is of cardiac origin.[16] However, most cases of chest pain in children are not severe and the pain is not acute in nature.[17] In previous paediatric studies, cardiac-induced chest pain was found at rates lower than 4%.[1,4,5,15] Mitral valve prolapse thought to be of cardiac origin was detected in only three patients in this study. However, it is generally reported that mitral valve prolapse can be detected incidentally during echocardiography and will not cause chest pain.[4,13,18,19] Similarly, in our study, these cases may have been found incidentally as the cause of chest pain.

Many studies show variations in the prevalence of asthma.[20–22] One of the recent studies reported that the prevalence did not change in high-income countries, decreased in low-income countries, and increased in low-middle-income countries.[22] The factors associated with variations and increases in this prevalence are not fully understood yet. The low prevalence in low-income countries may be due to underdiagnoses and inadequate treatment of asthma. [22] Meanwhile, these differences may be explained also by the heterogeneity of genetic and environmental factors.[20,23] In addition, it is difficult to compare studies because of different methods that are used, various definitions for asthma, and different age groups in research.[23]

Between the years 2000 and 2003, International Childhood Asthma and Allergy Study Phase 3 was conducted.[24] Children from 25 countries and adolescents from 42 countries participated in the study. According to the results, the use of paracetamol, exposure to open-fire cooking, and early exposure to antibiotics in children may be associated with the causes of variations and increases in asthma prevalence.[24] Furthermore, there may also be increased awareness and better diagnosis of this condition.[20]

According to the International Study of Childhood Asthma and Allergy protocol, in previous years, more regions reported more frequent increases than decreases in asthma, rhinitis, and eczema.. In addition, the increase was more pronounced in regions where the prevalence was previously low, such as Latin America, Africa, and parts of Asia.[20] As a result, the percentage of children and adolescents reported to have asthma has increased significantly.[20] This increase in the mean worldwide prevalence of asthma was reported from 13.2% to 13.7% in adolescents and from 11.1% to 11.6% in children.[20]

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**Table 1.** Demographics, laboratory results, and risk factors of groups.

| Parameters | Group 1 (n = 88) n (%) | Group 2 (n = 29) n (%) | Overall (n = 117) Mean ±SD; median (minimum–maximum) | p |
|---|---|---|---|---|
| F/M n (%) | 47(53.4)/41 (46.6) | 16 (53.8)/13 (46.2) | | 0.87 |
| Age (year) (Mean±SD) | 11.9 ± 3.4 | 11.4 ± 2 | 11.8 ± 3.1; 12 (6–17) | 0.41 |
| Hb (g/dL) (Mean±SD) | 13.8 ± 1.3 | 13.4 ± 1.2 | 13.7 ± 1.3; 13.6 (10.8–18.2) | 0.25 |
| Hct (%) (Mean±SD) | 41.9 ± 3.6 | 40.6 ± 3.3 | 41.6 ± 3.5; 41.2 (34.2–52.1) | 0.08 |
| MCV (fL) (Mean±SD) | 82.1 ± 6.6 | 82.4 ± 5.1 | 82.2 ± 6.3; 83.3 (59.4–93.8) | 0.71 |
| WBC ($\times 10^3$/mm3) (Mean±SD) | 6.9 ± 1.8 | 6.6 ± 2.3 | 6.8 ± 1.9; 6.7 (10–16) | 0.41 |
| Eos (cells/μL) (Mean±SD) | 124.3 ± 80 | 139.3 ± 101.2 | 128 ± 85.2; 110 (20–380) | 0.74 |
| Platelet ($\times 10^3$/mm$^3$) (Mean±SD) | 279.4 ± 88.2 | 275.6 ± 85.7 | 278.2 ± 86.4; 220 (95–404) | 0.27 |
| Total IgE (U/L) (Mean±SD) | 197.1 ± 370.4 | 90.4 ± 79.8 | 150.1 ± 286.8; 76.6 (0.5–1722) | 0.40 |
| FEV1 | | | | 0.014* |
| > 80% | 53 (60.2) | 25 (86.2) | | |
| 60–80% | 18 (20.5) | – | | |
| Not cooperated | 17 (19.3) | 4 (13.8) | | |
| **Environmental factors** | 48 (54.5) | 1 (3.4) | | < 0.001# |
| Smoking exposure | 32 (36.4) | 1 (3.4) | | 0.001# |
| Wool use | 22 (25) | 0 (0) | | 0.003# |
| Pet | 21 (23.9) | 0 (0) | | 0.004# |
| Stove use | 12 (13.6) | 0 (0) | | 0.036 |
| Mould | 4 (4.5) | 0 (0) | | 0.24 |
| **Family history of atopy** | 20 (22.7) | 0 (0) | | 0.006α |
| Asthma in mother | 6 (6.8) 3 (3.4) | | | |
| Asthma in father | 4 (4.6) | | | |
| Asthma in sibling | 2 (2.3) | | | |
| AR in mother | 2 (2.3) | | | |
| AR in father AR in sibling | 1 (1.1) | | | |
| AD in sibling | 2 (2.3) | | | |
| **SPT positivity** | 25 (28.4) | 2 (6.9) | | 0.017β |
| Tree mix | 7 (7.9) | 1 (3.4) | | |
| HDM | 5 (5.7) | - | | |
| Soya flour | 5 (5.7) | - | | |
| Grass pollens | 3 (3.4) | 1 (3.4) | | |
| Cat | 3 (3.4) | - | | |
| Dog | 2 (2.3) | - | | |
| Beef | 1 (1.1) | - | | |
| Milk | 1 (1.1) | - | | |

AR=allergic rhinitis; AD=atopic dermatitis; Eos=eosinophil; F=female; FEV1=forced expiratory volume 1; HDM=house dust mites; Hb=haemoglobin; Hct=haematocrit; IgE=immunoglobulin E; M=male; MCV=mean corpuscular volume; p < 0.05=significant; SPT=skin prick test; WBC=white blood cell.
*Lower values of FEV1 were significantly higher in Group 1 than Group 2.
#Smoking exposure, wool use, presence of pet, and stove use were significantly higher in Group 1 than Group 2.
α In Group 1, smoking exposure, wool use, presence of pet, and stove use were significantly higher than Group 2.
β In Group 1, positive SPT was significantly higher than Group 2.

Established in 2012, the Global Asthma Network identifies worldwide trends in asthma prevalence. It also provides comparisons with data from the International Childhood Asthma and Allergy Study.[22] A Global Asthma Network Phase 1 study was conducted between 2015 and 2020 to determine the prevalence of asthma in adolescents and children.[22] In total, 74,361 adolescents from 27 centres and 45,434 children from 19 centres were included in the study. Centers that participated in the study

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**Table 2.** Complaints, past history, family history, and final diagnosis of Group 1.

| Parameters | | |
|---|---|---|
| **Complaints** | n | % |
| Shortness of breath | 36 | 40.9 |
| Cough | 10 | 11.4 |
| Nasal congestion | 8 | 9.1 |
| Sneeze | 7 | 8.0 |
| GER symptoms | 7 | 8.0 |
| Effort dyspnoea | 5 | 5.7 |
| Palpitation | 5 | 5.7 |
| Itchy nose | 3 | 3.4 |
| Snoring | 2 | 2.3 |
| **Past history** | n | % |
| Asthma | 12 | 13.6 |
| AR | 9 | 10.2 |
| GER | 7 | 8 |
| AR with adenoidal hypertrophy | 17 | 19.3 |
| **Final diagnosis** | n | % |
| Asthma | 39 | 44.3 |
| Idiopathic | 21 | 23.9 |
| Musculoskeletal disorders | 10 | 11.4 |
| AR | 8 | 9.1 |
| GER | 7 | 8.0 |
| Pneumonia | 3 | 3.4 |

AD=atopic dermatitis; AR=allergic rhinitis; GER=gastroesophageal reflux.

consist of 14 countries in the Africa-Eastern Mediterranean, America, Europe, and Southeast Asia-West Pacific regions. The prevalence of current wheeze averaged 10.4% in adolescents (ranging from 0.9% in New Delhi, India to 21.3% in Cape Town, South Africa,) and an average of 9.9% in children (ranging from 1.1% in Lucknow, India to 23.2% in Costa Rica). When the prevalence in adolescents was analysed by regions, the total asthma prevalence was reported as 15.1%, 11.9%, 15.2%, and 5.8% in Africa-Eastern Mediterranean, America, Europe, and Southeast Asia-West Pacific regions, respectively. In children, the prevalence of current wheeze in the Africa-Eastern Mediterranean, America, Europe, and Southeast Asia-West Pacific regions was reported as 10.8, 12.5, 11.2 and 7.4%, respectively. In addition, an increase in prevalence of current wheeze in Africa-Eastern Mediterranean and a decrease in South-East Asia-Western Pacific regions were reported.[22]

In our country, Turkey, the research has been conducted using different methods with different age groups.[25–27] The prevalence of asthma has been reported as 9.1–15% in all regions of the country.[25] In previous years, it was reported as 12.9% in 2008 in the city of Denizli, located in the western part of Turkey,[26] and 11.9% in 2012 in the city of Erzurum, located in the east.[27] There has been also a slight increase in prevalence over time in some areas.[25] Two International Study of Childhood Asthma and Allergy studies conducted in Denizli reported that the prevalence of asthma increased from 2.1% to 12.9% in 2008 compared to 2002 in children aged 13–14.[26] In studies conducted in Ankara, the capital city of Turkey, in 2000 and 2005, the prevalence of asthma increased from 11.5% to 14.7%.[25,28] In a study using the same International Study of Childhood Asthma and Allergy questionnaire in Istanbul, Turkey's most populous city, the prevalence of asthma increased from 9.8% to 17.8% in children aged 6–12 from 1995 to 2004.[29] In the province of Konya, where the current study was conducted, it was seen that the prevalence in children aged 6–18 years (11.5% in 2007 and 21.8% in 2018) has approximately doubled as compared to the previous.[30,31] Recently, there has been an increase in urban sprawl of Konya. This situation, which is defined as a low-density, leapfrog development towards the peripheries of the cities, causes inefficient use of urban space and environmental problems.[32] The increase in the incidence of asthma may have been caused by the effect of urbanisation and lifestyle changes in Konya recently.[31] According to these studies, the prevalence of asthma in Konya, in 2018, is higher than the prevalence determined in previous years in other provinces of Turkey.[25] When the recent studies in other countries were examined, it is seen that the prevalence of asthma was reported as 15.3% in adolescents and 10.4% in children in Spain.[33] It was 10.1% and 8.4% in children in Southern Brazil and in the United States of America, respectively.[34,35] The prevalence of asthma in Konya (21.8%) appears to be considerably higher than the prevalence in these countries,[34,35] and the average prevalence reported in the Global Asthma Network Phase 1 study.[22] However, this high prevalence in Konya is found similar to the prevalence in some countries in the Global Asthma Network Phase 1 study, such as Cape Town, South Africa (21.3%), Costa Rica (23.2%), and Syria (19.8%).[22]

If there are symptoms such as wheezing, cough, chest tightness, and/or shortness of breath in the patient's history or physical examination, a diagnosis of asthma is suspected.[20,36] Measuring lung function and showing variable expiratory airflow limitations will further support the diagnosis.[20,36] Population-based studies have reported that 7–10% of the general population currently has asthma and that 20–73% of those cases go undiagnosed and therefore untreated.[20,36] Most people with asthma symptoms will be adequately controlled when asthma medications are administered. Indeed, when the term "asthma" is used as a clinical diagnosis for patients with asthma symptoms, it is useful in most cases because it is seen as the first step in initiating appropriate treatment to reduce the burden of the disease.[36] In this study, the asthma control of treated patients was evaluated with the Childhood Asthma Control Test, which was designed in 2007 by Liu et al.[14] A Turkish version of the test was created in 2009.[37] After treatment was given to the patients, the majority of their complaints were resolved, which suggests that we should question children in more detail and more frequently in terms of asthma and other disorders.[38,39]

In a previous study, in which 50 patients presented with chest pain, it was reported that costochondral/musculoskeletal chest pain, exercise-induced asthma, gastrointestinal-related chest pain, and psychogenic chest pain were detected in 76, 12, 8, and 6% of the cases, respectively.[19] In the study of Lin et al., the medical records of 103 children with chest pain presenting to an emergency department were retrospectively reviewed.[15] While idiopathic chest pain was the most common, pulmonary causes (bronchitis, pneumonia, asthma, and hyperventilation) ranked second at a rate of approximately 25% and musculoskeletal (6.7%), gastrointestinal (5.8%), and cardiac (2.0%) causes were identified to a lesser extent. The diagnosis of asthma was made based on past history, chest radiography, and clinical examination findings for 6.9% of the

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patients. In the two studies mentioned here, no data on pulmonary function tests were reported.[15,19]

In another previous study, patients presenting to the emergency department and paediatric cardiology with chest pain were evaluated at the same time.[30] Treadmill exercise tests were also performed by patients who presented to paediatric cardiology with exercise-related pain or cardiac problems. Forced expiratory volume in 1 second and peak expiratory flow rate were evaluated only for patients who underwent treadmill exercise testing. In both groups, chest wall pain was the most common diagnosis and exercise-induced asthma was identified in only 4% of the paediatric cardiology group. These authors emphasised that rates of exercise-induced asthma are underestimated if appropriate examinations are not performed.[30] The diagnosis of asthma is suspected based on the patient's history or physical examination, and laboratory tests will support the diagnosis.[20,36] Moreover, the asthma symptoms of most patients were relieved upon the administration of asthma treatment.[20,36] In this study, the past history of the patients, family history in terms of asthma, atopic history, and physical examination findings were examined in detail and pulmonary function tests were performed for all patients. Spirometry is not diagnostic for asthma, but together with history and physical examination, it is a complementary test in diagnosing asthma.[40] In this study, pulmonary function test results were found to be lower in the patient group than the control group. Improvement in the symptoms of most patients who received asthma treatment supported the accuracy of the diagnosis of asthma. Other causes of chest pain were also investigated in this study. Although idiopathic chest pain and musculoskeletal system disorders were seen most commonly after asthma, gastroesophageal reflux and pneumonia were also detected. In contrast, idiopathic chest pain and musculoskeletal system disorders were detected more commonly in some previous studies.[5,15] The reason for the higher diagnosis of asthma in this study may be that patients were examined in more detail in the differential diagnosis process in this respect. Furthermore, as compared to other places, the higher prevalence of asthma in local region may be another reason. If patients are directed to a specialist who deals with asthma, the diagnosis of asthma may be made more often because more detailed tests will be performed.

Chest pain may be the first sign of asthma in children. Some studies have suggested that this is the only or the most prominent symptom of "chest pain variant asthma."[7,41] The cause of chest pain seen in asthma may be related to chronic inflammation in the airway and the narrowing of the airway caused by this inflammation.[41] Furthermore, chest pain in a child with asthma may be caused by overuse or strain of the rib cage muscles.[7] In addition, bronchoconstriction in asthma can create a feeling of tightness in the chest, which may be perceived as pain by children.[7]

In the present study, the rates of atopic diseases such as asthma and allergic rhinitis were much higher. This may be due to the fact that the clinical findings of patients who present to emergency units and to other departments are different. In addition, the rates of allergic and exercise-induced asthma may be underestimated if appropriate examinations are not done.[39] In this study, when other complaints of the patients were investigated, shortness of breath was found to be the most common. Moreover, some patients were found to have effort dyspnoea and cough. These are important findings in terms of asthma.[7] In addition to the presence of these conditions, the diagnosis of asthma was higher in this study because the physicians who examined the patients were highly experienced in terms of asthma.

Useful pathognomonic markers are necessary for diagnosing asthma; however, they have not yet been developed. Therefore, many factors should be evaluated simultaneously when diagnosing asthma. The diagnosis is made with a detailed clinical history and physical examination.[8,42] It is also important to investigate the family history, as asthma is a genetic disease.[38,43] Studies have reported that approximately 60–70% of asthmatic patients are allergic, whereas 30% of allergic patients have asthma.[42] In the present study, according to the past medical histories and clinical findings of the patients, asthma and allergic rhinitis were diagnosed at rates of 44% and 9.1%, respectively. Additionally, a family history of atopy was found in 23% of the patients in Group 1, which was significantly higher compared to Group 2. This result is important because the presence of a family history of atopy is a risk factor in cases of asthma and other atopic diseases in children.[38,42,43] In addition, skin prick test positivity was detected in 28.4% of the patients. It was significantly more common in cases of allergic rhinitis, but this significance was not found for asthma.

In this study, environmental factors constituting important risks in the development of asthma were also examined and rates of exposure to these factors were observed to be higher in Group 1. Exposure to environmental factors can trigger asthma symptoms and cause chest pain.[44,45] In this study, rates of smoking exposure, presence of pets, and wool use were significantly higher in Group 1 than the control group.

These results draw attention to the importance of environmental factors in the development of such symptoms in children. The regulation of triggering environmental factors has a significant effect on the relief of patients' symptoms.[42] It is known that airway hypersensitivity and serum immunoglobulin E levels in asthma can be affected by genetics.[42,43] However, the total immunoglobulin E levels of our patients with chest pain diagnosed with asthma and allergic rhinitis were not different from those of the control group.

### *Limitations of the study*

The control group included completely healthy children seen in the cardiology clinic for pre-participation physical evaluations for sports. Therefore, their number was relatively lower than that of the patients presenting with chest pain. Besides this, the study population was overall relatively small. It would be more appropriate to have larger and closer numbers of control children and patients to produce more descriptive results.

In addition, the prevalence of asthma in the local area is higher than many regions around the world. This condition may result in more recognition of asthma as the cause of chest pain in this study. In order to determine and compare current prevalence, studies from different regions of Turkey and the world should be conducted simultaneously.

## Conclusion

In this study, patients' clinical findings, presence of atopic disease, and other causes of chest pain were investigated. Although very few cases of chest pain are caused by cardiac problems, families worry because they think chest pain is due to cardiac disease. Furthermore, since most cases are undiagnosed, there may be frequent applications to the hospital. Asthma is a known cause of chest pain and should always be considered in differential diagnoses for chest pain in childhood. Moreover, since asthma is a treatable disease, physicians may prevent repeated visits to the

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hospital by these patients by considering the possibility of asthma. Routine questioning of familial and environmental factors while taking the history of paediatric chest pain patients will be helpful in making the correct diagnosis. The cause of chest pain in most children may be identified accurately through a detailed history and careful physical examination.

Chest pain is the second most common complaint leading to referral to a paediatric cardiologist. The high rate of asthma in this study may be due to the high prevalence of asthma in this region compared to other parts of the world. Hence, the present study may create further awareness among physicians in this respect. Since asthma was found to be common in our study, we suggest that children with chest pain be evaluated especially in terms of asthma and other atopic diseases. It is particularly important that children with chest pain complaints be evaluated together by the paediatric cardiology and paediatric allergy and immunology departments.

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**Authors contributions.** F.D. and M.T.D. conceived and designed the evaluation and drafted the manuscript. M.T.D. participated in designing the evaluation, performed parts of the statistical analysis, and helped to draft the manuscript. F.D. re-evaluated the clinical data, revised the manuscript, and performed the statistical analysis. F.D. and M.T.D. collected the clinical data, interpreted them, and revised the manuscript. All authors read and approved the final manuscript.

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