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Case Report

Unusual case of bullous emphysema with superimposed pneumonia ☆

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ABSTRACT

Bullous emphysema is a chronic obstructive pulmonary disease (COPD) that results from chronic inflammation of the lung parenchyma leading to alveolar destruction. Etiology includes tobacco smoking and alpha-1 antitrypsin deficiency. In this article, we present a rare case of bullous emphysema in a nonsmoker with no genetic predisposition or social risk factors presenting with productive cough, fatigue, and shortness of breath. The patient was diagnosed with bullous emphysema with superimposed pneumonia based on clinical and radiological findings. The patients acute complaints were treated successfully with antibiotics, supplemental oxygen, systemic steroids, and, nebulizer treatments. With this case report the authors highlight an unusual presentation of pneumonia in a patient with underlying bullous emphysema. Environmental exposure is often overlooked and the outcomes cannot be turned to favor without a comprehensive approach in patient management from history and physical to deciding the right treatment and follow-up protocols.

☆ Competing Interests: The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Introduction

Bullous emphysema is classified as a COPD. Emphysema is characterized by chronic inflammation secondary to a noxious stimulus or irritants such as smoking, leading to emphysematous destruction of the lung parenchyma causing enlargement of the alveolar walls with air-filled spaces also known as "Bullae'' which are greater than 1 cm [1]. Bullous emphysema globally affects 5% of the population. Etiologies of bullous emphysematous are either hereditary environmental or social factors. Alpha-1 antitrypsin deficiency (A1AD OR AATD), an autosomal dominant genetic condition which affects the lungs and liver primarily, and tobacco/cigarette smoking are reported to be the leading causes [2]. Less frequently reported causes of emphysema include fiberglass inhalation, cocaine/marijuana use, sarcoidosis, and connective tissue disorders such as Marfan's syndrome, Ehlers-Danlos syndrome [2]. Environmental/occupational hazards and air pollutants such as dust, fumes, and sand may also contribute to the development of COPD and can often be overlooked in daily clinical practice [3]. Repetitive insults can lead to severe complications and damage to the lung parenchyma affecting pulmonary function and physiology. Here, we report a case of a 42-year-old nonsmoker with no underlying past medical history or comorbidities who presented with symptoms of productive cough, fatigue, shortness of breath, and radiologic finding of emphysematous changes bilaterally with multiple prominent pulmonary bullae bilaterally and multiple associated regions of bronchiectasis and bronchial wall thickening.

Case report

A 42-year-old woman presented to the ED with progressively worsening productive cough, fatigue, shortness of breath, and palpitations for 1 week duration. The patient noticed initial dyspnea and fatigue while walking more than 1 block duration which is new from her baseline as she was able to carry out regular activity with no limitations prior to presentation. She denied any chest pain, fever, nausea, vomiting, diarrhea, and abdominal pain. She reports no past medical history or smoking/tobacco use or illicit drugs use. The patient immigrated from Sierra Leone to the United States ten years ago. She works as a nursing aide at a skilled nursing facility and has close contact with multiple sick patients. She does report significant second-hand smoke exposure in her apartment complex for a 5-year duration. She has received 2 doses of the Pfizer vaccination. Upon the physical exam, the patient was fatigued and in mild respiratory distress with increased work of breathing and had diffuse rales and rhonchi bilaterally on auscultation. The remainder of the physical exam was unremarkable. Oxygen saturation (SPO2) was 98% on room air at rest and decreased to 69% on room air after ambulation. The patient was placed on 3 liters of oxygen via nasal cannula for dyspnea, which improved her SPO2 to 99%, she was admitted to the telemetry unit and diagnostic workup was initiated. Chest computed tomography showed emphysematous changes bilaterally with multiple prominent pulmonary bullae bilaterally (blue arrows), the largest in the anterior left upper lobe and posterior right lower lobes (Figs. 1–3). There were multiple associated regions of bronchiectasis (red arrowheads) and bronchial wall thickening. There was also dilatation of the pulmonary vasculature (red arrows), which should measure <3 cm just proximal to the bifurcation of the left and right pulmonary arteries (Figs. 1–3). Alpha-1 Antitrypsin levels were within normal limits. The patient tested negative for COVID-19 and reported no previous history of COVID-19. She also presented 2 years following the onset of the COVID-19 pandemic.

Empirical treatment with ceftriaxone and azithromycin was started initially and later switched to broad-spectrum antibiotics with vancomycin, cefepime, and doxycycline as the respiratory culture was positive for gram-negative bacillus *Pseudomonas aeruginosa* and moderate yeast growth of *Candida albicans*. Over the course of 6 days on the telemetry unit, her symptoms steadily improved. Alongside antibiotics, treatment regimen included supplemental O2, methylprednisolone, and albuterol/ipratropium bronchodilators. The patient was discharged on levofloxacin and doxycycline and home O2 for 14 days. The patient was counseled and educated on environmental exposure being the core cause of her symptoms and was advised to avoid any exposure to dust, smoke (passive), or irritants. During 2 two-week follow up the patients symptoms improved significantly. Spirometry results reported FEV1 of 23% and FEV1/FVC ratio of 65%. Patient continued using a Trelegy Ellipta 100-62.5-25 mcg inhaler once daily, Albuterol Sulfate HFA Aerosol Solution 108 (90 Base) mcg/ACT as needed.

Discussion

Bullous emphysematous is primarily seen in males with a significant past medical history of smoking, underlying systemic disease, and occupational/environmental exposures. Cigarette smoking is the single most significant risk factor predisposing to the development of COPD [1]. Differential diagnosis of bullous emphysema includes idiopathic giant bullous emphysematous, granulomatosis with polyangiitis, Sjogren disease, Fabry disease, neurofibromatosis, bullous sarcoidosis, and cystic lesions of the lung [4]. Environmental and occupational exposures and exposure to air pollutants can also contribute to COPD onset [5]. Other pulmonary irritants include fumes, sand, dust, silica, coal dust, agriculture work, and biomass fuels [6].

This case report underlines environmental and second hand smoke exposures as significant risk factors for bullous emphysema development and progression. Our patient risk factors were multifactorial which included being a migrant from Sierra Leone with biomass fuel smoke exposure, dust exposure, extensive second hand smoke exposure, and occupational exposure to individuals with potential respiratory illnesses. When observing diagnostic imaging in patients with environmental/hazard exposure-induced emphysema compared to A1AT-induced emphysema, the affected lung region is mainly centrilobular, whereas in A1AT deficiency panacinar damage is observed. In our patient, radiologic imaging showed

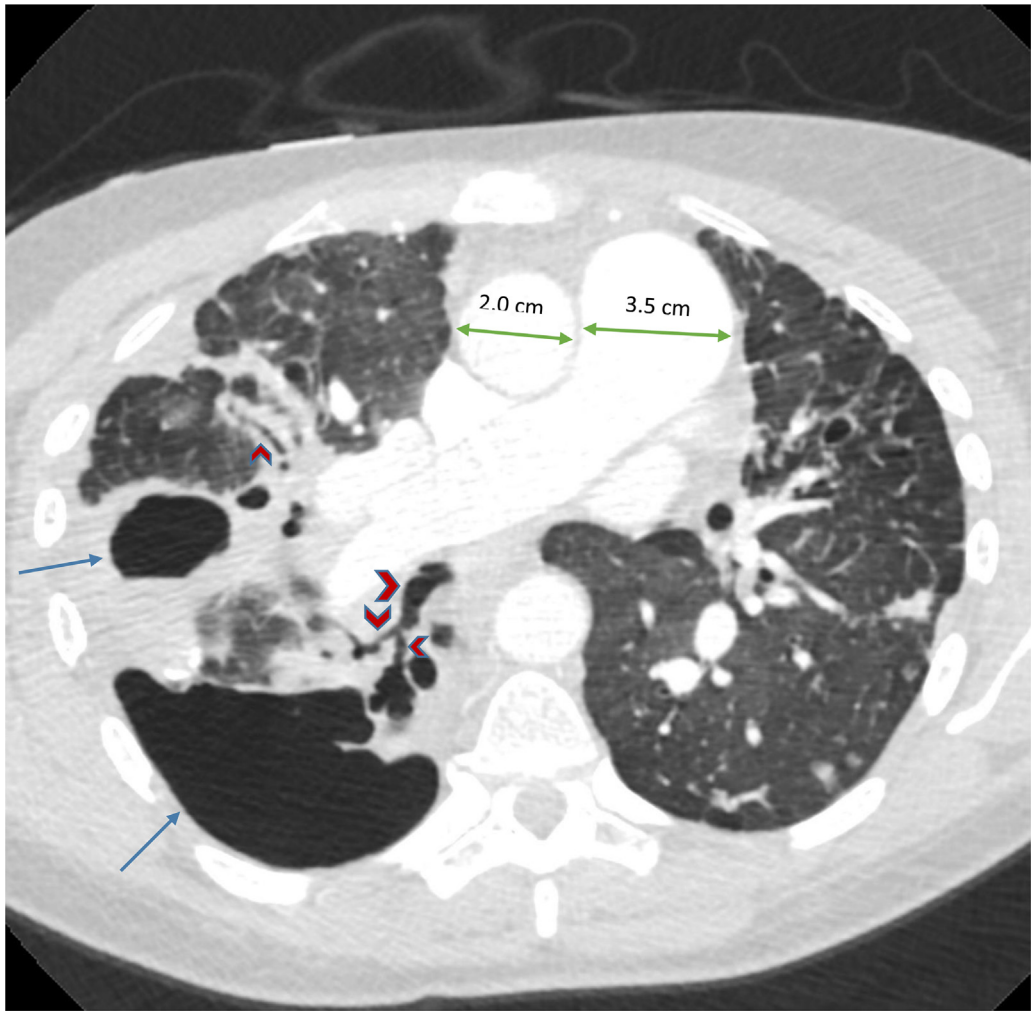

Fig. 1 – An axial image of a CTA chest for pulmonary emboli displayed in the lung window. Extensive emphysematous changes bilaterally with multiple prominent pulmonary bullae (blue arrows), the largest within the posterior right lobe. There are multiple associated regions of bronchiectasis (red arrowheads) and bronchial wall thickening. Dilatation of the pulmonary vasculature (green arrows) is also apparent.

emphysematous changes bilaterally with multiple prominent pulmonary bullae bilaterally and multiple associated regions of bronchiectasis and bronchial wall thickening.

The patient is from a developing nation where coal and biomass fuels are required. Burning wood, charcoal, crop waste, and animal dung over open fires or on inefficient stoves can produce extremely high levels of indoor air pollution in Sierra Leone and other low and middle-income countries [7]. Combusting these various energy sources with a mixture of pollutants has been associated with causing acute respiratory infections, COPD, lung cancer, and asthma [8]. A case-control study (N = 120) conducted in Spain hypothesized that wood smoke exposure could be a risk factor for COPD. The study determined that exposure to wood or charcoal exposure was strongly associated with COPD (adjusted for age and smoking) [9]. Wood or charcoal alone independently increased the risk of COPD (odds ratio 1.8 and 1.5, respectively, but only the combination of both was statistically significant). In Mexico, COPD-related symptoms such as severe dyspnea and cough were seen in females in their 60*s* living in the countryside [10]. Another study conducted in Colombia showed the development of COPD in low socioeconomic females with exposure to wood smoke in 50% of the COPD cases [11]. A meta-analysis conducted by Pathak et al. looked at the association between indoor air pollution and the risk of COPD in 35 studies (N = 73,122). The analysis showed that exposure to indoor air pollution due to biomass fuels increased the risk of COPD by 2.65 (CI 2.13-3.13 N = 73,122). The risk was higher in African regions (OR 3.19), Asia (OR 2.88), South America (OR 2.15), Europe (OR 2.30) and North America (OR 2.14) [12]. Smith et al. [13] suggested that wood smoke exposure could equal approximately 20 years of active exposure to tobacco smoke. Another study also looked at the effects of wood smoke exposure on MMP activity and expression from patients exposed to wood, tobacco smoke, and the control subjects. It concluded that chronic exposure to wood smoke increases MMP activity and expression, which can lead to lung damage observed in COPD patients with tobacco exposure [14].

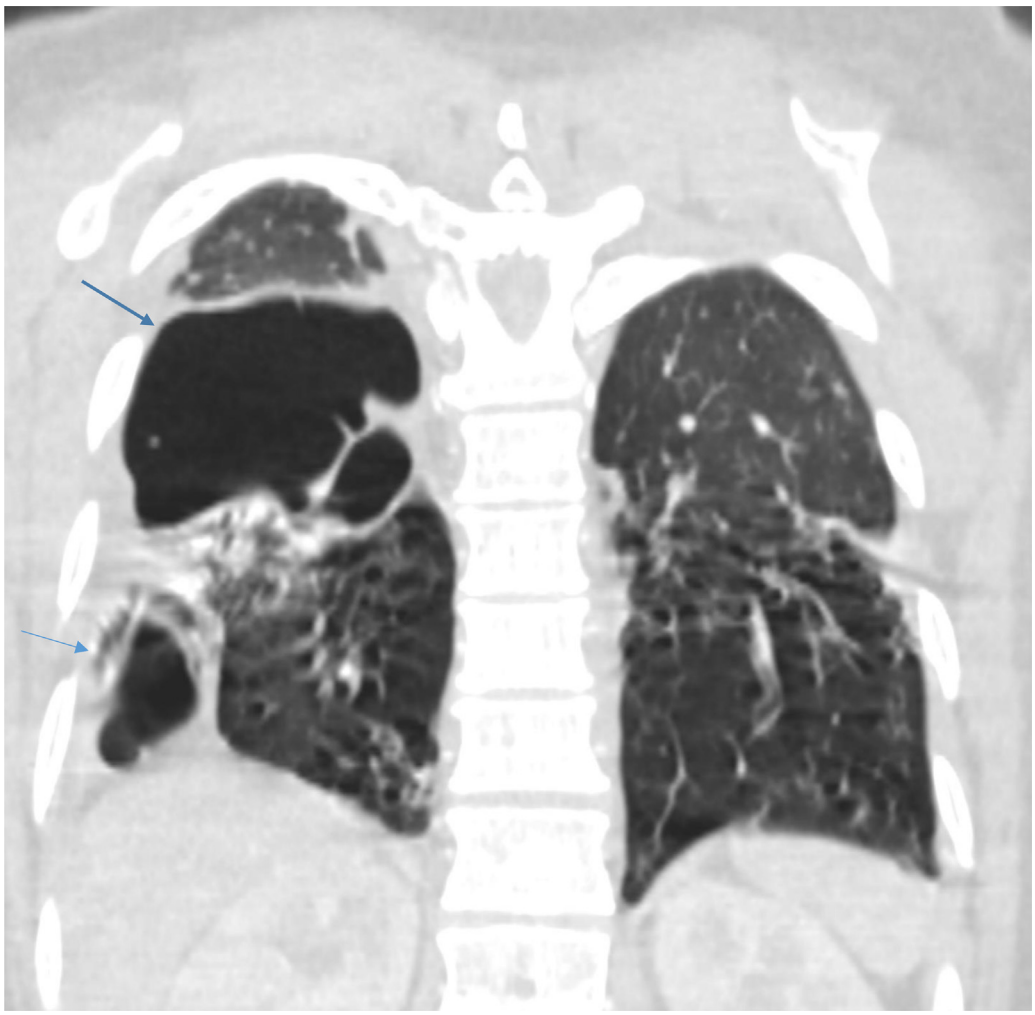

Fig. 2 – Another representative coronal image of the same study again displayed in the lung window displaying the right-sided pulmonary bullae (blue arrows).

Prolonged secondhand smoke exposure has been shown to increase respiratory symptoms and increase susceptibility to asthma and COPD [15]. Hagstad et al. studied secondhand smoking (environmental tobacco smoke [ETS]) as a risk factor for COPD in nonsmokers. Three cross-sectional studies were used (N = 2182), and the results concluded increased COPD with increased ETS exposure: No ETS (4.2%), ETS ever at home (8.0%) and ETS ever at home and at both previous and current work (14.7%). ETS in multiple settings (work, home, previous/current workplace) was strongly associated with COPD (OR 3.80; CI 1.29-11.2) [15]. Two meta-analyses have found elevated risk estimates for COPD of 1.20 and 1.66 among nonsmokers who had been exposed to secondhand smoking [16,17] and a Chinese study of 6497 adults found an increased odds ratio for spirometry-defined COPD of 1.48 in those individuals who had been exposed to secondhand smoking at home and work for at least 40 hours a week for more than 5 years [18]. Flexeder et al. [19] also discovered that those exposed to lifelong secondhand smoking in the general population had an elevated odds ratio for COPD of 1.24 (Without making any adjustments for age, sex, or smoking status) . A publication from Denmark by Korsbaek et al., with a cohort of 20,421 adults, recorded respiratory symptoms, lung function, asthma, and COPD. The findings from this study demonstrate a dose-response relationship between exposure to secondhand smoke and decreased lung function, as well as elevated risks of dyspnea, wheezing, coughing, and asthma in the general population [20]. In the general population, people who had been exposed to secondhand smoke throughout their lives had a 1.4-fold greater risk of asthma and a 1.2-fold increased risk of COPD. They also had a 1.6-2.1-fold increased risk of respiratory symptoms and 3%-4% reductions in FEV1% predicted and FVC% predicted [20].

It can be determined that a variety of environmental exposures, including biomass fuels and secondhand smoking, contributed to the patient's bullous emphysema. Environmental exposure to high levels of indoor air pollution in a region where burning coal and biomass fuels is commonplace. This in addition to substantial exposure to secondhand smoke in the apartment complex also increased her risk of acquiring COPD.

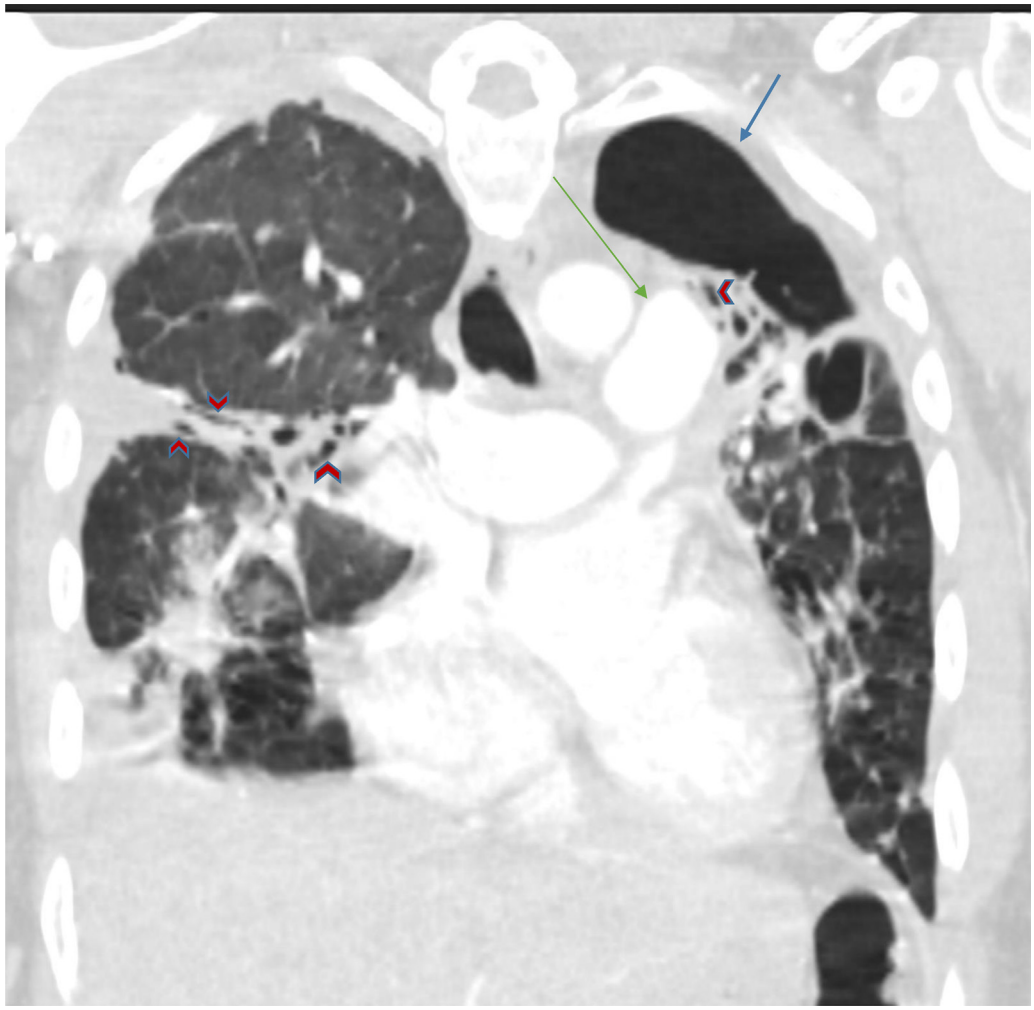

Fig. 3 – Another representative coronal image displayed in the lung window highlighting the extensive emphysematous changes bilaterally with another large bulla (blue arrow) in the LUL and multiple regions of bronchiectasis bilaterally (red arrowheads). The fibrotic changes within the LUL are causing traction on the left main pulmonary artery, resulting in it being directed more superiorly (green arrow) rather than laterally.

Conclusion

The authors report a unique case of COPD bullous emphysema with superimposed pneumonia in a woman of African origin with no hereditary predispositions or social risk factors. Early detection and recognition of environmental and hazardous exposure can help limit complications and progression of bullous emphysema. Early preventative and management modalities can improve the quality of life and further decrease disease burden and mortality.

Patient consent

Written informed consent for the publication of this case report was obtained from the patient.

REFERENCES

[1] Siddiqui NA, Mansour MK, Nookala V. Bullous emphysema. StatPearls [Internet] [Updated 2022 July 4], Treasure Island (FL): StatPearls Publishing; 2022. Available from https://www.ncbi.nlm.nih.gov/books/NBK537243/ .

[2] Perkins JT, Choate R, Mannino DM, Browning SR, Sandhaus RA. Benefits among patients with alpha-1 antitrypsin deficiency enrolled in a disease management and Prevention Program. Chron Obstr Pulm Dis 2017;4(1):56–64. doi:10.15326/jcopdf.4.1.2016.0161.

[3] Becklake MR. Occupational exposures: evidence for a causal association with chronic obstructive pulmonary disease. Am Rev Respir Dis 1989;140(3_pt_2):S85–91. doi:10.1164/ajrccm/140.3_pt_2.s85.

[4] Teramoto S, Fukuchi Y. Bullous emphysema. Curr Opin Pulm Med 1996;2(2):90–6. doi:10.1097/00063198-199603000-00003.

[5] Walter R, Gottlieb DJ, O'Connor GT. Environmental and genetic risk factors and gene-environment interactions in

the pathogenesis of chronic obstructive lung disease. Environ Health Prosepect 2000;108:733–42.
[6] Weinmann S, Vollmer WM, Breen V, Heumann M, Hnizdo E, Villnave J, et al. COPD and occupational exposures: a case-control study. J Occupat Environ Med 2008;50(5):561–9. doi:10.1097/jom.0b013e3181651556.
[7] KC R, Shukla SD, Gautam SS, Hansbro PM, O'Toole RF. The role of environmental exposure to non-cigarette smoke in lung disease. Clin Transl Med 2018;7(1):39. doi:10.1186/s40169-018-0217-2.
[8] Kurmi OP, Lam KB, Ayres JG. Indoor air pollution and the lung in low- and medium-income countries. Eur Respir J 2012;40(1):239–54. doi:10.1183/09031936.00190211.
[9] Orozco-Levi M. Wood smoke exposure and risk of chronic obstructive pulmonary disease. Eur Respir J 2006;27(3):542–6. doi:10.1183/09031936.06.00052705.
[10] Sandoval J, Salas J, Martinez-Guerra ML, Gómez A, Martinez C, Portales A, et al. Pulmonary arterial hypertension and COR pulmonale associated with chronic domestic woodsmoke inhalation. Chest 1993;103(1):12–20. doi:10.1378/chest.103.1.12.
[11] Dennis RJ, Maldonado D, Norman S, Baena E, Martinez G. Woodsmoke exposure and risk for Obstructive Airways disease among women. Chest 1996;109(1):115–19. doi:10.1378/chest.109.1.115.
[12] Pathak U, Gupta NC, Suri JC. Risk of COPD due to indoor air pollution from biomass cooking fuel: a systematic review and meta-analysis. Int J Environ Health Res 2019;30(1):75–88. doi:10.1080/09603123.2019.1575951.
[13] Smith KR, Aggarwal AL, Dave RM. Air pollution and rural biomass fuels in developing countries: a pilot village study in India and implications for research and policy. Atmosph Environ (1967) 1983;17(11):2343–62. doi:10.1016/0004-6981(83)90234-2.
[14] Montaño M, Beccerril C, Ruiz V, Ramos C, Sansores RH, González-Avila G. Matrix metalloproteinases activity in COPD associated with wood smoke. Chest 2004;125(2):466–72. doi:10.1378/chest.125.2.466.
[15] Hagstad S, Bjerg A, Ekerljung L, Backman H, Lindberg A, Rönmark E, et al. Passive smoking exposure is associated with increased risk of COPD in never smokers. Chest 2014;145(6):1298–304. doi:10.1378/chest.13-1349.
[16] Fischer F, Kraemer A. Meta-analysis of the association between second-hand smoke exposure and ischaemic heart diseases, COPD and stroke. BMC Public Health 2015;15(1). doi:10.1186/s12889-015-2489-4.
[17] Lee PN, Forey BA, Coombs KJ, Hamling JS, Thornton AJ. Epidemiological evidence relating environmental smoke to COPD in lifelong non-smokers: A systematic review. F1000Research 2018;7:146. doi:10.12688/f1000research.13887.1.
[18] Yin P, Jiang C, Cheng K, Lam T, Lam K, Miller M, et al. Passive smoking exposure and risk of COPD among adults in China: the Guangzhou Biobank Cohort Study. Lancet North Am Ed 2007;370(9589):751–7. doi:10.1016/s0140-6736(07)61378-6.
[19] Flexeder C, Zock J-P, Jarvis D, Verlato G, Olivieri M, Benke G, et al. Second-hand smoke exposure in adulthood and lower respiratory health during 20 year follow up in the European Community Respiratory Health Survey. Respir Res 2019;20(1):33. doi:10.1186/s12931-019-0996-z.
[20] Korsbæk N, Landt E, Dahl M. Second-hand smoke exposure associated with risk of respiratory symptoms, asthma, and COPD in 20,421 adults from the general population. J Asthma Allergy 2021;14:1277–84. doi:10.2147/jaa.s328748.