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Mid to Longer-term Harmful Effects of Riot Control Agents

Gösteri Kontrol Ajanlarının Uzun Dönem ve Zararlı Etkileri

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ABSTRACT

Civil authorities and armed forces have not infrequently had recourse to employ Riot Control Agents (RCAs). Such agents ideally possess a sufficiently high safety ratio. RCAs may induce both acute and chronic effects, while their safety ratios may be highly variable (espcially when used on vulnerable subjects). In this study we analyze whether RCAs induce readily discernible long-term effects. We show that exposure to RCAs can result in subacute and chronic effects, including serious respiratory system, dermatological, and allergic conditions. This research reveals risk factors for developing long term effects of RCAs among younger subjects, or in cases of prolonged or repeated exposure (e.g. occupational exposure). New research should be conducted in order to better characterize all possible harmful effects of RCAs.

Key Words: Riot control agents, Harmful effect

ÖZ

Güvenlik güçleri gösteri kontrol ajanlarını sıklıkla kullanmaktadırlar. Gösteri kontrol ajanlarının yüksek güvenlik düzeyine sahip olması gerekir. Bu ajanların kronik etkileri olabilmektedir ve pratikte özellikle duyarlı kişilere karşı kullanıldığında güvenlik düzeyleri beklenenin altında kalabilmektedir. Bu çalışmada gösteri kontrol ajanlarının olası kronik etkileri incelenmeye çalışılmıştır. Sonuçlarımız, gösteri kontrol ajanlarına maruziyetin kronik ve ciddi solunum sistemi, dermatolojik ve alerjik etkileri olduğunu göstermiştir. Olgu serimizden elde edilen verilere göre, uzun dönemli etkilerin ortaya çıkmasındaki risk faktörleri şunlardır; genç yaş, uzun süren ya da tekrarlayan maruziyet (mesleki maruziyet gibi). Gösteri kontrol ajanlarının tüm olası etkilerini ortaya koyabilmek için yeni çalışmalara ihtiyaç duyulmaktadır.

Anahtar Sözcükler: Gösteri kontrol ajanları, Zararlı etki

INTRODUCTION

Civil authorities and armed forces have not infrequently had recourse to employ Riot Control Agents (RCAs). The principal advantage of using RCAs is to minimize physical intervention between law enforcement and the affected population group in order to minimize risks associated with kinetic means of control (1). RCAs ideally possess a high safety ratio. Moreover, their effects start immediately upon exposure and can be relieved once decontamination procedures have been implemented (2).

RCA exposure can produce persistant effects and RCA safety ratios can be lower than anticipated, especially in more vulnerable populations. Humans display a range of physiological symptoms when subjected to RCAs. Factors affecting physiological responses include age and pre-existing medical conditions (i.e. co-morbidities) such as pulmonary/cardiovascular disease. In this study we attempt to further characterize certain RCA exposure effects.

CN (Chloroacetophenone)

CN was first synthesized in 1871 and has typically been employed by civil and military authorities to disperse and/or incapacitate crowds (1).

CS (Chlorobenzylidene malononitrile)

CS was discovered in 1928 and is the most persistent RCA and was used as early as 1950's in the UK as a riot control agent (3). It has an immediately detectable pungent pepper-like odor (3).

CR (Dibenz (b, f) 1: 4 –oxazepine)

CR was first synthesized in 1962 and is a more recent addition to standard RCA formulations. CR is understood to be less toxic than CN and CS (3).

OC (Oleoresin capsicum)

OC was first used in the USA in 1973 where it was approved for law enforcement use. OC became widely adopted by law enforcement agencies globally beginning in the 1980's (4).

OC is obtained by extracting the ripe fruit of chili peppers and allowing it to dry. OC contains various naturally occurring acids and esters, alcohols, aldehydes, ketones and carotenoid pigments. Thus, OC stands in contrast to other RCAs such as CR, CN, DM and CS that are synthesized through more traditional chemical syntheses processes (1,3).

Capsaicinoids are the key component in OC. The toxicological and pharmacological effects of this active ingredient (8-methyl-N-vanillyl-6-nonenamide) are well characterized. Capsaicin activates protective physiological reflexes such as bronchoconstriction, sneezing coughing, apnea, and rapid shallow breathing due to airway irritation (1).

Clinical findings of RCAs

RCAs are efficacious peripheral sensory irritants that create irritating or noxious sensations due to their acute action on the sensory nervous system of the eyes, respiratory tract, and skin.

Ocular effects

One of the main characteristics of RCAs are their ocular effects (both reversible and irreversible). Reversible effects comprise of an intense and rapid stinging sensation in the eyes with lacrimation, blepharospasm, itching, discomfort, pain, swelling, conjunctivitis, corneal ulceration and blurred vision. Also, exposure to OC spray can inhibit the blinking reflex (1,3).

Irreversible effects of RCAs have been found to be corneal scarring, corneal opacification, corneal revascularization, cataract, and glaucoma (3,4,5). Studies performed on Emergency Room patients exposed to pepper spray showed that from 7 to 9% of patients suffered from corneal abrasions (6,7).

Respiratory system effects

Victims may experience burning sensations throughout the respiratory tract, constriction sensation in the chest, and discomfort or pain of the throat, nasopharynx, and nose. Laboratory tests also may reveal decreased breathing rate, increased secretions in the trachea and bronchi, decreased tidal volume, sneezing, bronchoconstriction, and respiratory arrest (1). According to animal studies, the principal cause of death following CN inhalation results from the injurious action of the agent on the pulmonary system (3).

OC spray is known to result in inflammatory reaction in the airways. Deleterious effects, including congestion, hemorrhage, and emphysema, may stem from exposure to high levels of RCAs (1). OC may cause corneal pathology (8). CS or CS/OC exposure may result in long-term effects such as Reactive-airway Dysfunction Syndrome (1, 9).

The primary site of stimulation for inhaled capsaicin is the larynx, likely because capsaicin-sensitive nerve

terminals are situated in the larynx (10). Substance P can mediate the effects of capsaicin exposure including bronchoconstriction, vasodilatation, and plasma protein extravasation. The introduction of substance P can stimulate c-fibers involved in pulmonary and bronchial circulation and result in bronchoconstriction (11). However, human experimental studies have revealed OC does not cause bronchoconstriction (12).

Dermatological effects

RCA exposure have also been shown to cause various well-recognized cutaneous reactions that are generally short-lived. These include burning/stinging sensations, erythema, blistering, rashes, pruritis, vesicles, and desquamation. However, chronic and unpredictable cutaneous reactions to RCAs, such as contact dermatitis and contact allergy, may also occur (3,13,14). Histological findings of the skin typically comprise acute inflamatory cell infiltration, spongiosis of the outer dermis, and necrosis of the epidermis and collagen within the outer dermis (1).

Immunotoxicology

RCAs, especially CN and CS, may suppress humoral immunity and phagocytic capability of pulmonary immunocompetent cells (1). RCAs may also cause hypersensitivity reactions (15).

Injuries caused by solvent/carriers

Since RCAs are usually solids at standard temperature and pressure (STP) they are typically dissolved in solvents and transformed into a micro-powder form (e.g., siliconized) or dispersed using a thermal generator device. The solvent or powder preparation methods imply that the RCA agent itself is not the only cause of chronic and acute toxic effects, such as isopropanol-induced ocular injury (i.e. corneal erosion), methyl isobutyl ketone related skin injury (i.e. blistering), and dichlomethane-induced neurotoxic effects (1).

Some injuries and deaths have been linked to delivery system equipment coming into contact with the human body. Cartridges are the most widely employed delivery system because they allow for greater distance and accuracy than hand-thrown projectiles (1). Fatalities have been caused by cartridges that have come into forceful contact with vital areas of the body, such as the neck and head (16). The most common deaths in such cases are head injuries, including trauma to the eyes (17,18,19).

Also of concern is the cyanogenic potential of CS resulting from the potential for cyanide formation in vivo following exposure to CS, and potential exposure to hydrogen cyanide (HCN)–a byproduct of the thermal decomposition of CS. Determining toxicologically significant amounts of in vivo cyanide formation remains challenging and unlikely (1). The percentage of evolved HCN has nevertheless been found to multiply with an increase in temperature. When CS is discharged from grenades/shells the burst temperature of these munitions is high (>300°C), which in turn may create a high amount of HCN (20).

Postmortem findings

The primary cause of death following CN inhalation in animal studies has been linked to the injurious action of CN on the pulmonary system. In other animal studies no cases died immediately during CS and CN exposure. However, the majority died within two days following exposure. The lungs of animals that died within the first 48 hours after CS or CN exposure were edematous, congested, and exhibited hemorrhages of different sizes and severity. The tracheas of the animals were also congested and presented an inordinate amount of mucous. Histopathological lung examination revealed inter and intraalveolar hemorrhaging, moderate to extreme congestion of the alveolar capillaries, and excessive intrapulmonary bronchi and bronchioles secretions (1).

MATERIALS and METHODS

PubMed/MEDLINE and Web of Science up to August 2020 were searched with the following terms “Riot control agent”, “Crowd control agent”, “pepper spray”, “pepper gas”, “CN”, ”CS”, “CR”, “Chloroacetophenone”, “Chlorobenzylidene malononitrile” and “Dibenz [b,f]1: 4 -oxazepine”.. The reference lists in selected articles and the abstracts published at major international conferences were manually searched.

Some research results were published prior to the present study (21).

CASES

Case 1

53-year-old white male subject was performing his occupation as a prison guard. During a “shakedown” procedure on a housing unit in the prison, he turned over a mattress when he was exposed to a high level of a

mixture of 1% CS and 1% OC in a solvent blend for at least 30 seconds.

The subject experienced immediate symptoms of mucous membrane irritation, cough, and chest tightness. According to pulmonary-function tests, he was diagnosed with reversible and fixed obstructive pulmonary disease. Even 3 years later, he works in a restricted capacity (no irritant exposure, no strenuous activity) and requires several prescription medications for treatment of his pulmonary symptoms (9).

Case 2

A 24-year-old patient was exposed to CS gas twice and acute irritant reaction was noticed on both occasions. There was no past medical history of skin disease or allergy. 6 days after the second exposure, a bullous exposure-pattern dermatitis was seen. On the basis of histological findings (a spongiotic dermatitis), a diagnosis of contact allergy to CS gas was made (22).

Case group 3 *(the occupational exposure)*

Watson el al. described seven patients who developed chronic dermatologic diseases as a cause of chronic CS exposure. Patients were consisted of 6 police officer and a doorman.

These diseases included;

Allergic contact dermatitis,

Rosacea,

Chemical burn from CS spray, resulting in leukoderma and dysaesthesia,

Seborrhoeic dermatitis initiated or aggravated by CS spray

Irritant contact dermatitis from CS spray, with possible rosacea (13).

Case 4

A 24 year old female patient was exposed to chloroacetophenone (CN) and showed burning sensation in her eyes as well as tearing. She suffered from chest pain and shortness of breath of 3 hour duration. According to the chest X-ray, she was diagnosed with inhalation injury lung edema (23).

Case 5

A 16-year-old female patient was exposed to the CS spray. She experienced tearing, conjunctivitis, blinking and coughing only few minutes after exposure. 3 weeks later of exposure, she hospitalized with acute respiratory distress and inspiratory stridor. Laryngoscopic examination revealed a laryngeal obstruction due to vocal cord edema and extensive crusting at the glottic level. Also extensive crusting in the laryngeal and tracheal lumens was observed. Fiberoptic bronchoscopy showed that the trachea and main bronchi were found to be occluded with large casts of denuded tissues and carbonaceous deposits (24).

Case group 6

CN was released in a prison cell block by aerosolization. 44 Prisoners claimed they sprayed multiple times. Immediate effects were syncope, emesis, dyspnea, tachycardia, tachipnea and hypertension. Late complications included conjunctival edema, fever, pharyngeal erythema, wheezes, productive cough, hoarseness, bullae and rash. Lastly, the three patients developed grey pdeudomembranous pharyngeal exudates 5 days post-exposure (25).

Case 7

A 39-year-old man was exposed to CS gas to his face and neck. He has no past medical history. He presented to Accident and Emergency Department with pain, blistering and redness over face, neck and upper chest, decreased vision in the effected eye and reduced hear in the effected ear. Some effected areas became painful superficial burns with erythema and blistering in 2 days time (26).

Case 8

A 30-year-old Hispanic man was sprayed heavily with CS. The next day he developed a dry cough, erythema of the exposed skin, swelling about the eyes, and loss of appetite. A generalized, pruritic skin rash evolved during subsequent days, and dyspnea and chest discomfort worsened. Eight days after exposure, he was referred to the Emergency Department and the initial diagnosis was hypersensitivity reaction to CS with bronchoconstriction and pneumonitis. Spirometry showed a restrictive defect with mild airflow obstruction. 50 days after exposure, he was admitted to the intensive care unit of another hospital for severe asthma. 2,5 months later, he showed dermatitis and wheezing. During the subsequent 6 months of outpatient follow-up, the patient continued to complain of cough, dyspnea, and occasional wheezing provoked by exertion and exposure to cold air. Patch tests results were positive reactions at 48 hr to all dilutions of CS and a lesser response to CN, to which the patient had no known exposure (15).

Case 9

A 4 week old healthy male infant was exposed to 5% OC when a self defense spray accidentally discharged in his face. He had the rapid onset of gasping respirations and epistaxis followed by apnea and cynosis. He was transferred to hospital in 20 minutes and intubated and supported by mechanical ventilation for respiratory failure. His initial chest radiograph showed bilateral diffuse parenchymal infiltrates. At 30 hours postinjury he had marked worsening of hypoxemia, the next 60 hours of his course were complicated by hypotension requiring dopamine, bilateral tension pneumotoraces, and copious thick tracheal secretions. By postinjury second day, he developed an infection and received antibiotherapy. He was placed on venoarterial Extracorporeal Membrane Oxygenation (ECMO) 96 hours after injury. The infant was discharged home 9 days after his ECMO course (27).

Case 10

A total of 38 US marines received a training course involved in CN exposure. After strenuous physical exercise from 36 to 84 hours after heavy exposure of CS in a field training setting, four of them presented with hypoxia. Their findigs were consistent with a mild acute lung injury with pulmonary edema. Also the author stressed that the relationship of position in formation and position of the CS canister to those Marines later requiring hospitalization is suggestive of a dose-response effect (28).

Case 11 and 12

A 21 year old male exposed to pepper spray in a training. According to witnesses, there was not any issues at the event and decontamination process. However, immediately after that, he collapsed and become unresponsive. This case was accepted as “cardiac arrest triggered due to pepper spray (29). A similar case was reported from Tunisia. After direct exposure to CS gas, A 24-year-old male with no particular history, presented with acute chest pain, dyspnea and vomiting. According to medical tests; there was a myocardial infarction with an intra-ventricular thrombus (30).

Case 13

A teenage male was exposed to pepper spray and showed numbness in the hands and feet. By the time weakness progressed from the legs towards the upper parts, and disruption in walking. Taking into account the clinical and radiologic findings “polyneuropathy mimicking Guillain-Barre syndrome related to pepper spray was diagnosed. Despite a wide variety of medical tests, no other cause for Guillain-Barre syndrome was detected. Intravenous immunoglobulin treatment made him cure in one week time (31).

DISCUSSION

In this study we consider whether RCAs have readily discernible persistant effects. Our study confirms that exposure to RCAs can result in serious subacute to chronic respiratory, dermatological, and allergic conditions on the basis of case studies.

Legal and regulatory aspects

Various analysts, officials, and other interested observers have considered the legal and political aspects of the development and employment of RCAs and other incapacitants as utilized by law enforcement and the military (32,33). There is a growing overlap between domestic law enforcement and international interventions for counter-terrorism and peacekeeping purposes. It is increasingly feasible to selectively influence human physiology at the molecular level resulting in further legal and political uncertainty. In addition, each legal and regulatory framework possesses a distinct (sometimes overlapping) set of actors, political dynamics, and perceptions of what activity is permitted.

In broad terms, international law that is applicable (potential or otherwise) to the development and employment of RCAs and incapacitants include the law of armed conflict (which is both treaty-based and dependent on so-called “customary international law”). Relevant international agreements include a series of conventions concluded in The Hague that began in the late 19th century concerning the conduct of war (e.g. the 1907 Hague Convention (IV) respecting Laws and Customs of War on Land and its Annex: Regulations concerning the Laws and Customs of War on Land). Two key international agreements are the 1972 Biological and Toxin Weapons Convention (BWC) and the 1993 Chemical Weapons Convention (CWC). The former has no standing verification or implementing body, while the latter is implemented by The Hague-based Organisation for the Prohibition of Chemical Weapons (OPCW). The BWC parties undertake never under any circumstances “to develop, produce, stockpile or otherwise acquire or retain: (1) Microbial or other biological agents, or toxins whatever their origins or method of production, of types in quantities that have no justification for prophylactic, protective or other peaceful purposes; (2) Weapons, equipment, or other means of delivery designed to use such agents or toxins for hostile

purposes or in armed conflict." (BWC, Article I) The CWC prohibits the use of toxic chemicals and their precursors, including RCAs, as "a method of warfare" (CWC, Article I, para. 6). Both conventions cover toxins. However, under the CWC toxic chemicals and their precursors may be employed for a variety of permitted purposes (e.g. "law enforcement including domestic riot control"). (CWC, Article II, para. 9). Such toxic chemicals and their precursors must be in "types and quantities" that are consistent with nonprohibited purposes (CWC, Article II, para. 1(a)). In addition, the CWC's phrasing on domestic riot control has been understood to be a sub-set of "law enforcement." This can be taken to imply that the use of RCAs and incapacitants are permissable for law enforcement during international peacekeeping and/or counter-terrorism operations.

A major concern within the BWC and CWC regimes is that the development and employment of RCAs and incapacitants inadvertently undermine the international prohibitions against chemical and biological warfare. Political and legal uncertainties at the nationally and globally exist, notwithstanding the fact that RCAs and incapacitants are (at least in theory) distinct in terms of their physiological effects when properly employed

More broadly, legal requirements may be interpreted "according to the letter" (a form of legal reductionism) or more according to "their spirit" (i.e. overall purpose). States and other actors may (actively or passively) seek politically preferred outcomes or attempt to avoid the perception of politically unwelcome outcomes. Thus, legal experts may argue multiple sides of an issue depending on political preferences, including to permit law enforcement and military personnel to acquire and use the latest technology and equipment. The public should be provided with technically correct information on the safety of RCAs that prioritizes the broader principles and scientific transparency and accountability. (34).

Our research reveals risk factors for developing long term effects of RCAs include young age, prolonged and repeated exposure, and especially occupational exposure. Long-term effects can be divided into three main categories: respiratory system effects, dermatological effects, and allergic reactions. Acute or chronic respiratory system effects range from acute respiratory distress to fixed obstructive pulmonary diseases. Dermatological effects range from seborrhoeic dermatitis to chemical burns. Allergic reactions included irritant/allergic contact dermatitis and positive patch tests against RCAs. Unfortunately, biomarkers for low-level and long-term exposures of capsaicinoids, the most commonly used RCAs, have not been identified to date. (35).

CONCLUSION

RCAs can cause subacute or chronic effects especially in vulnerable subjects. Further research should be conducted in this field in order to reveal all harmful effects of RCAs using inter alia the latest case exposure data. RCA use should be restricted or modified on this basis. Further efforts should be undertaken to identify best practices and their harmonization regarding RCA training and use. Relevant research findings should inform training procedures for the employment of RCAs by law enforcement and military personnel.

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None

Authors Contributions

All authors contributed equally in designing the study, reviewing the literature, being prepared for the ethics committee, collecting, analyzing and reporting the data.

Conflict of Interest

The authors declare that they have no conflict of interest.

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Ethical Approve

Since the research nor experimental neither contains personal data, ethical approval was not necessary.

Review

A Blind peer review process was implemented.

REFERENCES

1. Olajos EJ, Stopford W. Introduction and historical perspectives. In: Olajos EJ, Stopford W, editors. Riot Control Agents. Boca Raton: CRC Press; 2004:1.
2. Sidell FR. Riot control agents. In Zajtchuk R, editor. Medical aspects of chemical and biological warfare. Washington. TMM Publications; 1997.307-322.
3. Olajos EJ, Salem H. Riot control agents: Pharmacology, toxicology, biochemistry and chemistry. J Appl Toxicol 2001;21:355-391.
4. Broadstock M. What is the safety of "pepper spray" use by law enforcement or mental health service staff?, NZHTA Tech Brief Series; 2002.
5. Smith J, Greaves L. The use of chemical incapacitant sprays: A review. J Trauma 2002;52:595-600.

6. Watson WA, Stremel KR, Westdorp EJ. Oleoresin capsicum (cap-stun) toxicity from aerosol exposure. Ann Pharmacother 1996;30:733-735.
7. Brown L, Takeuchi D, Challoner K. Corneal abrasions associated with pepper spray exposure. Am J Emerg Med 2000;18:271-272.
8. Yeung MF, Tang WY. Clinicopathological effects of pepper (oleoresin capsicum) spray. Hong Kong Med J 2015;21(6):542-552.
9. Roth VS, Franzblau A. RADS after exposure to a riot-contro agent: A case report. J Occup Environ Med 1996;38:863-865.
10. Collier JG, Fuller RW. Capsaicin inhalation in man and the effects of sodium cromoglycate. Br J Pharmac 1984;81:113-117.
11. Hilmas CJ, Poole M, Katos AM, Williams PT. Riot control agents. In: Gupta RC, editor. Handbook of toxicology of chemical warfare agents. London: Academic Pres, 2009;153-175.
12. Chan TC, Vilke GM, Clausen J, Clark RF, Schmidt P, Snowden T, Neuman T. The effect of oleoresin capsicum "pepper" spray inhalation on respiratory function. J Forensic Sci 2002;47(2):299-304.
13. Watson K, Raycroft R. Unintended cutaneous reactions to CS spray. Contact Dermatitis 2005;53:9-13.
14. Carron PN, Yersin B. Management of the effects of exposure to tear gas. BMJ 2009;19(338):1554-1558.
15. Hill AR, Silverberg NB, Mayorga D, Baldwin HE. Medical hazards of the tear gas CS: Case of persistent, multisystem, hypersensitivity reaction and review of the literature. Medicine 2000;79:234-240.
16. Rothschild MA, Vendura K. Fatal neck injuries caused by blank cartridges. Forensic Sci Int 1999;101:151-159.
17. Clarot F, Vaz E, Papin F, Clin B, Vicomte C, Proust B. Lethal head injury due to tear-gas cartridge gunshots. Forensic Sci Int 2003;137:45-51.
18. Jacob B, Huckenbeck W, Daldrup T, Haarhoff K. Suicides by starter's pistol and air guns. Am J Forensic Med Pathol 1990;11:285-290.
19. Giese A, Koops E, Lohmann F, Westphal M, Püschel K. Head injury by gunshots from blank cartridges. Surg Neur 2002;5:268-277.
20. Gutch PK, Raza SK, Malhotra RC. Studies on thermal degradation of benzylidene malononitrile. Journal of Thermal Analysis and Calorimetry 2003;71:593-599.
21. Toprak S. Long term effects of riot control agents. XI. Forensic Sciences Congress, 23-26 April 2014 Cyprus
22. Sommer S, Wilkinson SM. Exposure-pattern dermatitis due to CS gas. Contact Dermatitis 1999;40:46-47.
23. Vaca FE, Myers JH, Langdorf M. Delayed pulmonary edema and bronchospasm after accidental lacrimator exposure. Am J Emerg Med 1996;14(4):402-425.
24. Karaman E, Erturan S, Duman C, Yaman M, Duman GU. Acute laryngeal and bronchial obstruction after CS (o-chlorobenzylidenemalononitrile) gas inhalation. Eur Arch Otorhinolaryngol 2009;266:301-304.
25. Thorburn KM. Injuries after use of the lacrimatory agent chloroacetophenone in a confined space. Archives of Environmental Health 1982;37(3):182-186.
26. Agrawal Y, Thornton D, Phipps A. CS gas-Completely safe? A burn case report and literature review. Burns 2009;35:895-897.
27. Billmire DF, Vinocur C, Ginda M, Robinson NB, Panitch H, Friss H, Rubenstein D, Wiley JF. Pepper-spray-induced respiratory failure treated with extracorporeal membrane oxygenation. Pediatrics 1996;98(5):961-963.
28. Thomas RJ, Smith PA, Rascona DA, Louthan JD, Gumpert B. Acute pulmonary effects from o-chlorobenzylidenemalonitrile "tear gas": A unique exposure outcome unmasked by strenuous exercise after a military training event. Mil Med 2002;167(2):136-139.
29. Arora N. Cardiac arrest in a young patient triggered due to pepper spray: A case report. Austin Crit Care J 2019: 6(1):1026
30. Zakhama L, Ameur WB, Antit S, Slama I, Jallad AE, et al. Can CS gas induce myocardial infarction? Tunis Med 2016;94(10):626-628.
31. Kaya Özçora GD, Çıraklı S, Canpolat M, Doğanay S, Kumandaş S. Pepper spray inhalation-induced acute polyneuropathy mimicking Guillain-Barre syndrome. Turk Pediatri Ars 2019;54(1):53-56.
32. Davison N. "Off the Rocker" and "On the Floor": the Continued Development of Biochemical Incapacitating Weapons: Bradford University; 2007 Aug. Bradford Science and Technology Report no: 8.
33. International Committee of the Red Cross (ICRC). Incapacitating Chemical Agents: Implications for International Law. Geneva: ICRC, 2010.
34. Busker RW. Safety evaluation of Pepper Spray as a police weapon. In 1st European Symposium on Non-Lethal Weapons, 2001, Germany. Accessed Jan 12, 2020, http://www.non-lethal-weapons.com/sy01abstracts/v8.pdf.
35. Pesonen M, Vähäkangas K, Halme M, Vanninen P, Seulanto H, Hemmilä M, Pasanen M, Kuitunen T. Capsaicinoids, chloropicrin and sulfur mustard: Possibilities for exposure biomarkers. Front Pharmacol 2010;1:140.