

Idiopathic Chronic Eosinophilic Pneumonia: Retrospective Analysis of 17 Cases from a Single Center in Turkey

Sibel Arıncı¹, Umut Sabri Kasapoğlu², Sinem Güngör¹, Meltem Ağca¹, Murat Yalçınsoy³, İlim Irmak¹, Pınar Güney¹, Murat Kavas¹, Hatice Türker¹

¹Department of Chest Diseases, Süreyyapaşa Chest Diseases and Thoracic Surgery Training and Research Hospital, İstanbul, Turkey

²Department of Pulmonary and Critical Care Medicine, Marmara University School of Medicine, İstanbul, Turkey

³Department of Chest Diseases, İnönü University School of Medicine, Malatya, Turkey

Abstract

Objective: Idiopathic chronic eosinophilic pneumonia (ICEP) is a rare eosinophilic lung disorder with an unknown etiology and is characterized by subacute or chronic respiratory and general symptoms, alveolar and/or peripheral eosinophilia, and the accumulation of eosinophils in the lungs. We aimed to present diagnostic test results and follow-up outcomes of 17 patients who were diagnosed with ICEP in our hospital in light of literature.

Methods: Between 2008 and 2013, we examined 17 cases of ICEP. We evaluated clinical and laboratory findings together with the long-term follow-up data.

Results: The patients had a mean age of 40.8 years at presentation, and the female/male ratio was 0.8. The most common symptoms were cough (94%), shortness of breath (76%), and high fever (35%). Bronchoalveolar lavage eosinophil percentages of the patients ranged from 3% to 80%. Nine (53%) patients experienced recurrence. Six patients were maintained on low dose steroid due to repeating relapses. Among these patients, 7 (77.7%) had a total IgE level of above 500/IU/mL.

Conclusion: Relapses are common in ICEP after the withdrawal of corticosteroid treatment or during dose reduction. We point out the importance of the close monitoring of patients for identifying relapse. A higher total IgE level during diagnosis may serve as a predictor of recurrence.

Keywords: Corticosteroid, eosinophil, recurrence, total IgE

INTRODUCTION

Idiopathic chronic eosinophilic pneumonia (ICEP) is a rare eosinophilic lung disorder with an unknown etiology. It is characterized by peripheral eosinophilia and the accumulation of eosinophils in the lungs (1). The disorder was first described in 1960 by Christoforis and Molnar, and the study with the first large series of patients was published by Carrington in 1969 (2).

Its prevalence is unknown. ICEP is the most common cause of eosinophilic lung disorders in countries with a low rate of parasitic infections such as Europe and North America. It represents 3% of interstitial lung diseases in studies of large series (3). The diagnosis of ICEP is made by characteristic imaging findings of alveolar eosinophilia and/or peripheral eosinophilia after the exclusion of other potential causes (4). There is no standard dose and duration of corticosteroid therapy for treating ICEP. The initiation of therapy produces a dramatic improvement in symptoms within 24–48 h (5). We aimed to present diagnostic test results and follow-up outcomes of 17 patients who were diagnosed with ICEP in our hospital in light of the literature.

METHODS

Patients and Data

A retrospective cohort study was performed between 2008 and 2013 at the Süreyyapaşa Chest Diseases and Thoracic Surgery Training and Research Hospital. The patient files were reviewed, and 17 patients diagnosed with ICEP were included.



Received Date: 28.11.2015

Accepted Date: 21.03.2016

Available Online Date: 12.07.2016

DOI: 10.5152/ejp.2016.29291

Corresponding Author

Umut Sabri Kasapoğlu

E-mail: umutkasapoglu@gmail.com

• Available online at www.eurasianpulmonol.com



This work is licensed under a Creative Commons Attribution-NonCommercial 4.0 International License.

The patients were evaluated with respect to age, gender, presenting complaints, presence of any comorbid disease, smoking status, and laboratory and radiological findings. Complaints of cough, shortness of breath, fever, sputum, and weight loss were considered as presenting complaints. A patient had smoked cigarettes at least for 1 year to have been considered as smoker.

The peripheral blood eosinophil count, erythrocyte sedimentation rate (ESR), serum total IgE level, serum C-reactive protein (CRP) level, and eosinophils in bronchoalveolar lavage (BAL) were recorded during the diagnostic stage and throughout the clinical follow-up. The serum CRP and total IgE levels of the patients were evaluated by a nephelometric assay using a BN Prospec device of Dade Behring Corporation (Istanbul, Turkey). Hemogram values were evaluated using a Coulter STKS hematological analyzer (Beckman Coulter Cooperation, Miami, FL, USA).

The results of the posterior–anterior (PA) chest X-ray and high-resolution thoracic computed tomography (CT) (Somatom Emotion; Siemens, Erlangen, Germany) during clinical follow-up and spirometric examinations (Spirolab III, MIR, Roma, Italy) at the presentation of the patients were recorded.

This study was approved by the Local Ethical Committee of a state training and research hospital and fulfilled the principles of Declaration of Helsinki (09.12.2014–89513307/1009/375; Dr. Lütfi Kırdar Kartal Training and Research Hospital, İstanbul, Turkey).

Statistical Analysis

The patients' data were collected from hospital files and the hospital operating system. Data without a normal and homogeneous distribution were shown as median (min–max) values as well as numbers and percentages. For statistical analyses, Statistical Package for Social Sciences for Windows 22.0 (SPSS IBM Statistics New York, Armonk, USA) was used. The results were analyzed with a confidence level of 95% and a significance level of $p < 0.05$.

Diagnostic Criteria

The diagnosis of ICEP was based on the presence of respiratory symptoms for usually more than 2 weeks, alveolar and/or blood eosinophilia (alveolar eosinophilia $\geq 40\%$ at BAL differential cell count and blood eosinophilia $\geq 1000/\text{mm}^3$), the presence of pulmonary infiltrates often with a peripheral predominance on chest imaging, and exclusion of any known cause of eosinophilic lung disease (Table 1) (4).

RESULTS

Patients and Clinical Data

The median age of the 17 patients was 35 years (range, 21–71 years); 9 (53%) were males and 8 (47%) were females. At presentation, 4 (23.5%) patients were on treatment for asthma and 4 (23.5%) were on treatment for allergic rhinitis. Seven (41.1%) patients had a history of smoking. The most common presenting symptoms were cough and shortness of breath (Table 2).

Laboratory Findings

All patients had an increased eosinophil count and increased ESR at the baseline. The serum CRP level was higher in 14 of 16 patients with available measurements. Two patients had hypoxic respiratory

Table 1. Criteria for idiopathic chronic eosinophilic pneumonia

1	Respiratory symptoms for 2–4 weeks
2	BAL eosinophilia (whole cell count $\geq 40\%$) or peripheral blood eosinophilia ($> 1000/\text{mm}^3$ and preferably $1500/\text{mm}^3$)
3	Diffuse alveolar consolidations with air bronchogram and/or ground-glass opacities, particularly peripheral, on chest imaging
4	Elimination of other potential causes of eosinophilic lung disease (particularly pulmonary eosinophilia associated with drugs)

BAL: Bronchoalveolar lavage

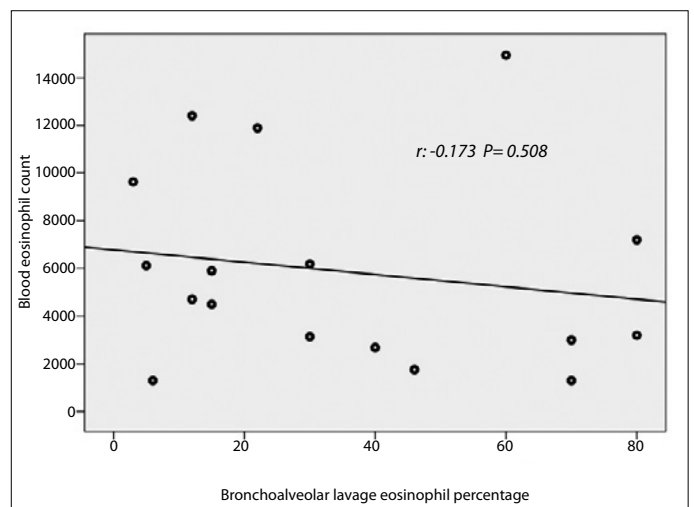


Figure 1. Correlation between blood eosinophil count and bronchoalveolar lavage eosinophil percentage

failure that did not require intensive care. Pearson correlation was performed to examine the correlation between the blood eosinophil count and BAL eosinophil percentage. The blood eosinophil count did not correlate with the BAL eosinophil percentage ($r = -0.173$, $p = 0.508$) (Figure 1).

Radiological Findings and Pulmonary Function Tests

The baseline posterior-anterior chest X-ray images of the patients showed pulmonary infiltrates with peripheral upper and middle zone predominance (Figure 2). The thoracic CT images showed non-homogeneous increases in density, mostly involving the upper lobe and periphery (Figures 3, 4). The baseline pulmonary function tests (PFTs) showed obstructive type in 3 (18%), normal type in 3 (18%), and restrictive type of the disease in 11 (64%) patients; there was a reduction in the diffusing capacity of the lungs for carbon monoxide (DLCO) test in 7 (41%) patients, while 10 (59%) patients had normal DLCO results.

Diagnostic Method

The diagnosis of ICEP was made based on the patients' dramatic responses to treatment with steroids after the exclusion of other causes after the determination of eosinophils in the BAL fluid and/or peripheral blood. The percentage of eosinophils in the BAL fluid ranged from

Table 2. Demographics and admission symptoms of patients

Patient no	Sex	Age (years)	Smoking status	History of asthma	Symptoms
1	Female	35	Smoker	Yes	Dyspnea, cough, fever, weight loss
2	Male	26	Nonsmoker	No	Dyspnea, cough, fever
3	Female	54	Nonsmoker	No	Dyspnea, cough
4	Female	50	Nonsmoker	No	Dyspnea, cough
5	Male	21	Smoker	Yes	Dyspnea, cough, fever
6	Female	48	Smoker	Yes	Dyspnea
7	Female	30	Smoker	No	Dyspnea, cough
8	Female	27	Nonsmoker	No	Dyspnea, cough, sputum
9	Male	62	Nonsmoker	No	Dyspnea, cough, sputum
10	Male	46	Smoker	No	Cough, fever
11	Male	70	Smoker	No	Cough, fever
12	Male	24	Nonsmoker	Yes	Dyspnea, cough, fever
13	Female	24	Nonsmoker	No	Dyspnea, cough, sputum
14	Female	28	Nonsmoker	No	Dyspnea, cough
15	Male	71	Smoker	No	Dyspnea, cough, sputum, weight loss
16	Male	34	Nonsmoker	No	Cough, sputum, weight loss
17	Male	44	Nonsmoker	No	Cough

3% to 80%. None of the patients required open lung biopsy, which is the gold standard diagnostic method. The baseline laboratory and radiological findings of the patients are summarized in Table 3.

Treatment and Clinical Follow-up

Following the diagnosis of ICEP, there was a remarkable improvement in the infiltrates on chest imaging and in presenting symptoms within 1 week after the initiation of systemic steroids (Figure 5).

Recurrence occurred at months 4 and 6 during the clinical follow-up in 2 patients whose treatment was completed and in 7 patients while reducing the dose of steroids. Of these 9 patients with recurrence, 7 (77.7%) had a total IgE level of above 500/IU/mL. No recurrence was observed in 8 patients during treatment and follow-up, with a treatment duration of 12–18 months. Six patients with recurrence were maintained on a lower dose of steroids (methylprednisolone, 4–8 mg). The treatment and follow-up results of the patients are summarized in Table 4.

DISCUSSION

In clinical practice, ICEP is included in idiopathic eosinophilic pneumonias in the classification of eosinophilic pneumonias (4). Although it may occur in any age group, the mean age at diagnosis is 45 years, and it affects twice as many women as men (6, 7). While two-thirds of patients have prior asthma at presentation, approximately 50% may also have a history of atopy, drug allergy, nasal polyposis, and urticaria (6-8). Our patients had a mean age of 40.8 years at presentation, and the female/male ratio was 0.8. At baseline, 23.5% of our patients had a history of asthma and 23.5% had allergic rhinitis, which was lower than the rates in the literature.

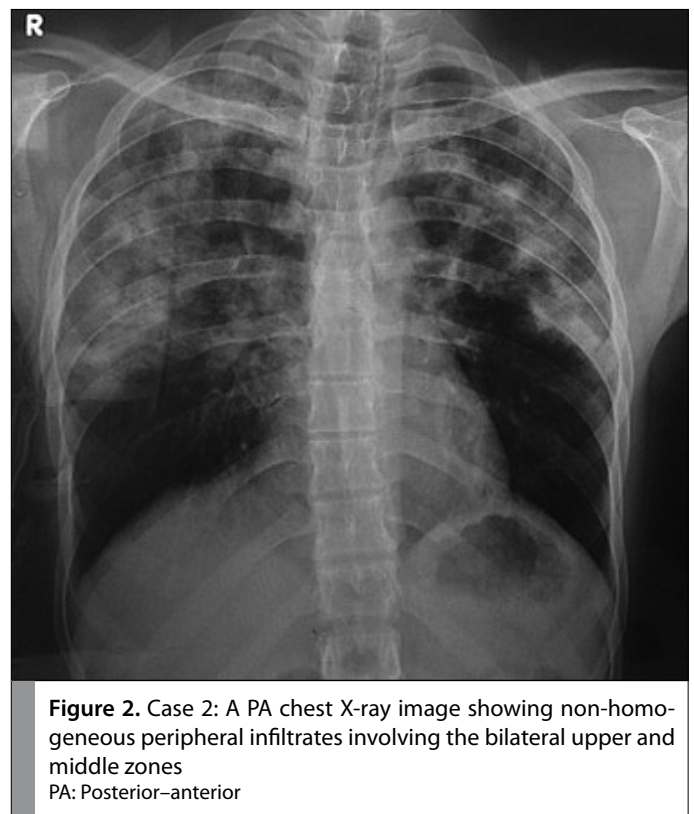


Figure 2. Case 2: A PA chest X-ray image showing non-homogeneous peripheral infiltrates involving the bilateral upper and middle zones
PA: Posterior–anterior

The onset of symptoms is progressive and subacute. There are a few weeks or a month between the onset of symptoms and diagnosis in ICEP. In total, 60–90% of patients have a moderate

Table 3. Baseline laboratory and radiological findings

Patient no	Blood eosinophil count, / mm ³	BAL eosinophil, %	ESR, mm/h	Serum total IgE, / IU/mL	Serum CRP, mg/dL	PFTs	Thorax computed tomography findings	Fulfilling the diagnostic criteria
1	9630	3	110	722	170	Restrictive	Bilateral widely peripheral non-homogeneous consolidation and ground-glass opacities	Yes
2	6120	5	120	928	117	Restrictive	Bilateral peripheral non-homogeneous consolidation in upper lobes	Yes
3	5900	15	100	986	153	Normal	Bilateral peripheral non-homogeneous consolidation in upper lobes	Yes
4	1300	70	90	270	17.6	Restrictive	Bilateral patchy infiltrations predominate in upper lobes	Yes
5	6190	30	60	347	4.18	Restrictive	Bilateral patchy infiltrations predominate in upper lobes	Yes
6	1760	46	80	110	30.1	Normal	Bilateral peripheral non-homogeneous consolidation in upper lobes	Yes
7	14940	60	80	565	21	Restrictive	Bilateral widely peripheral non-homogeneous consolidation	Yes
8	3140	30	50	334	3.34	Obstructive	Bilateral widely peripheral non-homogeneous consolidation	Yes
9	3000	70	100	829	48.5	Restrictive	Bilateral widely peripheral non-homogeneous consolidation	Yes
10	12400	12	55	2200	120	Restrictive	Bilateral widely peripheral non-homogeneous consolidation and ground-glass opacities	Yes
11	1300	6	104	560	277	Restrictive	Bilateral widely peripheral non-homogeneous consolidation and ground-glass opacities	Yes
12	4710	12	120	1340	125	Obstructive	Bilateral widely peripheral non-homogeneous consolidation and ground-glass opacities	Yes
13	11890	22	80	120	78.90	Restrictive	Bilateral peripheral non-homogeneous consolidation in upper lobes	Yes
14	4500	15	80	80	-	Restrictive	Bilateral peripheral non-homogeneous consolidation	Yes
15	7200	80	70	463	20.2	Obstructive	Bilateral patchy infiltrations predominate in upper lobes	Yes
16	3200	80	100	59	68.3	Restrictive	Bilateral peripheral non-homogeneous consolidation	Yes
17	2680	40	60	606	33	Normal	Bilateral peripheral non-homogeneous consolidation predominate in upper lobes	Yes

BAL: Bronchoalveolar lavage; CRP: C-reactive protein; ESR: erythrocyte sedimentation rate; PFTs: pulmonary function tests

Table 4. Treatment and follow-up results of patients

Patient no	Recurrence	Time of recurrence	Number of recurrences	Total IgE level >500/	Maintenance dose of methylprednisolone, mg
1	No	-	0	Yes	0
2	Yes	During dose reduction	3	Yes	4 mg
3	Yes	During dose reduction	4	Yes	4 mg
4	Yes	During dose reduction	3	No	4 mg
5	No	-	0	No	0
6	No	-	0	No	0
7	Yes	After completion of treatment	1	Yes	8 mg
8	No	-	0	No	0
9	Yes	During dose reduction	3	Yes	4 mg
10	Yes	During dose reduction	2	Yes	4 mg
11	Yes	During dose reduction	1	Yes	0
12	Yes	During dose reduction	3	Yes	4 mg
13	No	-	0	No	0
14	No	-	0	No	0
15	No	-	0	No	0
16	Yes	After completion of treatment	1	No	8 mg
17	No	-	0	Yes	0

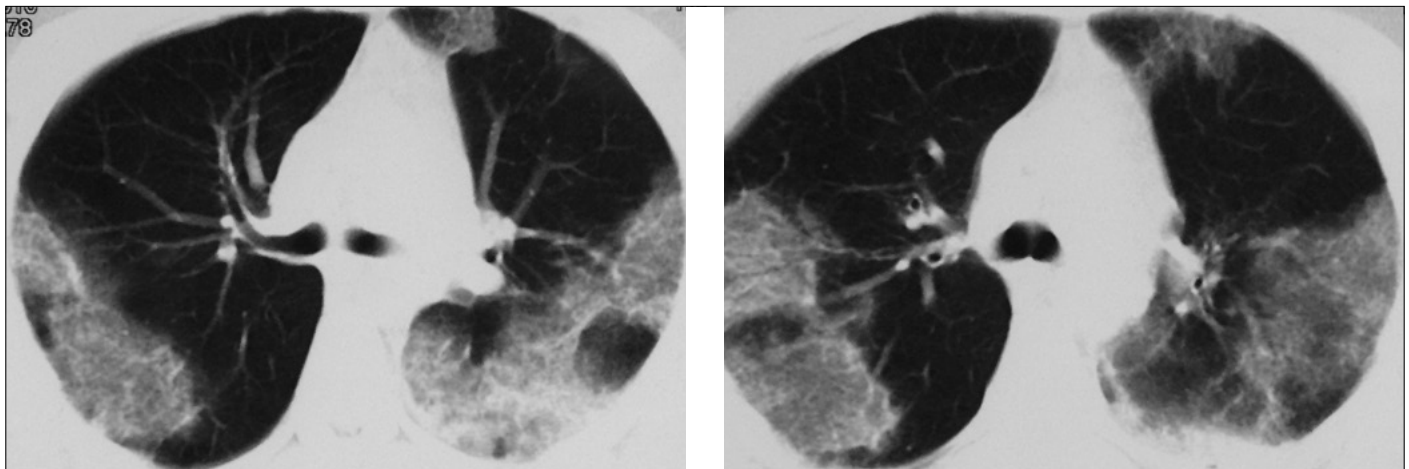


Figure 3. Case 13: A thorax CT image showing non-homogeneous bilateral diffuse infiltrates of peripheral localization with upper lobe predominance
CT: Computed tomography

shortness of breath, and the most common cause is cough in 90% of the patients. While 20% of the patients have rhinitis and sinusitis, chest pain and hemoptysis are rare symptoms. Other than common respiratory symptoms, systemic symptoms, such as weight loss, anorexia, and fatigue, may also occur (6, 7).

In our patients, the most common symptoms included cough (94%), shortness of breath (76%), and high fever (35%). Respiratory failure requiring mechanical ventilation is rare compared to idiopathic acute eosinophilic pneumonia (9). Two of our patients had hypoxic respiratory failure that did not require mechanical ventilator support.

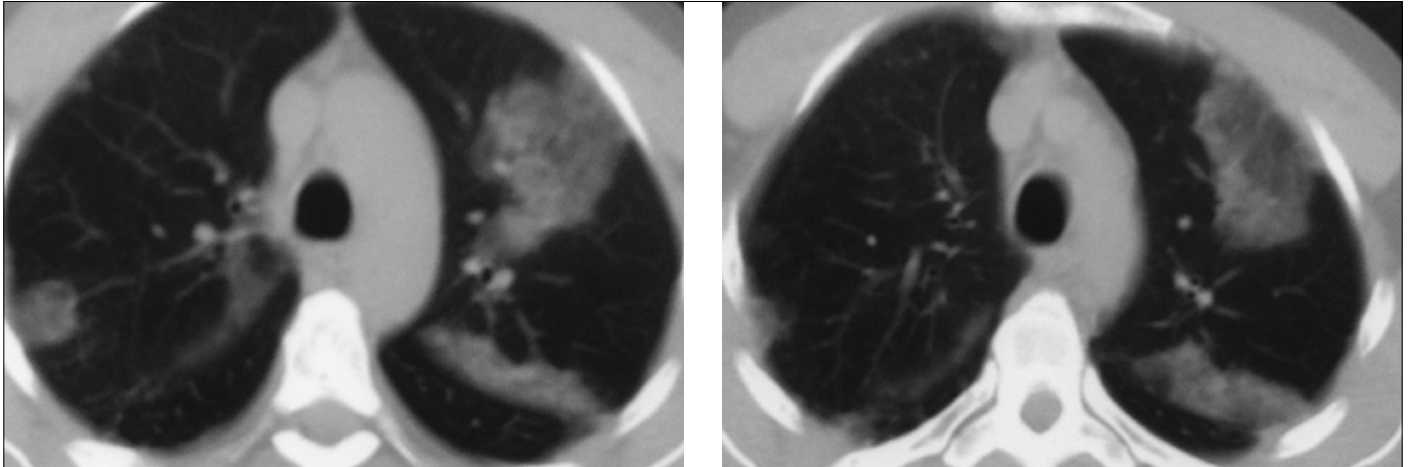


Figure 4. Case 17: A thorax CT image showing non-homogeneous bilateral infiltrates in the periphery and upper lobe
CT: Computed tomography

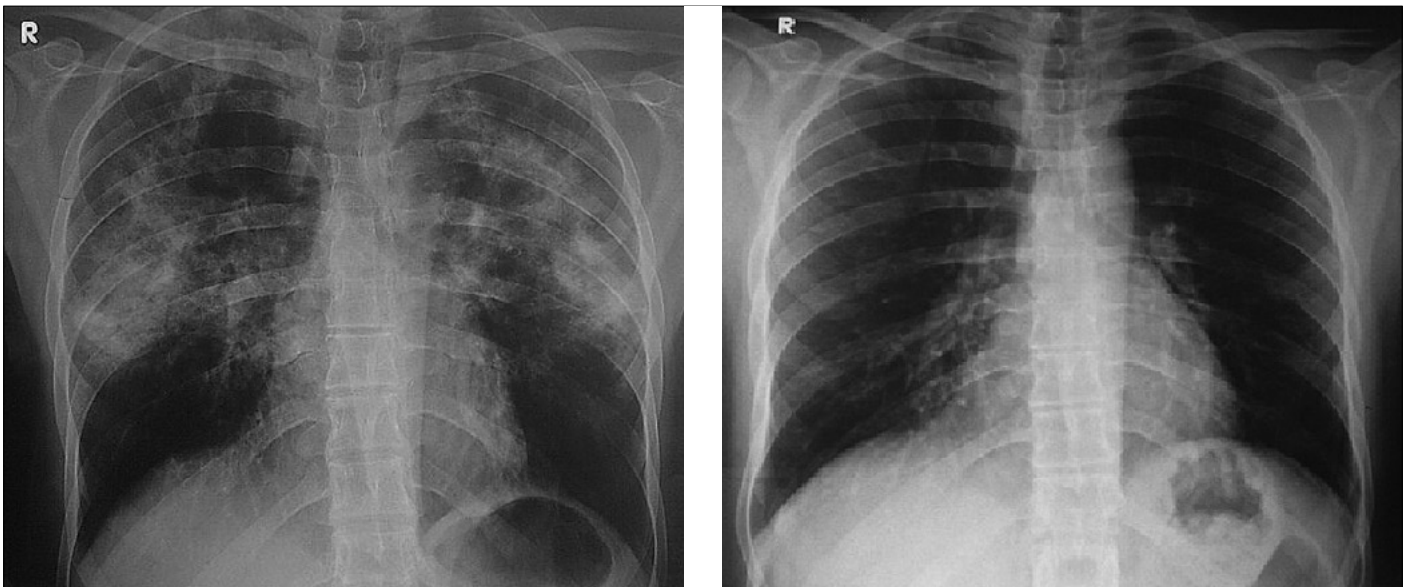


Figure 5. Case 2: Complete recovery was seen in infiltrates on the PA chest X-ray 1 week after the initiation of treatment with systemic steroids
PA: Posterior-anterior

Many patients who have not received systemic treatment with steroids have increased levels of peripheral blood eosinophilia, which is the most important key to the diagnosis. In studies with large series, the presence of peripheral blood eosinophilia (representing 20–30% of blood leukocytes) and BAL eosinophilia (more than 25% eosinophils at the BAL cell count) have been considered to be major criteria (4, 6, 7). Although non-specific, increased serum CRP levels and ESRs may also occur. Total IgE levels may be elevated in approximately 50% of the patients (6, 7). In our study, the average concentration of eosinophils in the peripheral blood was $6160/\text{mm}^3$ ($1300\text{--}14940/\text{mm}^3$), with percentages of eosinophils ranging from 17 to 67.7. The BAL eosinophil percentages of our patients ranged from 3 to 80. Among those 14 patients whose total IgE level was measured, only 2 had a normal total IgE level, which was, in fact, higher compared to that in the literature. Furthermore, ESRs were higher in all our patients, while serum CRP levels were within the normal range in only 2 patients.

On chest imaging, ICEP is characterized by bilateral alveolar infiltrates of a migratory, temporary, patchy pattern in the periphery (2, 6, 7). Thoracic CT reveals opacities, mostly bilateral with upper lobe predominance (10-12). Peripheral symmetrical alveolar lesions, defined as the “photographic negative” of pulmonary edema, occur in 25% of the patients, and they are diagnostic of ICEP (13, 14). Pleural effusions are present in 10% of the patients, while cavitory lesions, nodular infiltrations, and atelectasis are among the rare radiologic findings (7, 12, 15). Our patients showed non-homogeneous bilateral patchy alveolar infiltrates in the periphery with upper and mid zone predominance, which is consistent with the radiological findings of ICEP.

PFT results vary from person to person; approximately 50% have obstructive type and 50% have restrictive type of lung defects. DLCO is frequently reduced (6, 7). In a study investigating the results of long-term PFTs in patients with ICEP, Durieu et al. (16) reported that

obstructive defects are common in ICEP and that obstruction might appear even in the absence of any clinical and radiological signs of relapse. Among our patients, 64% showed restrictive type of defects and 18% showed obstructive type of defects on PFTs, while 41% showed reduction in DLCO.

The gold standard for the diagnosis of ICEP is open lung biopsy or tissue eosinophilia confirmed at transbronchial lung biopsy; however, histological confirmation of the disease is not necessary (4). The diagnosis of ICEP is based on the demonstration of characteristic imaging features, i.e., alveolar eosinophilia and/or peripheral eosinophilia and exclusion of other potential causes. The diagnosis is strongly supported by markedly elevated peripheral blood eosinophilia along with typical clinical radiologic findings. Other potential causes of eosinophilia must be carefully investigated, including the history of drug use, exposure to toxics, and parasitic and fungal infections in particular (4).

Despite spontaneous resolution, dramatic clinical and radiologic improvement is observed by treatment with systemic steroids in ICEP. However, there is no consensus in the literature on the initial dose and duration of treatment, and steroid treatment is usually initiated at a dose of 0.3–1 mg/kg/day (6, 8, 10, 16, 17). Our patients were initiated on therapy with methylprednisolone at a dose of 0.5 mg/kg/day, and the dose was rapidly reduced based on the clinical and radiological response. Symptoms improve within the first 2 days, and infiltrates on chest imaging completely disappear in all patients within 1 week without any sequelae (6–8, 18). Relapse occurs in 50% of the patients during reduction or discontinuation of the dose. Relapse is more frequent in patients treated for less than 6 months (5, 6, 15, 19). Prolonged corticosteroid treatment may be required due to frequent relapses in some patients, and the prednisone dose needed to prevent relapse in such cases is often <10 mg (6). In our study, 9 (53%) patients experienced recurrence. Of these, recurrence was after the completion of the steroid treatment in 2 and during the dose reduction of steroids in 7. Six patients were maintained on a low dose of steroids (methylprednisolone, 4–8 mg) due to repeating relapses. Among these patients, 7 (77.7%) had a total IgE level of above 500 IU/mL.

CONCLUSION

Relapses are common in ICEP after the completion of the treatment with corticosteroids or during dose reduction. The close monitoring of patients is recommended for identifying relapse. Furthermore, we believe that a higher total IgE level during diagnosis serves as a predictor of recurrence; therefore, studies with larger series are required to further elucidate it.

Ethics Committee Approval: This study was approved by the Local Ethical Committee of a state training and research hospital and fulfilled the Declaration of Helsinki (09.12.2014 – 89513307/1009/375 - Dr. Lütfi Kırdar Kartal Training and Research Hospital, Istanbul, Turkey).

Informed Consent: Due to the retrospective design of the study, informed consent was not taken.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept - S.A., U.S.K., S.G.; Design - S.A., U.S.K., S.G., M.Y.; Supervision - S.A., M.A., M.Y., H.T.; Resources - S.A., H.T.; Materials - U.S.K.,

S.G., M.A., M.Y., M.K., H.T., İ.L., P.G.; Data Collection and/or Processing - U.S.K., S.G., M.A., M.Y., M.K., İ.L., P.G.; Analysis and/or Interpretation - S.G., M.A., M.Y., M.K., H.T.; Literature Search - S.G., M.A., M.Y., M.K.; Writing Manuscript - S.A., U.S.K., S.G., M.Y.; Critical Review - S.A., S.G., M.Y., H.T.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study has received no financial support.

REFERENCES

- Allen JN, Davis WB. Eosinophilic lung diseases. *Am J Respir Crit Care Med* 1994; 150: 1423-38. [\[CrossRef\]](#)
- Carrington CB, Addington WW, Goff AM, Madoff IM, Marks A, Schwaber JR, et al. Chronic Eosinophilic Pneumonia. *N Engl J Med* 1969; 280: 787-98. [\[CrossRef\]](#)
- Thomeer MJ, Costabe U, Rizzato G, Poletti V, Demedts M. Comparison of registries of interstitial lung diseases in three European countries. *Eur Respir J Suppl* 2001; 32: 114s-8s.
- Cottin V. Idiopathic eosinophilic pneumonias. In: Cordier JF editor, *European Respiratory Society Monograph Orphan Lung Diseases*. Vol. 54. Plymouth: Latimer Trend & Co. Ltd; 2011. p118-39.
- Çelik G. Eozinofilik Akciğer Hastalıkları. In: Özlü T, Metintaş M, Karadağ M, Kaya A editörler, *Solunum Sistemi ve Hastalıkları*. 1.Baskı. İstanbul: Medikal Yayıncılık; 2010. p.1113-27.
- Marchand E, Reynaud-Gaubert M, Lauque D, Durieu J, Tonnel AB, Cordier JF. Idiopathic chronic eosinophilic pneumonia. A clinical and follow-up study of 62 cases. *The Groupe d'Etudes et de Recherche sur les Maladies "Orphelines" Pulmonaires (GERM"O"P)*. *Medicine (Baltimore)* 1998; 77: 299-312. [\[CrossRef\]](#)
- Jederlinic PJ, Sicilian L, Gaensler EA. Chronic eosinophilic pneumonia. A report of 19 cases and a review of the literature. *Medicine (Baltimore)* 1988; 67: 154-62. [\[CrossRef\]](#)
- Naughton M, Fahy J, FitzGerald MX. Chronic eosinophilic pneumonia. A long-term follow up of 12 patients. *Chest* 1993; 103: 162-5. [\[CrossRef\]](#)
- Libby DM, Murphy TF, Edwards A, Gray G, King TK. Chronic eosinophilic pneumonia: an unusual cause of acute respiratory failure. *Am Rev Respir Dis* 1980; 122: 497-500.
- Bancal C, Sadoun D, Valeyre D, Roucou Y, Clerici C, Georges R, et al. Chronic idiopathic eosinophilic pneumopathy. Carrington's disease. *Presse Med* 1989; 18: 1695-8.
- Mayo JR, Müller NL, Road J, Sisler J, Lillington G. Chronic eosinophilic pneumonia: CT findings in six cases. *AJR Am J Roentgenol* 1989; 153: 727-30. [\[CrossRef\]](#)
- Ebara H, Ikezoe J, Johkoh T, Kohno N, Takeuchi N, Kozuka T, et al. Chronic eosinophilic pneumonia: evolution of chest radiograms and CT features. *J Comput Assist Tomogr* 1994; 18: 737-44. [\[CrossRef\]](#)
- Gaensler E, Carrington CB. Peripheral opacities in chronic eosinophilic pneumonia: the photographic negative of pulmonary edema. *AJR Am J Roentgenol* 1977; 128: 1-13. [\[CrossRef\]](#)
- Zimhony O. Photographic negative shadow of pulmonary oedema. *Lancet* 2002; 360: 33. [\[CrossRef\]](#)
- Alam M, Burki NK. Chronic eosinophilic pneumonia: a review. *South Med J* 2007; 100: 49-53. [\[CrossRef\]](#)
- Durieu J, Wallaert B, Tonnel AB. Long term follow-up of pulmonary function in chronic eosinophilic pneumonia. *Eur Respir J* 1997; 10: 286-91. [\[CrossRef\]](#)
- Pearson DL, Rosenow EC 3rd. Chronic eosinophilic pneumonia (Carrington's): a follow-up study. *Mayo Clin Proc* 1978; 53: 73-8.
- Hayakawa H, Sato A, Toyoshima M, Imokawa S, Taniguchi M. A clinical study of idiopathic eosinophilic pneumonia. *Chest* 1994; 105: 1462-6. [\[CrossRef\]](#)
- Rochester CL. The eosinophilic pneumonias. In: Fishman AP, Ed. *Fishman's Pulmonary Diseases and Disorder*. International edition. 3rd ed. New York: Mc Graw Hill; 1998; p.1138-50.