Abstract

OBJECTIVES: The most common chemical substances used as mass control agents are chloroacetophenone, chlorobenzylidene malononitrile, and oleoresin capsicum. These agents not only have local and rapid effects but also have systemic and long-term effects. The aim of the present study was to discuss the patterns of tear gas exposure and to investigate its effects on respiratory functions.

MATERIALS AND METHODS: A face-to-face survey was conducted in 86 individuals who had been exposed to tear gas indoor and outdoor during the public protests in June 2013.

RESULTS: The most frequently reported respiratory complaints included cough, dyspnea, phlegm, and chest pain. Spirometry measurements including forced vital capacity (FVC) and forced expiratory volume in 1 second (FEV₁) were also performed. Indoor exposers have lower mean % predicted FVC and FEV₁ values than outdoor exposers. All complaints and signs were more common in indoor exposure to tear gas than in outdoor exposure.

CONCLUSION: Safety of the chemicals used as mass control agents during protests is doubtful as these agents are associated with several health risks.

KEYWORDS: Respiratory effects, spirometry, tear gases

INTRODUCTION

There are several different chemical substances used as mass control agents by public authorities, government agencies, or military forces in order to neutralize, deter, or disperse the masses or individuals in times of disturbance, such as uprisings or wars. Owing to their fast-onset and short duration of action, chloroacetophenone (CN), chlorobenzylidene malononitrile (CS), and oleoresin capsicum (OC), collectively known as tear gases, have been the most commonly used agents to suppress protests. These agents, typically used through aerosolization methods (spray, smoke, grenade, and cartridges, etc.), temporarily neutralize exposed individuals due to fast and intense irritation they cause on the eyes, nose, mouth, skin, and respiratory system. While they rarely have severe systemic effects, they may still result in serious side effects and even death when exposed to high concentrations [1].

Chlorobenzylidene malononitrile and OC are the most commonly used protest control agents by the police forces in Turkey [2]. These gases, used on the grounds of their high safety ranges and incapability to cause any long-term health problems, are now known to have potential chronic effects and a much worse safety profile than expected.

Oleoresin capsicum contains capsaicin, a substance that directly acts as an irritant and also causes neurogenic inflammation by inducing the release of substance P through its effects on the peripheral sensory nerve endings [3,4]. Respiratory problems noted after acute exposure to tear gases include a burning sensation in the throat, cough, wheezing, shortness of breath, and laryngospasm, while death due to respiratory failure has also been reported [5,6].

The major effect of CS is irritation on the mucous membranes, and this irritation is enhanced under warm and humid air conditions [7]. Inhalation toxicity studies demonstrated that exposure to high levels of CS may result in chemical pneumonitis and fatal pulmonary edema. In addition, cases of heart failure, hepatocellular damage, and death have been reported in adults exposed to high concentrations of CS [7].

Police forces extensively used tear gases during the Gezi Park Resistance, which included protests performed at several cities in Turkey between June and July 2013. During that period, individuals joining the protests in Ankara and even...
residents of the regions where the protests occurred were exposed to tear gases. In Ankara, police forces dispersed the protests mostly during the evening hours and every day in June. At the beginning of the protests, police forces used pepper and tear gases by shooting canisters and driving water cannon vehicles, and a common observation was that the police force was not adequately trained or supervised for the use of tear gases to disperse these peaceful protests. Injuries caused by direct trauma due to targeted horizontal shooting of the gas canisters by police forces instead of vertical shooting to the air accounted for almost 60% of all recorded injuries [8].

Reports and visual recordings relevant to that period indicate that tear gases were also used indoors, and even some of these indoor environments were those providing healthcare services [9]. Tear gases affected not only the protesters but also the individuals living in the regions where the protests occurred were exposed.

In a previous review article, Hu et al. [7] addressed the effects of tear gases on respiratory health in real-life. However, there are a limited number of in vivo field studies evaluating the respiratory functions of protesters exposed to tear gases used during protests. The purpose of the present study was to discuss the patterns of tear gas exposure and to investigate the effects on respiratory functions of 86 subjects who were exposed to tear gas in Ankara in the 2013 Gezi Park Protests and volunteered to participate in the study.

MATERIALS AND METHODS

A face-to-face survey was conducted using a questionnaire, and spirometry measurements were performed in 86 (52 male and 34 female, mean age: 29.5 years) individuals who had been exposed to tear gas during the public protests in June 2013 in Ankara. The time interval between gas exposure and performance of questionnaire and spirometry was 1-4 days. The Turkish Police Forces mostly used OC via spraying and CS within gas canisters. The present study was organized by Turkish Thoracic Society. The Ethics Committee of Istanbul University approved the study (Date: August 22, 2014, No: 1277).

Spirometry measurements including forced vital capacity (FVC) and forced expiratory volume in 1 second (FEV₁) were performed using a CPFS/DUSB spirometer (Medical Graphics Corporation, St. Paul, MN, USA) in accordance with the standards recommended by the American Thoracic Society, including reproducibility criteria that the two largest FVCs and FEV₁s of at least three acceptable measurements be within 0.200 L [10]. Raw spirometry data (FVC and FEV₁) were converted to percentage of predicted % pred FVC and % pred FEV₁ for each subject to normalize for height, gender, age, and race as per standard practice.

Statistical Analysis

Questionnaire items included the following: general information (age, gender, occupation, graduate, smoking status, and previous diseases), tear gas exposure patterns (time and place of exposure, color and odor of gas, availability of medical management, and complaints related to gas exposure), and symptoms after exposure (dyspnea, cough, phlegm, chest pain, runny nose, eye redness, and dermatitis). Statistical analysis was performed using the IBM Statistical Package for the Social Sciences 20.0 software package (IBM SPSS Statistics Corp.; Armonk, NY, USA). Non-parametric and parametric variables were compared using chi-square test and two-sample t-test, respectively. A p value <0.05 was considered as significant.

RESULTS

The mean age of the protesters (52 male and 34 female) was 29.5±10.3 (16-62) years. Table 1 shows the general characteristics of the subjects.

Almost half of the subjects were smokers. Majority of the subjects were university graduates and students. Of the subjects, 78% were exposed to tear gas >24 h ago. Almost 70% of the subjects were exposed to tear gas outdoor.

Figure 1 shows the respiratory, nasal, eye, and skin complaints of the subjects.

Table 2 shows the respiratory complaints of the subjects based on smoking status, exposure place, and exposure time. Smokers reported higher phlegm rates than non-smokers (58.5% vs. 17.5%, p=0.0001). Subjects exposed to tear gases indoors had higher rates of dyspnea (78.3% vs. 65.5%), cough (78.3% vs. 68.4%), and chest pain (50.0% vs. 28.6%), although the differences did not reach statistical significance. Subjects exposed to tear gases >24 h ago more commonly

| Table 1. General characteristics of the subjects |
| Aspect | n | % |
| Smoking status |
| Smoker | 45 | 52.3 |
| Non-smoker | 41 | 47.7 |
| Occupation |
| University student | 32 | 37.2 |
| Others | 34 | 39.5 |
| Non-responders | 20 | 23.3 |
| Education |
| University graduate | 29 | 33.7 |
| University student | 32 | 37.2 |
| Others | 25 | 29.1 |
| Time of tear gas exposure |
| Within 24 h | 18 | 20.9 |
| >24 h ago | 67 | 77.9 |
| Non-responder | 1 | 1.2 |
| Place of tear gas exposure |
| Outdoor | 59 | 68.6 |
| Indoor | 23 | 26.7 |
| Non-responder | 4 | 4.7 |
reported chest pain (37.3% vs. 16.7%), but the difference was not statistically significant.

Table 3 shows the expiratory flow rates of the subjects based on smoking status, exposure place, and exposure time.

The mean % predicted FVC (96.1% vs. 100.1%) and FEV<sub>1</sub> (94.7% vs. 99.8%) values of the subjects who had been exposed to gases indoors were lower than those exposed outdoors. The mean % predicted FVC (94.5% vs. 100.5%), FEV<sub>1</sub> (94.5% vs. 99.4%), and maximal mid-expiratory flow rate (MMFR) (92.5% vs. 106.6%) values of the subjects who had been exposed to gases within 24 h were lower than those of the subjects exposed to gases at least 24 h ago. None of the differences reached statistical significance.

Table 4 shows the pulmonary functions based on smoking status and exposure place.

Non-smokers who were exposed to tear gases indoor had lower % predicted FVC (94.3% vs. 99.8%), FEV<sub>1</sub> (89.5% vs. 98.0%), and FEV<sub>1</sub>/FVC (99.9% vs. 101.1%) values than those outdoor exposers. There were no significant differences.

Table 5 shows the pulmonary functions based on smoking status and exposure time.

Smokers had low % predicted FVC (93.5% vs. 102.7%, p=0.032) and FEV<sub>1</sub> (94.3% vs. 103.5%, p=0.026) values when exposed to tear gases within 24 h.

DISCUSSION

The present study including subjects who had participated in the Gezi Park Protests in Ankara and been exposed to OC and CS tear gases demonstrated that the most common complaints, in order of frequency, were eye redness, runny nose,
cough, dyspnea, phlegm, chest pain, and dermatitis. In line with the literature, the most common symptom was eye redness, whereas the most frequently reported respiratory complaints included cough, dyspnea, phlegm, and chest pain. In 2015, Dimitroglou et al. [11] reported that ocular findings, such as lacrimation, burning eyes, blepharospasm, conjunctivitis, and vision loss due to exposure to CS, can persist for variable durations, from minutes to weeks. Among the most common respiratory symptoms, cough, dyspnea, and chest tightness resolve in a short time, and these symptoms may also be accompanied by findings, such as laryngeal obstruction, hypersensitivity pneumonitis, reactive airways dysfunction syndrome, and laryngospasm [11].

In the present study, comparison of the subjects’ complaints based on their smoking status showed that phlegm was a significantly more common complaint among smokers than non-smokers (58.5% vs. 17.5%, p=0.0001), whereas no significant difference was noted between these two groups in terms of the frequency of dyspnea, cough, chest pain, eye redness, or runny nose. We believe that the significantly higher rate of phlegm as reported by smokers exposed to tear gas can be attributed to high levels of exposure, in addition to the fact that subjects described and reported viscous nasal flow and sputum, which increased in response to gas exposure, as phlegm. We also suppose that this finding is more in line with a real-life scenario contrary to laboratory conditions. For instance, a study performed in 1985 of capsaicin in volunteers demonstrated that the level and duration of bronchoconstriction caused by exposure do not differ between healthy subjects, smokers, and individuals with asthma [12], whereas in real-life conditions, a case of fatal bronchospasm due to OC exposure was previously reported [13].

We found that the frequencies of dyspnea (78.3% vs. 65.5%), cough (78.3% vs. 68.4%), and chest pain (50.0% vs. 28.6%) were higher among individuals with a history of indoor gas exposure than those exposed outdoors, but the differences were not statistically significant. In a previous study, Karagama et al. [14] evaluated 34 individuals exposed to CS spray in a non-ventilated bus and reported that respiratory symptoms, such as inability to breathe, chest tightness, cough, wheezing, and asthma exacerbation, occur in 67% of the individuals within the first hour after exposure, and these symptoms persist in 41% and 14% on the first and the 10th month of exposure, respectively. Moreover, a growing amount of evidence in the literature indicates that indoor exposure to tear gases may have fatal outcomes. Toprak et al. [13] re-evaluated 10 previously reported fatal cases of CN, CS, and OC exposures and noted that 6 of these fatalities are associated with a history of indoor exposure to tear gas. While spirometric assessments performed in the present study did not indicate a statistically significant difference based on indoor and outdoor exposure patterns, individuals exposed to tear gas indoors had numerically lower % predicted FVC (96.1% vs. 100.1%) and FEV₁ (94.7% vs. 99.8%) values than those exposed to tear gas outdoors. When the same comparison was performed among non-smokers, FVC and FEV₁ values were found to be 94.3% vs. 99.8% and 89.5% vs. 98.0% in those with a history of indoor and outdoor exposures, respectively. This finding suggests that when smoking, as a confounding factor, is excluded from the analysis, the use of tear gases indoors results in a more pronounced decrease in pulmonary function parameters.

Chest pain was more common among those exposed to tear gases >24 h before the assessments than those exposed within the last 24 h (37.3% vs. 16.7%), suggesting that chest pain can be a late symptom of tear gas exposure. In the study by Anderson et al. [15], 19% of 184 individuals interviewed on average within 5 days after CS exposure reported chest pain. In the study by Wheeler et al. [16] including 597 individuals exposed to CS, cardiac symptoms, such as hypoten-
Assessment of smokers exposed to tear gases showed that the FVC and FEV$_1$ values measured among those exposed within the last 24 h were significantly lower than the FVC (93.5% vs. 102.7%, p=0.032) and FEV$_1$ (94.3% vs. 103.5%, p=0.026) values of those exposed >24 h ago. Studies performed on experimental animal models and human lung epithelial cells indicated that smoking results in an early inflammatory response by stimulating capsaicin-sensitive sensory neurons in the airways of rodents [17], and capsaicin found in pepper gas leads to acute pulmonary inflammation and respiratory cell damage via transient receptor potential vanilloid 1 and the other vanilloid receptor proteins [18]. The decrease in FVC and FEV$_1$ levels as demonstrated among smokers in the present study can be a result of the same neurogenic inflammation mechanisms. Moreover, tear gases are toxic irritants that can possibly have synergistic interactions with tobacco smoke. Arbak et al. showed significantly lower mean FEV$_1$/FVC and % predicted MMFR in smoker tear gas-exposed subjects than those in non-smoker ones (19). In a study investigating chlorine gas, a gas that damages the airway epithelium through different mechanisms, permanent airway obstruction was reported in smokers inhaling chlorine gas, and it was asserted that chlorine exposure and the damage caused by smoking may have additive or synergistic effects [20].

The major limitation of our study was the small sample size as only 86 subjects agreed to participate in the study due to fear of stigmatization, whereas thousands of individuals were indeed exposed to tear gases in Ankara. Another limitation is the subjectivity of information provided by the protesters regarding the environment they were exposed to tear gases (indoors or outdoors), the number of exposures, and gas concentrations, since exposures occurred hours ago. Moreover, we could not include protesters with more severe symptoms into the present study as they had already been referred to a hospital.

In conclusion, among individuals exposed to tear gases, complaints of eye redness, runny nose, cough, dyspnea, phlegm, chest pain, and dermatitis, in order of frequency, were more common in smokers. One-fourth of the protesters had a history of indoor exposure, and all complaints and signs were more common in these individuals than those exposed to tear gases outdoors. Among smokers, the decline in FEV$_1$ and FVC was significantly greater within the first 24 h after exposure than later hours, indicating a possible synergistic interaction between the gases and tobacco smoke.

Safety of the chemicals used as mass control agents during protests is doubtful as these agents are associated with several health risks, and the duty of scientists is not to straighten out these doubts but to lead the way for elimination of all factors threatening human health at their source.

**Informed Consent:** Written informed consent was obtained from subjects exposed to the tear gases and participated in this study.

**Conflict of Interest:** The authors have no conflicts of interest to declare.

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

**REFERENCES**

1. Schepp LJ, Slaughter RJ, Mc Bride DI. Riot control agents: the tear gases CN, CS and OC—a medical review. J R Army Med Corps 2015;161:94-9. [CrossRef]
2. Chemical Riot Control Agents. First Edition, August 2011, Ankara, Turkish Medical Association Publication (In Turkish).
3. jancs6 Y, jancs6-G6bor A, szolcs6nay I. direct evidence for neurogenic inflammation and its prevention by denervation and by treatment with capsaicin. Br J Pharmacol Chemother 1967;31:138-51. [CrossRef]
4. Sanico AM, Atsuta S, Proud D, et al. Dose-dependent effects of capsaicin nasal challenge: in vivo evidence of human airway neurogenic inflammation. J Allergy Clin Immunol 1997;100:632-41. [CrossRef]
5. Smith J, Greaves I. The use of chemical incapacitant sprays: a review. J Trauma 2002;52:595-600. [CrossRef]
6. Steffee CH, Lantz PE, Flannagan LM, et al. Oleoresin capsicum (pepper) spray and “in-custody deaths.” Am J Forensic Med Pathol 1995;16:185-92. [CrossRef]
7. Hu H, Fine J, Epstein P, et al. Tear gas-harassing agent or toxic chemical weapon? JAMA 1989;262:660-3. [CrossRef]
8. Gezi Park Protests. Amnesty International, 2013, Brutal Denial of the Right to Peaceful Assembly in Turkey, Available from: http://www.amnestyusa.org/sites/default/files/eur440222013en.pdf
9. Medical World. Turkish Medical Association, 2013, Ankara/Turkey. Available from: http://www.ttb.org.tr/TD/TD197/index.pdf
10. Standardization of Spirometry, 1994 Update. American Thoracic Society. Am J Respir Crit Care Med 1995;152:1107-36. [CrossRef]
11. Dimitroglou Y, Rachiotis G, Hadjichristodoulou C. Exposure to the riot control agent CS and potential health effects: a systematic review of the evidence. Int J Environ Res Public Health 2015;12:1397-411. [CrossRef]
12. Fuller RW, Dixon CM, Barnes PJ. Bronchoconstrictor response to inhaled capsaicin in humans. J Appl Physiol (1985) 1985;58:1080-4. [CrossRef]
13. Toprak S, Ersoy G, Hart J, et al. The pathology of lethal exposure to the Riot Control Agents: Towards a forensics-based methodology for determining misuse. J Forensic Leg Med 2015;29:36-42. [CrossRef]
14. Karagama YG, Newton JR, Newbegin CJ. Short-term and long-term physical effects of exposure to CS spray. J R Soc Med 2003;96:172-4. [CrossRef]
15. Anderson PJ, Lau GS, Taylor WR, et al. Acute effects of the potent lacerator o-chlorobenzylideneononitrile (CS) tear gas. Hum Exp Toxicol 1996;15:461-5. [CrossRef]
16. Wheeler H, MacLehose R, Euripidou F, et al. Surveillance into crowd-control agents. Lancet 1998;352:991-2. [CrossRef]

17. André E, Campi B, Materazzi S, et al. Cigarette smoke-induced neurogenic inflammation is mediated by α,β-unsaturated aldehydes and the TRPA1 receptor in rodents. J Clin Invest 2008;118:2574-82. [CrossRef]

18. Reilly CA, Taylor JL, Lanza DL, et al. Capsaicinoids cause inflammation and epithelial cell death through activation of vanilloid receptors, toxicological sciences. Toxicol Sci 2003;73:170-81. [CrossRef]

19. Arbak P, Başer I, Kumbasar OO, et al. Long term effects of tear gases on respiratory system: analysis of 93 cases. Scientific World Journal 2014;2014:963638. [CrossRef]

20. White CW, Martin JG. Chlorine gas inhalation, human clinical evidence of toxicity and experience in animal models. Proc Am Thorac Soc 2010;7:257-63. [CrossRef]