

## Case Report

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DOI: 10.4103/ejop.ejop_3_18

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

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### 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. Echocardiographic 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.<sup>[1,2]</sup>

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).<sup>[3-10]</sup> 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.<sup>[7]</sup> 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

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**How to cite this article:** Annakkaya AN, Balbay EG, Öztürk Ö, Önal B, Aytekin F, Balbay Ö. Waterpipe (narghile, hookah) tobacco smoking-induced acute eosinophilic pneumonia. *Eurasian J Pulmonol* 2018;20:46-9.

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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. Echocardiographic 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.

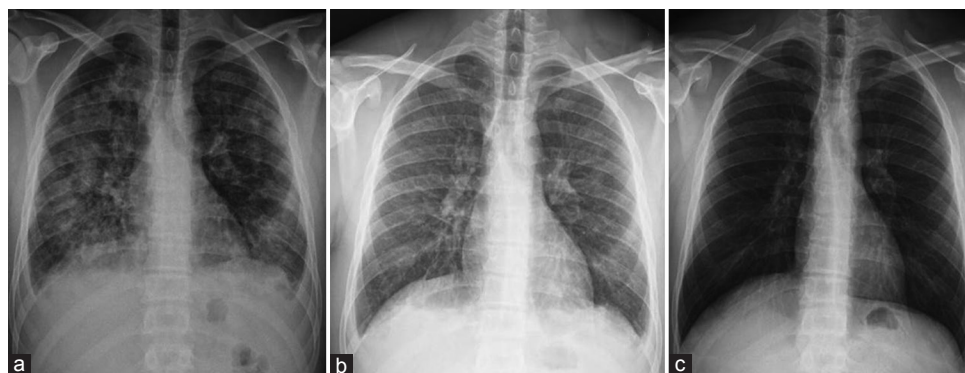
## Discussion

AEP can be caused by drugs, toxins, infections, smoking, and lung irradiation; these possibilities are best investigated by a careful history.<sup>[11]</sup> 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

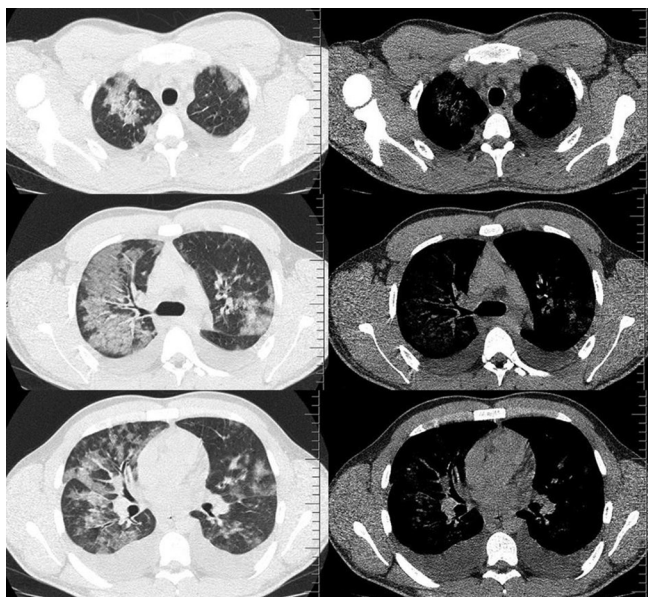
been described in a number of patients between the development of AEP and the recent onset of cigarette smoking.<sup>[3-5]</sup>

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.<sup>[12]</sup> 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.<sup>[7]</sup> 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.<sup>[13]</sup> 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.<sup>[14]</sup>

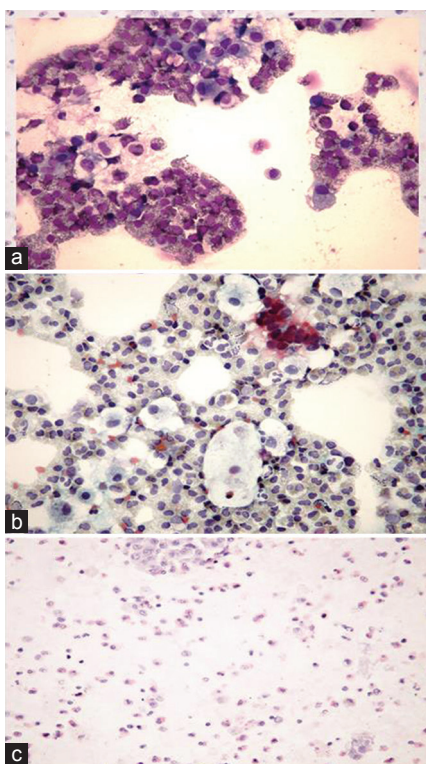
The diagnosis of AEP can usually be made in patients who meet the following clinical criteria including a febrile illness of short duration (often <1 week), hypoxemic respiratory failure, diffuse pulmonary infiltrates on chest radiograph, BAL differential cell count showing eosinophilia >25% with exclusion 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.<sup>[2,3,10,15]</sup> 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,  $\times 400$ ). (b) (Cytocentrifuge, PAP,  $\times 400$ ). (c) (Cell block, H and E,  $\times 400$ )

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.<sup>[16]</sup> 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.

## Financial support and sponsorship

Nil.

## Conflicts of interest

There are no conflicts of interest.

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