Case Report

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Waterpipe (narghile, hookah) tobacco smoking-induced acute eosinophilic pneumonia

Ali Nihat Annakkaya, Ege Güleç Balbay, Özlem Öztürk, Binnur Önal[1], Fuat Aytekin, Öner Balbay

Abstract:

Acute eosinophilic pneumonia (AEP) is characterized by a febrile illness, diffuse pulmonary infiltrates, hypoxemic respiratory failure, and pulmonary eosinophilia. A temporal relationship has been described in a number of patients between the development of AEP and the recent onset of cigarette smoking. A 22-year-old male patient with no known chronic disease was admitted to the emergency service for 3 days with the complaints of cough, fever, dyspnea, and pleuritic chest pain. He was formerly a nonsmoker but was a waterpipe (narghile, hookah) tobacco smoker as a new habit once a night for the last 2 months. The erythrocyte sedimentation rate and C-reactive protein are elevated. Arterial blood gases revealed moderate hypoxemia. Computed tomography scans included bilateral, random, and patchy ground-glass opacities and also small bilateral pleural effusions. Echocardiograhic examination was normal. We had accepted the patient as an acute eosinophilic pneumonia due to rapid response to corticosteroid treatment, no microorganism in bronchoalveolar lavage (BAL) culture, and predominant eosinophilia was observed (70%–80%) in BAL.

Keywords:

Acute eosinophilic pneumonia, cigarette, narghile, tobacco

Introduction

Acute eosinophilic pneumonia (AEP) was first described as a cause of acute respiratory failure in 1989. AEP is characterized by a febrile illness, diffuse pulmonary infiltrates, hypoxemic respiratory failure, and pulmonary eosinophilia. The mechanism of AEP remains unknown. Some investigators have suggested that AEP is an acute hypersensitivity reaction to an unidentified inhaled antigen in an otherwise healthy individual.[1,2]

A temporal relationship has been described in a number of patients between the development of AEP and the recent onset of cigarette smoking either the first time or following a period of smoking cessation and also waterpipe tobacco smoking (WPS).[3-10] Although the underlying mechanisms of WPS leading to AEP are not well understood, case reports and case series suggest a strong correlation between recent cigarette smoking or alteration in smoking habits and development of AEP.[7] We reported the case with AEP following recent onset of WPS.

Case Report

A 22-year-old male patient with no known chronic disease was admitted to the emergency service for 3 days with the complaints of cough, fever, dyspnea, malaise, myalgias, and pleuritic chest pain. He was formerly a nonsmoker but was a waterpipe (narghile, hookah) tobacco smoker as a new habit once a night for the last 2 months. Physical examination

Departments of Pulmonology and [1]Pathology, Faculty of Medicine, Duzce University, Duzce, Turkey

Address for correspondence:
Prof. Ege Güleç Balbay, Department of Pulmonology, Faculty of Medicine, Duzce University, Duzce, Turkey.
E-mail: egegulecbalbay@gmail.com

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revealed fever (38.5°C), tachypnea, and desaturation with pulse oximetry. Bibasilar inspiratory crackles are heard on auscultation of the chest. The patient was presented with an initial neutrophilic leukocytosis (white blood cell: 17.000, eosinophil 1.9%). The erythrocyte sedimentation rate (12 mm/h) and C-reactive protein (2.02 mg/dL) were elevated. Arterial blood gases revealed moderate hypoxemia. Posterior-anterior chest film showed bilateral pulmonary infiltrates with bilateral blunted costodiaphragmatic angle [Figure 1a]. High-resolution computed tomography scans included bilateral, random, and patchy ground-glass opacities and also small bilateral pleural effusions [Figure 2]. To both diagnose AEPs and to rule out possible infectious and cardiac etiology, fiberoptic bronchoscopy for bronchoalveolar lavage (BAL) and echocardiography was performed. Echocardiograhic examination was normal. On BAL corticosteroid therapy (0.5 mg/kg) and nonspecific antibiotics were initiated. The parenchymal infiltration except bilateral mild pleural effusion was almost resolved radiographically following 2 days of treatment [Figure 1b] and was completely resolved after 2 weeks of treatment [Figure 1c]. We had accepted the patient as AEP due to rapid response to corticosteroid treatment, no any microorganism in BAL culture and predominant eosinophilia (lymphocyte 5%, macrophage 7%–8%, eosinophil 70%–80%, epithelial 1%–2%, and PNL 7%–8%) in BAL [Figure 3a-c]. Corticosteroid therapy has been continued for 2 months after THE initiation of therapy.

Discussion

AEP can be caused by drugs, toxins, infections, smoking, and lung irradiation; these possibilities are best investigated by a careful history.[11] Recent alterations in smoking habits, not only beginning to smoke but also restarting to smoke and increasing daily smoking doses, are associated with the development of AEP. AEP has been associated most often with new onset or resumption of cigarette smoking. A temporal relationship has been described in a number of patients between the development of AEP and the recent onset of cigarette smoking.[3-5]

WPS called narghile in Turkish has becoming widespread worldwide and is also one of the most popular and epidemic forms of tobacco use among Turkish youth.[12] Narghile is a preparation of tobacco, glycerol, and other additives and flavors using charcoal separated by thin aluminum foil to heat the mixture. Air drawn in over the charcoal passing through the tobacco mixture through the waterpipe before the vapors cool are released as a dense aerosol produces the smoke. Hookah smoking, compared with cigarette smoking, produces a greater content of tobacco smoke, tar, carbon monoxide, polyaromatic hydrocarbons, and carbonylic compounds.[7] Possible mechanisms of respiratory diseases in WPS were explored in *in vitro* and *in vivo* studies. WPS resulted in increased airway resistance, lung inflammation, oxidative stress, and catalase activity in animal lungs.[13] In humans, levels of inflammatory cytokines were decreased in the exhaled breath of WP smokers, while the bronchoalveolar lavage fluid of WPS with chronic obstructive pulmonary disease (COPD) had increased metalloproteinase two and nine gene expression similar to that of cigarette smokers with COPD.[14]

The diagnosis of AEP can usually be made in patients who meet the following clinical criteria including a febrile illness of short duration (often <1 week), hypoxemic respiratory failure, diffuse pulmonary infiltrates on chest radiograph, BAL differential cell count showing eosinophilia >25% with exclusion of known causes of pulmonary eosinophilia plus recent alterations in smoking habits, and the absence of infection or alternative causes for eosinophilia. BAL with differential cell count should be performed early if the etiology is in question.[2,3,10,15] We had accepted the patient as AEP based on typical clinical criteria above and also due to rapid clinical and radiological response

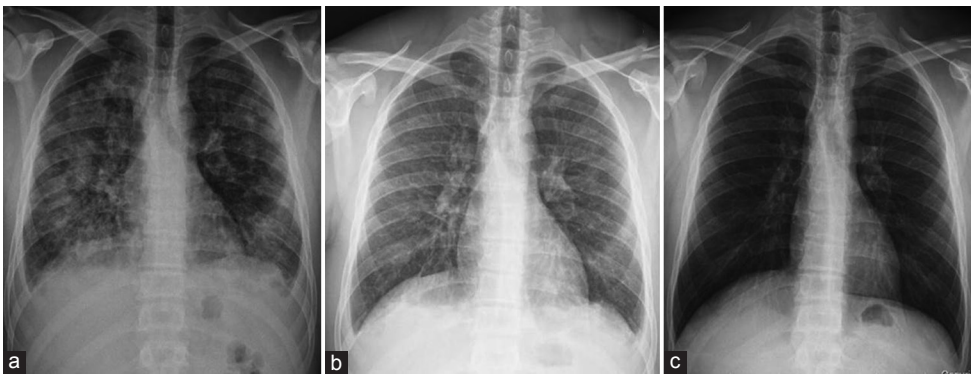

Figure 1: (a) Admission chest X-ray showed bilateral pulmonary infiltrates with bilateral blunted costodiaphragmatic angle. (b) The parenchymal infiltration except bilateral mild pleural effusion was almost resolved radiographically following 2 days of corticosteroid treatment. (c) Completely resolved chest X-ray after 2 weeks of treatment

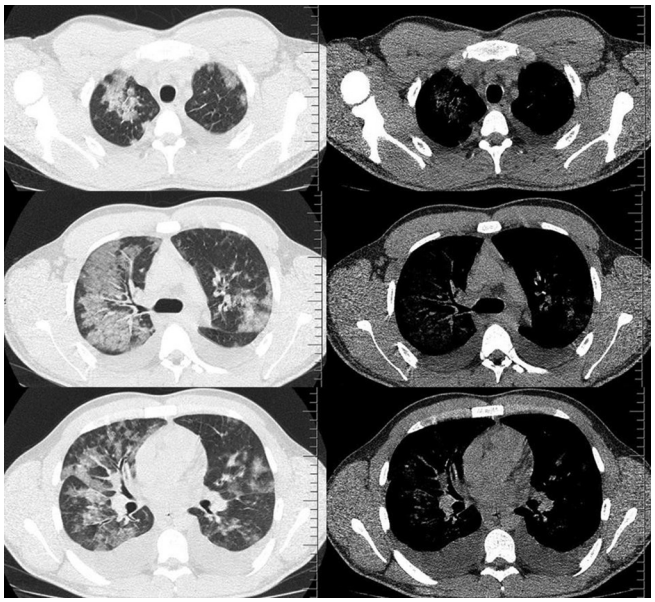

Figure 2: High-resolution computed tomography scans included bilateral, random, and patchy ground-glass opacities, and also small bilateral pleural effusions

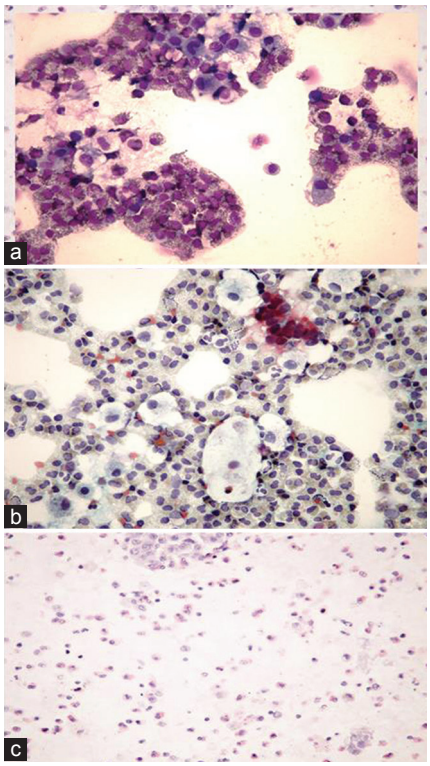

Figure 3: (a-c) Cellular infiltration composing predominantly of eosinophils and accompanying macrophages and mesothelial cells (a) (Cytocentrifuge, MGG, ×400). (b) (Cytocentrifuge, PAP, ×400). (c) (Cell block, H and E, ×400)

to corticosteroid treatment, the delayed response to pleural effusion. The pleural effusion has resolved more slowly than the pulmonary parenchymal infiltrates in the present case which is compatible with the literature.[16] Written informed consent was obtained from a patient who participated in this study.

Conclusion

As tobacco initiation or increase in amount is often associated with AEP and as relapses have been associated with resumption of smoking, complete smoking cessation should be strongly encouraged. AEP should be considered as a possible diagnosis in patients with unexplained acute febrile respiratory failure and pulmonary infiltrates after recent exposure to recent inhaled hookah smoking.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Badesch DB, King TE Jr., Schwarz MI. Acute eosinophilic pneumonia: A hypersensitivity phenomenon? Am Rev Respir Dis 1989;139:249-52.
2. Slabbynck H, Coeck C, Galdermans D, Van Schaardenburg C, Coolen D. Dyspnea, fever, and eosinophilia. Chest 1993;104:585-6.
3. Philit F, Etienne-Mastroïanni B, Parrot A, Guérin C, Robert D, Cordier JF, *et al*. Idiopathic acute eosinophilic pneumonia: A study of 22 patients. Am J Respir Crit Care Med 2002;166:1235-9.
4. Uchiyama H, Suda T, Nakamura Y, Shirai M, Gemma H, Shirai T, *et al*. Alterations in smoking habits are associated with acute eosinophilic pneumonia. Chest 2008;133:1174-80.
5. Natarajan A, Shah P, Mirrakhimov AE, Hussain N. Eosinophilic pneumonia associated with concomitant cigarette and marijuana smoking. BMJ Case Rep 2013;2013. pii: bcr2013009001.
6. Retzky SS. FDA encourages reporting of tobacco product adverse experiences. Chest 2016;150:1169-70.
7. Kang M, Raj V, Berman AR. A man in his 20s with diffuse lung opacities and acute respiratory failure after hookah smoking. Chest 2016;150:e175-e178.
8. Dyal H, Singhvi A, Patel R, Mendez M, Thavarajeh K, Jennings JA. Case of eosinophilic pneumonia following recent onset of hookah smoking. Chest 2014;146 Suppl 2:406.
9. Hilts J, Skinner A, Bellam S. Hookah, an unexpected precipitant of acute eosinophilic pneumonia. Am J Respir Crit Care Med 2014;189:6487.
10. Umeki S, Soejima R. Acute and chronic eosinophilic pneumonia: Clinical evaluation and the criteria. Intern Med 1992;31:847-56.
11. Nadarajan P, Fabre A, Kelly E. Sulfasalazine: A rare cause of acute eosinophilic pneumonia. Respir Med Case Rep 2016;18:35-6.
12. Günen H, Tarraf H, Nemati A, Al Ghobain M, Al Mutairi S, Aoun Bacha Z. Waterpipe tobacco smoking. Tuberk Toraks 2016;64:94-6.

13. Nemmar A, Yuvaraju P, Beegam S, John A, Raza H, Ali BH, *et al.* Cardiovascular effects of nose-only water-pipe smoking exposure in mice. Am J Physiol Heart Circ Physiol 2013;305:H740-6.
14. Bentur L, Hellou E, Goldbart A, Pillar G, Monovich E, Salameh M, *et al.* Laboratory and clinical acute effects of active and passive indoor group water-pipe (narghile) smoking. Chest 2014;145:803-9.
15. Cheon JE, Lee KS, Jung GS, Chung MH, Cho YD. Acute eosinophilic pneumonia: Radiographic and CT findings in six patients. AJR Am J Roentgenol 1996;167:1195-9.
16. Pope-Harman AL, Davis WB, Allen ED, Christoforidis AJ, Allen JN. Acute eosinophilic pneumonia. A summary of 15 cases and review of the literature. Medicine (Baltimore) 1996;75:334-42.