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Case Report / Olgu Sunumu

Pepper spray inhalation-induced acute polyneuropathy mimicking Guillain-Barre syndrome

Biber gazı maruziyeti sonucu gelişen Guillain-Barre sendromunu taklit eden polinöropati

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Abstract

Peripheral neuropathy is the most common reaction to toxic chemical substances in the nervous system. Toxic neuropathies are often misdiagnosed because there are no easily available specific or biologic tests for the diagnosis. Guillain-Barre syndrome is the most common cause of acute flaccid paralysis in children and adolescents. Clinical signs of the disease are often at the beginning of the distal symmetric weakness and areflexia progresses rapidly. Although capsaicin is widely used in the treatment of so many diseases, especially of neuropathic pain, cancer and osteoarthritis, it is known to be toxic in many systems such as the eye, skin, respiratory, and circulatory systems. Although there is inadequate information about its long-term effects, it has also been reported that in large quantities there is increased risk of toxicity and prolonged exposure can lead to death. In our case, we present acute polyneuropathy mimicking Guillain-Barre syndrome after exposure to pepper spray because it is noteworthy and interesting.

Keywords: Acute flask paralysis, Guillain-Barre syndrome, pepper spray

Öz

Periferik nöropati sinir sisteminin toksik kimyasal maddelere karşı verdiği en sık görülen reaksiyonudur. Tanı için özgün ya da biyolojik testlerin kolaylıkla bulunmaması ve maruziyetin bilinmemesi nedeni ile toksik nöropatiler sıklıkla yanlış tanı alırlar. Guillain-Barre sendromu çocuk ve ergenlerde akut flask paralizinin en sık nedenidir; klinik bulguları hastalığın başlangıcında distalde olup sıklıkla hızlı ilerleme gösteren simetrik güçsüzlük ve arefleksidir. Ağrı, kanser, osteoartrit vb. birçok hastalık tedavisinde kullanım alanı bulan kapsaisinin başta göz, deri, solunum ve dolaşım sistemi olmak üzere birçok sistemde toksik etki gösterdiği, hatta ölüme götüren hastalık süreçlerini tetiklediği bilinmektedir. Uzun dönemdeki etkileri ile ilgili yeterli bilgi bulunmamakla birlikte, yüksek miktarlarda ve uzamış maruziyet durumunda toksik risklerin arttığı ve ölüme yol açabileceği de bildirilmektedir. Olgumuzda biber gazı maruziyeti sonrası Guillain-Barre sendromunu taklit eden polinöropati olgusu ilgi çekici olması nedeni ile sunulmuştur.

Anahtar sözcükler: Akut flask paralizi, biber gazı, Guillain-Barre sendromu

Introduction

Peripheral neuropathy is the most common reaction of the nervous system against toxic chemical substances. Although the cause that leads to injury is not clearly known in toxic neuropathies, industrial, environmental and biologic agents, heavy metals, and pharmacologic agents may lead to this picture (1, 2). Neuronal injury may be in the form of distal axonal degeneration (axonopathy), degeneration of the neuronal body (neuronopathy) or primary demyelination (myelinopathy). Guillain-Barre syndrome (GBS) is a polyneuropathy and is the most common cause of acute flaccid paralysis in children and adolescents. The clinical sings are observed in the distal part at the beginning of the disease and include rapidly progressing symmetrical weakness and areflexia. This picture frequently emerges a few days or weeks after nonspecific infection. It is thought to be an autoimmune disease that results in the production of antibodies against antigenic proteins in peripheral nerves as a result of T cell activation. Although

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Table 1. Effects of capsaicin

	Clinical status	Potential complication	Potential sequela
Eye	Lacrimation, sense of burning, pain, blepharospasm, photophobia, corneal edema (OC)	Keratitis (CN), corneal erosion, ulceration (OC), intraocular hemorrhage	Cataract, glaucoma
Respiratory tract	Severe rhinorrhea (CS), sneeze, cough, dyspnea (CS), pharyngitis, tracheitis, bronchitis	Bronchospasm, hypoxia (CN), pulmonary edema (CS), asthma attack	Reactive airway dysfunction syndrome, asthma
Cardio-vascular system	Hypertension (CS)	Heart failure, cerebral hemorrhage	Undefined
Skin	Hyperemia, erythema, edema, sense of burning, bulla (CS)	Irritant contact dermatitis (CN), facial edema (CN), exacerbation of dermatitis	Allergic dermatitis (CN)
Gastrointestinal System	Irritation in the lips, increased saliva (CS), painful swallowing, dysphagia, abdominal pain, diarrhea, nausea, vomiting (DM)	Hepatic toxicity (CS)	Undefined
Central Nervous System	Tremor (DM), agitation, anxiety	Hysterical reaction, panic attack	Undefined
Genotomic effect			Clastogenic, mutagenic, carcinogenic

CS: Chlorobenzylidenemalononitrile; CN: Chloroacetophenone; DM: Chlorodihydrophenarsazine; OC: Oleoresincapsicum

the antibodies target myelin proteins, axonal structures are the primary target in immune-mediated injury in some cases. Various infectious, immunologic, and genetic causes are blamed in the etiology (3).

Acute neuropathies are misdiagnosed as GBS because electrophysiologic and clinical findings support GBS and specific diagnostic tests or biologic tests are not easily accessible (1, 2). We present a case of acute polyneuropathy mimicking GBS following exposure to pepper spray because it is noteworthy.

Case

A sixteen-year-old male patient presented to our clinic on the seventh day of exposure to pepper spray because of numbness in the hands and feet on the first day following inhalation of and exposure to pepper spray, weakness and progression of weakness from the legs towards the upper parts, and disruption in walking. There was no pathology in his personal and familial history. His systemic examination was found to be normal. On neurologic examination, cranial nerve examination was found normal, upper extremity muscle strength was found normal, abdominal skin reflex and lower extremity deep tendon reflexes were absent, muscle strength in the distal part of both lower extremities was found as 4/5, and sensorial defect on a level and sphincter dysfunction were not found. Lumbar puncture performed with a prediagnosis of GBS revealed the following cerebrospinal fluid findings: protein: 90 mg/dL (15–45 mg/dL), glucose: 69 mg/dL, chlorine: 122 mmol/L, white blood cells (WBC) 2 cells/uL. Electromyography (EMG) revealed demyelination in the peroneal nerves and axonal and demyelinating neuropathy in the tibial nerves. Contrast-enhanced magnetic resonance imaging (MRI) of the medulla spinalis revealed mild contrast uptake following intravenous injection of contrast agent in the cauda equina fibers. A diagnosis of GBS was made after assessment of the clinical and radiologic findings. Viral [herpes simplex virus (HSV), cytomegalovirus (CMV), Epstein-Barr virus (EBV), hepatitis A virus (HAV), hepatitis B virus, hepatitis C virus (HCV), human immunodeficiency virus (HIV)], bacterial (Borrelia burgdorferi, Brucella melitensis, Campylobacter jejuni) and autoimmune serology [anti-nuclear antibodies (ANA), and anti-double-stranded DNA (Anti dsDNA)] tests directed to the etiology were found to be negative. Urine and stool cultures showed no growth. There was no history of infection, surgical operations, and use of substance, and medication in the last four weeks. The only factor that might have created an extraordinary tendency was inhalation of pepper spray and his symptoms had begun following exposure. A diagnosis of polyneuropathy mimicking GBS related to pepper spray was made after assessment of the

history and clinical and laboratory findings. The level could not be evaluated because there was no clinical test that could show the amount of exposure to pepper spray. Intravenous immunoglobulin treatment was initiated at a dose of 0.4 mg/kg and continued for five days. Regression in hand-food numbness occurred on the second day of treatment and improvement in walking was observed on the seventh day. After discharge, a neurologic examination was found to be normal in the follow-up visit at one month and EMG was found to be normal on the follow-up visit at two months. Written informed consent was obtained from the patient and his parents.

Discussion

Pepper spray use spread quickly worldwide throughout the 1990s and it has been used in different forms (spray, bomb-like lachrymose, irritant, aerosol and fluid with blocker property). The three widely used forms in America and Europe include chlorobenzylidenemalononitrile (CS), chloroacetophenone (CN) and oleoresincapsicum (OC) (4, 5). The oral lethal dose of capsaicin, which is the active ingredient of pepper spray, is 0.5–5 g/kg for humans. Capsaicin is synthesized with the addition of a fatty acid chain to vanillylamide in the interlocular septae of hot pepper. It is produced mostly form chili or hot cayenne pepper because these contain a high amount of capsaicin. Its characteristic effects include excitation, desensitization, and neurotoxicity. It is known that it causes neurogenic inflammation by leading to a release of neuropeptides including substance P and neurokinin A by inducing sensory neurons (6, 7).

It is known that capsaicin, which has an area of use in the treatment of many diseases including neuropathic pain, cancer, and osteoarthritis, leads to toxic effects in many systems including the eye, skin, respiratory system, and cardiovascular system, and even triggers disease processes that lead to mortality. Its potential effects are shown in Table 1. Although there is insufficient information related to its long-term effects, it has been reported that toxic risks are increased and even mortality may occur with high amounts and prolonged exposure (4).

In the literature, muscle weakness, tingling and numbness in the hands and feet, imbalance in walking, and epilepsy attacks have been reported following exposure to pepper spray in community-based assessments. Numbness in the hands and feet and weakness in the legs began in the first 24 hours after exposure in our patient. It has also been reported that these effects vary depending on the dose, age, underlying chronic disease, and presence of hypersensitivity (4). Again, cases of acute myocardial infarction, respiratory arrest, and chronic lung disease developing in relation to pepper spray have been reported; it was observed that life-threatening conditions increased in cases of chronic disease and at advanced ages (7–9). It has been reported that capsaicin is neurotoxic because it has polymodal distribution in the nerve endings and inhibits neuronal transmission by opening non-selective ion channels. We present this case of acute polyneuropathy mimicking GBS following exposure to pepper spray related to the primary neurotoxic effect of capsaicin because it is noteworthy (10). The awareness of all citizens including mainly children on the issue of the potential harmful effects of pepper spray and treatment after exposure should be raised.

Informed Consent: Written informed consent was obtained from the patients' parents and himself.

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References

1. Jalal MJ, Fernandez SJ, Menon MK. Acute toxic neuropathy mimicking guillain barre syndrome. J Family Med Prim Care 2015; 4: 137–8.
2. Kim S, Takeuchi A, Kawasumi Y, et al. A Guillain-Barré syndrome-like neuropathy associated with arsenic exposure. J Occup Health 2012; 54: 344–7.
3. Ozdemir O, Okan MS. Guillain Barre Sendromu-Derleme. J Curr Pediatr 2005; 4: 131–5.

4. Canıvar C. Solunum Sistemine Etkili Kimyasal Savaş Ajanları. Biber gazı yasaklansın tıbbi sempozyumu. İstanbul: İstanbul Tabip Odası; 2015. p. 25–33.
5. Orhan H, Erkekoğlu P, Kadıoğlu E. Göz Yaşartıcı Gazlar ve Toksik Etkileri. Türk Toksikoloji Derneği Yayın Organı 2013; 36: 9–22.
6. Dursun AZ, Sari S, Özkök A, ve ark. Gazi Üniversitesi Tıp Fakültesi Gazi Hastanesi Çocuk Acil Servisi'ne başvuran biber gazına maruz kalmış olguların değerlendirilmesi. GMJ 2015; 26: 110–1.
7. Cil H, Atilgan ZA, Islamoğlu Y, et al. Is the pepper spray a triggering factor in myocardial infarction? A case report. Eur Rev Med Pharmacol Sci 2012 ;16 Suppl 1: 73–4.
8. Şener E, Şahin S. Kapsaisin: Farmakokinetik, toksikolojik ve farmakolojik özellikleri. J Pharm Fac Hacettepe Univ 2010; 29: 149–63.
9. Safçi SB, Arbak PM, Balbay EG. Göz Yaşartıcı gaz inhalasyonu sonrası gelişen pulmoner restriksiyon olgusu. Respir Case Rep 2015; 4: 105–7.
10. Rasier R, Kukner AS, Sengul EA, et al. The decrease in aqueous tear production associated with pepper spray. Curr Eye Res 2015; 40: 429–33.