

SCORPION STINGS AND MANAGEMENT WITH REFERENCE TO EGYPT

By

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Abstract

Scorpions are predatory arachnids of order Scorpiones (Phylum: Arthropoda, Kingdom: Animalia). They have eight legs, and are easily recognized by a pair of grasping pincers and a narrow, segmented tail, often carried in a characteristic forward curve over the back and always ending with a stinger. The sting may be painful or even deadly, depending on the species. Of 1,500 species of scorpions worldwide, only about 20 to 25 are dangerous. Its' venom is a mixture of compounds, including neurotoxins that affect the victim's nervous system.

Key words: Scorpions, Sting envenomation, Manifestations, Treatment, Prevention, Nursing.

Introduction

Scorpion envenomation is a significant problem in the southwestern United States and throughout Mexico. In the United States, the *Centruroides exilicauda* (sculpturatus) stings are associated with major neurologic toxicity, especially in young children (Klauber, 1997). In Mexico, multiple toxic species exist, and annual mortality due to scorpion envenomation is ten times higher than that due to snakebite (Morsy *et al*, 2021). An estimated 5000 deaths occur annually from scorpion stings worldwide (LoVecchio and McBride, 2003).

Supportive care is the management component key. Anti-venom therapy with equine derived Fab fragments reduced the symptoms duration, which *Centruroides* antivenom is available in Mexico, but in USA, its use was restricted to that of an approved investigational drug (Boyer *et al*, 2009).

Taxonomy: Scorpions, which are grouped in the Phylum Arthropoda, have a lobster-like body with seven sets of paired appendages: the chelicerae, pedipalps (claws), four sets of legs, and pectines (a pair of comb-like structures on ventral surface). The segmented tail curves upward dorsally, ending in a terminal bulbous segment called the telson contains paired venom glands and stinger. In the USA, a subaculear tooth on a small, slender scorpion is specific to *Centruroides exilicauda* (sculpturatus), also known as the

bark scorpion (Curry *et al*, 1984).

Envenomation occurs via stinging and not biting. Scorpions clutch prey in their pedipalps (claws) and thrust the tail overhead to sting. Envenomations are sometimes wrongly reported as bites, but true scorpion bites have not been documented and would be inconsequential if they did occur. Scorpions can sting multiple times, although the first one depletes or nearly depletes the telson of venom (Russell, 1991).

A characteristic physical property of scorpions is that they fluoresce when illuminated by ultraviolet light, as from a black light or a medical Wood's lamp. This property is used in collecting scorpions for breeding or venom harvesting and in providing pest control. The fluorescent pigment in scorpion cuticle is most likely riboflavin (Krifi *et al*, 1992).

Overview and Discussion

Scorpion geography and appearance: Scorpions are found on all continents worldwide except Antarctica (Dehghani *et al*, 2018). They live in habitats ranging from tropical rainforests to grasslands and deserts with life span 5 to 25 years, depending on species. As adults, most scorpions are nocturnal and solitary, usually staying in the same territory throughout their lives. Many scorpions live in burrows they dig or claim and defend from other wildlife. They use the burrows and other types of shelters to hide from predators and to stay cool during the hot days

and warm during cold nights. The burrows are typically small and snug. Scorpions that don't burrow may climb trees or hide under bark or leaf litter for shelter (Sharma, 2018).

Estimation of scorpion species number varied; as 1400 species divided into nine families. Buthidae is the largest and the most dangerous family but, with few exceptions, contains the only species capable of producing clinically significant envenomations, via their neurotoxic venoms. At least 30 species can inflict potentially fatal stings (Gambhir *et al.*, 1998). All the genera commonly recognized as dangerous were buthid scorpions in the United States and Mexico this includes *Centruroides* and *Tityus* (Hutt and Houghton, 1998). Zourgui *et al.* (2008) in Libya recognized 9 different scorpions' species out of 5000 samples. These are *Leiurus quinquestriatus*, *Androctonus bicolor*, *A. australis*, *A. amoreuxi*, *Buthacus leptochelys*, *Buthus occitanus*, *Buthacus arenicola*, *Orthochirus innesi*, and *Scorpio maurus*. They added that *L. quinquestriatus* was restricted to the Southern areas, *B. occitanus* was in coastal areas, and *Androctonus* species were widely spread in all regions.

Ebrahimi *et al.* (2017) in Iran reported that more than 1.2 million scorpion stings occur annually worldwide, particularly in tropical regions, with two effective climate factors associated positively and negatively with scorpion sting cases are temperature and relative humidity, respectively. Abd El-Aziz *et al.* (2019) in Luxor Egypt reported that *Leiurus quinquestriatus* and *Androctonus crassicauda* were the commonest endemic scorpions, which sting caused acute health threatening. Amr *et al.* (2021) in Jordan reported in the 12 Arab Countries of the Middle East are inhabited by 117 species of scorpions of various medical importance within six families, with common occurrence scorpion stings throughout the region. Twenty-two species are considered to be dangerously venomous, causing potentially life threatening stings. Accessible literature in English and Arabic on scorpions, scorpion stings and available

anti-venoms was reviewed to document the scorpion fauna and scorpion stings in each country. Saudi Arabia, Iraq and Jordan reported the highest numbers of stings and envenomings. Alhamoud *et al.* (2021) in Saudi Arabia identified 28 species of scorpions, and approximately 14,500 scorpion stings are annually reported, with full recovery and low morbidity and mortality rates.

Centruroides exilicauda: *C. exilicauda* (or *C. sculpturatus* or the bark scorpion, due to of its preference for residing in or near trees) measures 4 to 7cm long in length and varies in color from yellow to brown or tan. The presence of a subaculear tooth, a tubercle at the stinger base is specific to *C. exilicauda* and is helpful in differentiating this highly neurotoxic scorpion from other species (Lo Vecchio *et al.*, 1999). *C. exilicauda* is found primarily in northern Mexico and southwestern United States (e.g., Arizona, New Mexico, western Texas, southeastern California, and near Lake Mead, Nevada).

Centruroides vittatus: *C. vittatus*, the striped scorpion, has a black intraocular triangle and black stripes on the thorax. *C. vittatus* is found primarily in the Southwest and Texas, but also extends into southern Indiana and Illinois (Stipetic *et al.*, 1998).

Centruroides suffusus: *C. suffusus* also known as the Mexican scorpion, is yellow to tan with dark longitudinal strips on the abdomen and measures 5 to 7.5cm. *C. suffusus* is found throughout Mexico and is considered the most dangerous among the many species there (Isbister and Bawaskar, 2014).

Venom: Scorpion venoms are complex mixtures containing mucopolysaccharides, hyaluronidase, phospholipase, acetylcholinesterase, serotonin, histamine, protease inhibitors, histamine releasers, and neurotoxins. Neurotoxins are the most important venom constituents in human envenomations (Sofer, 1995). In neuronal membranes, these toxins cause incomplete inactivation of sodium channels during depolarization that result in slow inward sodium current. This action caused membrane hyperexcitability and

led to repetitive uncontrolled firing of axons (Wainger *et al*, 2014). As a result, release of neurotransmitters at synapses and the neuromuscular junction is enhanced and leads to excessive neuromuscular activity and autonomic dysfunction (Vatanpour *et al*, 1993). Some scorpion neurotoxins also have effects on calcium-activated potassium channels (Garcia *et al*, 1998), chloride channels, and L-type calcium channels. The *C. exilicauda* venom does not cause local tissue destruction (Arie-Saadia *et al*, 1996).

USA epidemiology: *Centruroides exilicauda* (sculpturatus) is the culprit in most reported envenomations. Its sting causes a significant number of systemic reactions, and is known to be potentially fatal and *C. vittatus*, the commonest striped scorpion, is the second-most commonly reported species in envenomations (Pickett, 2003).

From 1931 to 1940, more than 40 deaths were due to *C. exilicauda* envenomation, mainly in young children and infants. No deaths were reported from scorpion envenomation in Arizona since 1968, a scorpion-related fatality was reported, although death was ascribed to anaphylaxis rather than to direct venom toxicity. Mortality was avoided with appropriate supportive care; prior fatalities were caused by respiratory failure that was complicated by metabolic acidosis, hyperthermia, and rhabdomyolysis from excessive muscular activity (Boyer *et al*, 2001).

Mexico epidemiology: At least 134 native species are regarded as dangerous. Among the eight members of genus *Centruroides*, *C. exilicauda* (sculpturatus), *C. noxius*, and *C. suffusus* (Mexican scorpion) are usually cited as the most dangerous (Francke, 2013). Most fatalities occur in the summer months from April through July. In the 1980s, hundreds of scorpion fatalities occurred nationwide per year, with respiratory failure as the proximate cause of death; this figure probably underestimates the true total by a factor of two to three, but in Mexico, scorpion deaths outnumber snakebite deaths ten to one (Dehesa-Dávila and Possani, 1994).

Clinical pictures: Human scorpion stings are usually accidental as scorpions would rather escape humans than attack. Stings most commonly occur when a human unintentionally steps on a scorpion or reaches under wood or rocks (Bouaziz *et al*, 2008). The majority of scorpion envenomations in the United States and Mexico cause local or no pain with or without inflammation. But, *C. exilicauda* (sculpturatus) and *C. suffusus* often cause similar neuromuscular effects that can be life-threatening. *C. vittatus* stings are less likely to cause systemic effects.

Centruroides exilicauda (sculpturatus): Post *C. exilicauda* envenomation four clinical grades are categorized, and after envenomation by *C. suffusus* follow a similar pattern: 1- Grade I envenomations produce local pain and paresthesias at the sting site. Usually, no local inflammation occurs, and the puncture wound is too small to be observed. If no scorpion is seen, diagnosis may require historical or epidemiologic clues or other physical signs. Tap test has been empirically recommended to confirm a *C. exilicauda* (sculpturatus) sting, although its reliability has not been rigorously tested. With the patient looking away or otherwise distracted, gently tapping the sting site would greatly exacerbate the pain, a sign that didn't occur with other scorpion envenomations (Wexler, 2014). 2- Grade II envenomations produce local symptoms as well as remote pain and paresthesias. The more distant symptoms often radiate proximally up the affected extremity but may occur in even more remote sites (e.g., contralateral limbs) or as generalized paresthesias. Children may present with unexplained agitation or inconsolable crying. 3- Grade III envenomations produce either cranial nerve or somatic skeletal neuromuscular dysfunction. Nijssen *et al*. (2017) in Sweden reported that in fatal disease-amyotrophic lateral sclerosis (ALS)-upper (corticospinal) motor neurons (MNs) and lower somatic MNs, which innervate voluntary muscles, degenerate. Importantly, certain lower MN subgroups were relatively

resistant to degeneration, even though pathogenic proteins are typically ubiquitously expressed. The ocular MNs (OMNs), including the oculomotor, trochlear and abducens nuclei (CNIII, IV, & VI) that regulate eye movement, persist via disease. Consequently, eye-tracking devices are used to enable paralyzed ALS patients (unable to speak) to communicate. Besides, there is a gradient of vulnerability among spinal MNs.

Cranial nerve dysfunction can manifest as blurred vision, abnormal eye movements, slurred speech, tongue fasciculations, and hypersalivation. The combination of bulbar neuromuscular dysfunction and increased oral secretions may cause problems with airway maintenance. Abnormal eye movements most often are involuntary, conjugate, slow, and roving. Chaotic multidirectional conjugate saccades like opsoclonus and unsustained primary positional nystagmus was seen (Clark *et al.*, 1991). Many patients with abnormal eye movements prefer keeping their eyes closed.

Somatic skeletal neuromuscular dysfunction typically appears as restlessness, fasciculations, shaking and jerking of the extremities, alternating opisthotonos (arching of the back), and emprosthotonos (tetanic forward body flexion). These signs can be mistaken for a seizure. However, the abnormal skeletal muscle activity appears more undulating and writhing than the tonic-clonic movements of generalized seizures. Also, unlike patients with seizures, victims of scorpion stings often remain awake and alert.

4- Grade IV envenomation: The envenomations manifested both cranial nerve dysfunction, and somatic skeletal neuromuscular dysfunction. By close examination, victims with skeletal muscle hyperactivity usually also have cranial nerve dysfunction, meet criteria for Grade IV. In the most severe cases, stridor and wheezing can occur.

Hmimou *et al.* (2008) in Morocco identified Abrog's classification of scorpion stinging's signs and symptoms grading as Grade I: Pain and/or paresthesia at sting site,

gling, numbness, and minor swelling in skin area encompassing sting (local symptoms), Grade II: Fever, chills, tremor, excessive sweating, nausea, vomiting, diarrhea, hypertension and priapism (systemic \pm local ones), & Grade III: Cardiovascular, respiratory, and/or neurologic distresses complications.

Hyperthermia up to 40°C (104°F) may occur due to excess motor activity. Respiratory failure, pulmonary edema, metabolic acidosis, sterile cerebrospinal fluid pleocytosis, rhabdomyolysis, coagulopathy, pancreatitis, and multiple organ failure have all to describe, especially in children with severe envenomation (Berg and Tarantino, 1991). Pancreatitis, commonly associated with *Tityus* scorpion envenomation, rarely occurs with Grade IV *Centruroides* envenomation and is typically transient (Kallel *et al.*, 2016).

Scorpion venoms generally do not produce coagulopathy or other significant hematologic effects, although disseminated intravascular coagulation was reported in Saudi Arabia (Annobil, 1993). Abdel-Rahman *et al.* (2010) in Egypt reported that (i) the venom of the *Scorpio maurus palmatus* scorpion gave significant neurotoxic and cytotoxic effects on insect cells, (ii) its efficacy, as assessed by the PED(50) unit, exhibited variation across its geographic range, and (iii) components in venom may have the potential for being developed into effective and environmentally friendly bioinsecticides.

Symptoms onset and duration: After *C. exilicauda* (sculpturatus) envenomation, symptoms may begin immediately and typically progress to maximum severity within five hours. Infants can reach Grade IV in as quickly as 15 to 30 minutes after being stung (Amaral and Rezende, 1997). Symptoms abate at a rate, which varied with victim's age and grade of envenomation. Symptomatic improvement typically occurs within 9 to 30hrs without antivenom therapy in patients with Grade III or IV envenomation, and pain and paresthesias are exceptions and persisted for up to two weeks (Corneille *et al.*, 2016).

Toxicity in children: Although adults appear to be envenomed more often, children are more likely to develop severe illness requiring intensive supportive care (Gateau *et al.*, 1994). In *C. exilicauda* (*sculpturatus*) envenomations, over two thirds of stings were in adults (Chippaux and Goyffon, 2008). But, 26% of children less than six years of age had severe envenomations (Grade III or IV) versus 6% of adults over 20 years.

Centruroides vittatus envenomation: *C. vittatus* stings commonly produce local symptoms of pain, bleeding, burning sensations, erythema, edema, hives, local paresthesias, and pruritus.

Systemic reactions occur in approximately 20% of victims, but are typically less severe than those caused by *C. exilicauda* (*sculpturatus*) and *C. suffusus*. Commonest systemic features include paresthesias (face, tongue, & perioral region), dysgeusia (unpleasant or altered taste), chills, sweating, dysphagia, fasciculations, nausea, and vomiting (Coward, 2011).

Laboratory evaluation: Laboratory studies are not needed in patients with mild (Grade I to II) envenomation. The following must be obtained from severe envenomed patients (Grade III to IV): 1- Serum electrolytes, 2- Liver enzymes (AST & ALT), 3- Blood urea nitrogen and serum creatinine, 4- Serum lipase, 5- Serum creatine kinase, & 6- Urinalysis.

Additional testing may be needed depending upon the clinical scenario. As an example, it is reasonable to obtain a complete blood count and coagulation studies in a young child with an unwitnessed bite. For those patients showing signs of exacerbation of underlying ischemic heart disease, obtain an electrocardiogram, cardiac biomarkers, and chest radiograph. Agrawal *et al.* (2015) in India reported a rare case of scorpion sting presented as myocardial infarction and heart failure, successfully treated with Intensive Care Unit care, noninvasive ventilation, vasopressors, and anti-ischemic treatment.

Diagnosis: The diagnosis of *Centruroides* scorpion sting is based upon clinical findings including recent visit to or residing in an endemic region for the scorpion, history of a scorpion sting (often not present) and characteristic signs of envenomation including local pain exacerbated by tapping near the sting site, hypersalivation, abnormal eye movements (i.e., chaotic multidirectional conjugate saccades resembling opsoclonus and unsustained primary positional nystagmus), alternating opisthotonus, fasciculations, and clonus. Mental status is typically preserved. Support laboratory findings include evidence of rhabdomyolysis and pancreatitis in patients with moderate to severe envenomation (Grade III or IV).

Differential diagnosis: Young children in endemic areas showed unusual neurologic symptoms (as agitation, choreiform movement, or abnormal eye movements) may be assumed to have been envenomed without history of a sting or scorpion. Abroug *et al.* (2020) in Tunisia reported that the Old and New World scorpions are usually contrasted because of differences in venom composition, clinical presentation and severity, and, accordingly, different therapeutic approaches. The majority of scorpion stings are either dry or result in low amounts of injected venom, thus explaining why up to 95% of scorpion stings ensue only in local signs. However, the clinician should always consider other possible diagnoses when evaluating a patient suspected of scorpion envenomation.

In older patients, a history of scorpion sting is typically present. However, scorpion stings may be confused with a wide array of other diagnoses. Although geographically confined in the United States, scorpions are known to "hitchhike" on passenger baggage or freight and cause stings in regions far removed from the Southwest. The abnormal eye movements following severe *C. exilicauda* envenomation are rarely seen in other diseases.

Conditions with symptoms that may overlap with scorpion envenomation include:

1-Spider bite: Bites or stings from other arthropods may present with symptoms similar to scorpion envenomation. Specifically, black widow envenomation may produce hypertension, tachycardia, sweating, and other signs of adrenergic excess. However, it does not produce the abnormal eye movements, fasciculations, or paresthesias, or induce a positive tap test as found with *C. exilicauda* (sculpturatus) scorpion stings. Also, black widow spider bites frequently produce a characteristic halo lesion at site, but *C. exilicauda* stings produce no such lesion (Cohen and Bush, 2005). Venom properties: *Loxosceles* venom contains a large number of enzymes and biologically active substances, of which sphingomyelinase D is the most important. This enzyme is unique in nature to *Loxosceles* and its sister genus, *Sicarius*, but is absent in all other spiders including closely related haplogyne spiders (Binford and Wells, 2003). Al-Agroudi *et al.* (2016) reported that spider bites are uncommon medical events, since there are limited number of spiders worldwide with fangs strong enough to pierce human skin, and most spiders bite humans only as a final defense when being crushed between skin and another object. Thus, most lesions attributed to spider bites are caused by some other etiology. The spiders that can cause medically significant bites include widow and false widow spiders (worldwide), recluse spiders (mostly North and South America), Australian funnel web spiders (eastern coastal Australia) and Phoneutria spiders (Brazil). Acute spider bites most commonly result in a solitary papule, pustule, or wheal. Systemic symptoms can accompany envenomation of widow; funnel web, and Phoneutria spiders, and less often, those of recluse spiders.

2- Hymenopterous stinging: Abdel-Rahman *et al.* (2015) in Egypt reported that the order Hymenoptera is third largest one of insects, comprising the sawflies, wasps, bees and ants. Worldwide, over 150,000 species are recognized, with many more remaining to be described. The name refers to the wings of the insects, but the original derivation is ambiguous. The Ancient Greek ὑμήν (*hymen*) for membrane provides a plausible

etymology for the term because these insects have membranous wings. However, a key characteristic of this order is that the hind wings are connected to the fore wings by a series of hooks called hamuli. Thus, another plausible etymology involves, Hymen, the Ancient Greek god of marriage, as these insects have "married wings" in flight. Stinging insects and the medical risk associated with their venoms are complex topics, and presentation of information pertaining to them requires the use of technical terms. The most common reactions to these stings are transient pain and redness at the site lasting a few hours (local reaction), and exaggerated swelling lasting a few days (large local reaction). The most dangerous immediate reaction is anaphylaxis, which is potentially fatal.

3- Botulism: Botulism is a rare but potentially life-threatening neuroparalytic syndrome caused by the action of a neurotoxin elaborated by the microorganism *Clostridium botulinum*. Botulism is classically described as the acute onset of bilateral cranial neuropathies associated with symmetric descending weakness. The CDC (2019a) suggested that the following points are considered as key for botulism syndrome features: 1- Absence of fever, 2- Symmetric neurologic deficits, 3- Patient remains responsive, 4- Normal or slow heart rate and normal blood pressure, & 5- No sensory deficits with the exception of blurred vision. El Bahnasawy *et al.* (2014) in Egypt reported that botulism is rare but potentially life-threatening neuroparalytic syndrome due to a neurotoxin caused by *Clostridium botulinum*. First investigation of botulism was in the 1820s with a case report on 100s of patients with "sausage poisoning" in a German town. Several decades later in Belgium, the association was reported between a neuromuscular paralysis and ham infected by a spore forming bacillus that was isolated from the ham. The organism was named *Bacillus botulinus* after the Latin word for sausage, botulus. They added that botulism in-

cludes 1- Infant botulism: commonest form of botulism begins after *C. botulinum* spores grow in a baby's intestinal tract, between ages of 2 & 6 months, 2- Foodborne botulism: harmful bacteria thrive and produce toxin in environments with little oxygen, such as in canned food, 3- Wound botulism: If bacteria get into a cut, they can cause a dangerous infection that produces toxin, & 4- Inhalational botulism: if aerosolized toxin released in an act of bioterrorism.

Thus, in contrast to a scorpion sting, botulism does not cause hypersalivation, fasciculation, or painful skeletal muscle contractions, but Duplantier *et al.* (2016) in USA reported that botulism caused neurotoxins (BoNTs), the most potent known toxins, cause severe muscle paralysis and death at the nanogram exposures as bio-threat agents. They added that BoNTs target the neuromuscular junction where they release smaller zinc metalloprotease light chains (LCs) into the neuron cytosol that selectively cleave snare proteins and thus block the exocytosis of acetylcholine neurotransmitters necessary for skeletal muscle contraction.

4- Esophageal or airway foreign body: Excessive oral secretions and respiratory distress from a scorpion sting may mimic the presentation of an upper airway or esophageal foreign body. Boo and Kim (2018) in Korea reported that major complications occur as a result of esophageal perforation; in particular, sharp foreign bodies, such as fish bones, are more likely to cause perforation. Complications include mediastinitis, paraesophageal abscess, pneumo-mediastinum, subcutaneous emphysema, pneumothorax, tracheoesophageal fistula, aorto-esophageal fistula, aspiration, and asphyxia. They added that unnecessary delays must be avoided in endoscopic intervention for esophageal foreign bodies to prevent complications.

Status asthmaticus: Occasional wheezing may follow the *C. exilicauda* (sculpturatus) or *C. vittatus* sting and be mistaken for an asthma exacerbation until systemic symptoms (e.g., paresthesias, neurologic abnor-

malities) become evident (Ulahannan *et al.*, 1996).

5- Toxic exposure: Several poisons have the physical findings similar to the scorpion envenomation. El-Bahnasawy *et al.* (2014a) in Egypt reported that insecticides are used to control diseases spread by arthropods, but they vary greatly in toxicity. Toxicity depends on the chemical and physical properties of a substance, and may be defined as the quality of being poisonous or harmful to animals or plants. Poisons have many different modes of action, but in general cause biochemical changes which interfere with normal body functions. Toxicity can be either acute or chronic. Acute toxicity is the ability of a substance to cause harmful effects which develop rapidly following absorption, i.e. a few hours or a day. Chronic toxicity is the ability of a substance to cause adverse health effects resulting from long-term exposure to a substance. There is a great range of insecticidal toxicity to humans. Organophosphorus, and carbamate insecticides (Bardin *et al.*, 1994), and nicotine and others ingestion can cause excessive oral secretions and muscle fasciculations. Organophosphate and nicotine overdose frequently cause paralysis, which is not seen with scorpion envenomation. Badr (2020) in Egypt reported that malathion is the commonest used organophosphates nowadays, as being considered to possess relatively low toxicity compared with other organophosphates, but with effects on liver, kidney, testis, ovaries, lung, pancreas, and blood. Lewander and Aleguas (2007) reported that hydrocarbons contain carbon and hydrogen as liquid at room temperature. All the petroleum distillates (kerosene, gasoline, mineral seal oils, & naphtha) are hydrocarbons but, not all hydrocarbons are petroleum distillates. Turpentine, for example, is a hydrocarbon from pine oil. Hydrocarbons are often mixed with agents that have systemic toxicity such as camphor, aniline dyes, heavy metals, and pesticides. Ingestion of large quantities of hydrocarbons alone by children

is unusual because hydrocarbons are foul-tasting. Poisoning in young children typically results from an exploratory occurrence that can be prevented by safe packaging and storage. In contrast, in teenagers and adults, aspiration may occur during intentional behaviors e.g., during inhalant abuse, or when attempting to siphon gasoline (Bronstein *et al.*, 2008). Hydrocarbons are toxic to all body systems, the most important toxicities occur in lungs, brain, and heart. Nicotine is an alkaloid obtained from leaves of the tobacco plant, and it is the main constituent of tobacco smoke but, nicotine causes acute and long-term toxicity with special attention to reproductive, cardiovascular, pulmonary, gastrointestinal, immunological and genetic toxicity (Karaconji, 2005).

5- Sympathomimetic agents (as methamphetamines, cocaine), hallucinogenics (as phencyclidine [PCP]), and anticholinergic agents (as antihistamines, Jimson weed) often cause motor excitability and hyperthermia similar to skeletal muscle dysfunction seen after a severe scorpion sting (Kent, 2004) one out of 18 inadvertent methamphetamine poisonings among children in central Arizona included three victims initially misdiagnosed with a scorpion sting and inappropriately treated with antivenom, had an anaphylactic reaction (Kolecki, 1998). But, poisoning with these agents does not typically cause cranial nerve findings and is usually associated with delirium. Bergman and Soares-Weiser (2018) in UK reported that the antipsychotic (neuroleptic) medication is used extensively to treat people with serious mental illnesses. Nevertheless, it is associated with a wide range of adverse effects, including movement disorders. Because of this, many people treated with antipsychotic medication also receive anticholinergic drugs in order to reduce some of the associated movement sideeffects. They added that from animal experiments chronic administration of anticholinergics could cause tardive dyskinesia. Wang and Hoyte (2019) in USA reported that novel drugs of abuse are syn-

thetic illicit drugs, or analogues of known illicit drugs, that can be more potent, and are often labeled as designer drugs, research chemicals, legal highs, or psychoactive substances. They added that pediatricians and emergency medicine physicians should be knowledgeable about novel drugs of abuse and their resulting symptoms for prevention and identification of their use.

6- Strychnine intoxication, tetanus, or dystonic reactions from medicines that antagonize dopamine receptors (as antipsychotic agents) may all present with painful muscle contraction (as opisthotonus), tonic-clonic movements, and a preserved mental status, similar to the clinical picture of severe scorpion envenomation. Smith (1990) in USA reported that strychnine poisoning is an unusual but dramatic poisoning in which convulsions are the major threat to life. Convulsions are predominantly at the spinal level, and the key to recognition of this poisoning is observation of convulsive activity in the awake patient without a postictal phase. They added that in absence of trauma, compartment syndrome, rhabdomyolysis, or anoxic central nervous system injury without neurologic or musculoskeletal squeals were expected.

CDC (2019b) reported that people often call tetanus “lockjaw” because one of the most common signs of this infection is tightening of the jaw muscles. Tetanus infection can lead to serious health problems, including being unable to open the mouth and having trouble swallowing and breathing, which possibly led to death (1 to 2 in 10 cases are fatal) Khoury and Cahill (2020) reported that tetanus is a life-threatening but vaccine-preventable disease caused by the toxin of the bacterium *Clostridium tetani* and is characterized by muscle spasms and autonomic nervous system dysfunction. It is prevented through vaccination with tetanus toxoid, but because the causative agent is widespread in the environment, eradication is impossible.

Balint *et al.* (2018) in UK reported that the

dystonia is a neurological condition characterized by abnormal involuntary movements or postures owing to sustained or intermittent muscle contractions. Dystonia can be the manifesting neurological sign of many disorders, either in isolation (isolated dystonia) or with additional signs (combined dystonia). They added that dystonia diagnosis is largely based on clinical signs, and the diagnosis and aetiological definition of this disorder remain a challenge.

7- Seizure: Patients with seizures may have muscle movements similar to victims of scorpion envenomation, but usually do not have an intact mental status or the characteristic roving eye movements that are seen in victims after the scorpion sting. Shellhaas (2019) in USA reported that the first weeks of life are a time of heightened risk for seizures due to age-dependent physiologic features of the developing brain that lead to increased neuronal excitation and decreased inhibition. Usually, seizures in neonates are a symptom of an acute brain injury; seizures are only rarely due to neonatal-onset epilepsy syndromes. Neonatal seizures are harmful to the developing brain; early and accurate diagnosis is critical. He added that seizure etiology evaluation must occur in parallel with initiation of appropriate treatment. It is critical that neonatologists and neurologists develop hospital-specific, consensus-based practice pathways for neonatal seizure evaluation and treatment.

8- Meningitis: Meningitis is defined as inflammation of the meninges, in almost all cases identified by an abnormal number of white blood cells in the cerebrospinal fluid and specific clinical signs/symptoms, fever, neck stiffness, hypertonia, and cerebrospinal fluid pleocytosis are seen in children with meningitis and, rarely, in those with severe scorpion envenomation (Putz *et al*, 2013). Abdelrahman *et al*. (2016) in Egypt reported that diagnosis of aseptic meningitis patient may be so-difficult because of the large variety of potential etiologic agents as viruses, fungi, parasites and some drugs and the

overlap between self-limited viral illnesses and potentially fatal bacterial infections. They added that a careful history should include travel and medical history as exposure to rodents (LCMV), ticks (Lyme *Borrelia*, RMSF, ehrlichia), mosquitoes (West Nile virus, St. Louis encephalitis virus), severe scorpion sting, and patients with T.B., sexual activity (HSV-2, HIV, syphilis), travel (*Coccidioides immitis*, *Angiostrongylus cantonensis*) and contact with others with similar symptoms or viral exanthems (enteroviruses), and he must also be questioned about medications and other comorbidities. Also, the commonest symptoms of envenomation included local pain, restlessness, and roving eye movements, but roving eye movements were not seen in meningitis (Rimsza *et al*, 1980).

9- Neuroblastoma: Opsoclonus-myoclonus syndrome (OMS) is a rare neurological disorder characterized by progressive opsoclonus (irregular, rapid, horizontal, and vertical eye movements), myoclonus, and cerebellar dysfunction (Sheila and Mani, 2004). Pang *et al*. (2010) in U K reported that, as many as 50% of children with opsoclonus-myoclonus have an underlying neuroblastoma, with characteristic symptoms of OMA were rapid, dancing eye movements, rhythmic jerking, and/or ataxia. Unlike children with severe scorpion envenomation, Opsoclonus-myoclonus syndrome is a very rare disorder with onset usually in the second year of life, those with neuroblastoma do not typically show hypersalivation, acute onset of cranial nerve deficits, or skeletal muscle effects. (Oh *et al*, 2019)

Nursing supportive treatment or comfort care, palliative care, and management of symptoms: It is important for the patient/guardian to understand that scorpion envenomation is unlikely to be a fatal disease process. So, antivenom is not life-saving. However, without antivenom, the patient will likely have a prolonged period of distressing symptoms, and of all available treatments, current evidence indicates antivenom is like-

ly to be effective and may significantly reduce the duration of suffering and hospitalization.

1- Asymptomatic or mild envenomation: Most scorpion stings result in mild envenomations (Grade I or II). Pain management by oral medications (as Ibuprofen[®] 10mg/kg; maximum single dose 800mg), cleansing of sting site, and tetanus prophylaxis typically suffice. Patients must be observed for four hours to ensure no more symptoms progression, although experience with *C. exilicauda* envenomation in Arizona showed that progression of envenomation grade in children occurs rapidly (mean time: 14 minutes, range 0 to 140 minutes). Prior to discharge, patients should tolerate oral intake, have no progression of symptoms, and their pain should be adequately controlled with oral medications.

2- Severe envenomation: Victims with significant systemic symptoms (e.g., restlessness, muscle fasciculations, hypersalivation, cranial nerve dysfunction, roving eye movements) are at heightened risk for respiratory compromise, myocardial infarction (in adults with ischemic heart disease), hyperthermia, rhabdomyolysis, and multiple organ failure. They warrant close monitoring for these complications.

3- Pregnancy envenomation: Ates *et al.* (2018) in Turkey reported that scorpion stings during pregnancy may not have significant adverse effects on the fetus and the mother. Decisions regarding the use of antivenom in pregnant women should be considered carefully when only limited safety information, especially in those patients with only local symptoms. Najafian *et al.* (2020) in Iran reported that envenomation significantly contributed to preterm birth. Also, bites location and the scorpion species have a decisive role in the pregnancy outcome of scorpion-envenomed pregnant women.

Key supportive interventions for these patients include: 1- Frequent suctioning of oral secretions, 2- Endotracheal intubation in patients with significant difficulties maintain-

ing their airway or with pulmonary edema accompanied by hypoxemia, 3- Close monitoring for and treatment of myocardial ischemia and/or acute decompensated heart failure in patients at risk, 4- Intravenous fentanyl 1mcg/kg for pain. Fentanyl was preferred if antivenom administration was planned because, unlike morphine, fentanyl does not cause histamine release, & 5- Intravenous benzodiazepines (lorazepam[®] or continuous midazolam infusion), titrated to effect, for sedation and to treat muscle spasticity if antivenom is not used.

Benzodiazepines should be used carefully or avoided if antivenom administration is planned. Antivenom reverses the excitatory effects of the scorpion venom and children who have received high doses of long-acting benzodiazepines (as lorazepam[®]) may become over-sedated, occasionally requiring intubation (Buisse, 2013). The short-acting benzodiazepine (as Midazolam[®], initial dose: 0.05 to 0.1mg/kg) was preferred to manage excess muscle activity and anxiety in pediatric patients. Once antivenom is given, the clinician should closely monitor respiratory and mental status for signs of over sedation and further dosing with benzodiazepines should be avoided.

4- Treatment by *Centruroides* antivenom: The use of antivenom for envenomation in the United States is controversial because of the low risk of mortality from the scorpion sting (given proper supportive care), lack of availability of an FDA approved antivenom, and the approximately 3% risk of anaphylaxis from administration of the goat-derived antivenom (not available). Cupo (2015) reported that the efficiency of antivenom serum treatment was controversial, in Brazil is that the management of patients with systemic manifestations of scorpion stings is based on three approaches, all of which are extremely important. These include symptomatic treatment, antivenom serum, and cardiorespiratory support.

Indications: Patients in the United States or Mexico with Grade III or IV symptoms

after *Centruroides* scorpion envenomation (skeletal muscle and/or cranial nerve dysfunction) received intravenous scorpion-specific F(ab')₂ equine antivenom.

Antivenom (Anascorp[®], US; Alacramyn[®], Mexico) is widely available in Mexico. Anascorp[®] is approved for use in USA (Boyer *et al*, 2013). Klotz *et al*. (2021) in Arizona reported that patients treated with (Anascorp[®]) antivenom exhibited a rapid resolution of symptoms without immediate or delayed hypersensitivity reactions, and recommended broadened availability of antivenom at sites where it is most needed.

To prevent allergy: Prior to administration of scorpion-specific F(ab')₂ antivenom, medications and equipment to treat anaphylaxis should be immediately available, including IV fluids, epinephrine, and intubation equipment (Erickson and Cheema, 2017).

Allergic reactions should be managed by immediately stopping intravenous infusion of the antivenom (if applicable) and treating symptoms appropriately. Key points emphasized anaphylaxis include the following: (1) validated clinical criteria are available to facilitate prompt diagnosis of anaphylaxis; (2) prompt intramuscular epinephrine injection in the mid-outer thigh reduces hospitalizations, morbidity, and mortality; (3) prescribing EAs facilitates timely epinephrine injection in community settings for patients with a history of anaphylaxis and, if specific circumstances warrant, for some high-risk patients who have not previously experienced anaphylaxis; (4) prescribing epinephrine for infants and young children weighing <15 kg, especially those who weigh 7.5kg and under, currently presents a dilemma, because the lowest dose available in EAs, 0.15mg, is a high dose for many infants and some young children; (5) effective management of anaphylaxis in the community requires a comprehensive approach involving children, families, preschools, schools, camps, and sports organizations; and (6) prevention of anaphylaxis recurrences involves confirmation of the trigger, discussion of sp-

ecific allergen avoidance, allergen immunotherapy (e.g., with stinging insect venom, if relevant), and a written, personalized anaphylaxis emergency action plan; and (7) the management of anaphylaxis also involves education of children and supervising adults about anaphylaxis recognition and first-aid treatment (Sicherer and Simons, 2017).

Delayed serum sickness-like reactions were not seen in patients receiving scorpion-specific F(ab')₂ antivenom reported in one case series (Bell *et al*, 2010). However, all patients received antivenom must be informed of the possibility of serum sickness and the symptoms suggestive of serum sickness and advised to seek medical care if such symptoms occur. Dose administration: The scorpion-specific F(ab')₂ equine antivenom is given intravenously in a dose of three vials dissolved in 20 to 50mL of normal saline infused over 30 minutes. Subsequent single vial doses of *Centruroides* antivenom, up to a total of five vials administered at thirty minutes intervals may be given until resolution of symptoms.

Goat-derived *Centruroides* antivenom: Scorpion-specific F(ab')₂ equine antivenom was distributed to Arizona Rural Hospitals by legislative mandate was no longer used. Bond (1992) in USA reported that the benefit of rapid resolution of life-threatening symptoms and potential for outpatient management of severe envenomated young children may justify the risk of acute and delayed reaction associated with Goat-derived antivenom, as its use as antivenin for the less severe envenomation common in youth and adults may subject them to unjustified with a frequency of immediate hypersensitivity reactions (3%) and up to 60% risk of delayed serum sickness. Al-Asmari and Al-Saif (2004) in Saudi Arabia found that beneficial effect of antivenom in protecting victims against scorpion stings is still questionable. They added that the risky systemic toxicity patients were either that with ages less than 10 or greater than 50 years, due to their decreased physiologic reserves

and increased debilitation.

Prevention: Reducing small cracks and crevices in homes decreases risk of human-scorpion interactions. In scorpion-infested areas, clothing, shoes, packages, and camping gear should be shaken out and checked for scorpions. Footwear is recommended. Unnecessary ground cover and debris must be removed to reduce the potential scorpion nesting places.

Home lifestyle: 1-Clean the wound with mild soap and water, 2-Apply a cool compress to affected area to reduce pain. 3- Don't consume food or liquids if you're having difficulty swallowing, & 4-Take an over-the-counter pain reliever as needed.

Certain insecticides, including organophosphates, nonsystemic organophosphate diazinon, pyrethrins, and several chlorinated hydrocarbons killed scorpions (Tomlin, 2006). Spraying insecticides around the home can work indirectly by killing other insects in the area and reducing the scorpions' food supply. A village-wide scorpion eradication program with pyrethroid insecticides in the state of Morelos, Mexico reduced the incidence of scorpion stings by 17% (Ramsey *et al*, 2002). One must remember that all insecticides are risks for environment and human health as well as domestic animals (El Bahnasawy *et al*, 2015)

Conclusion and Recommendations

Scorpion envenomation is a risky problem worldwide. In USA *Centruroides exilicauda* (sculpturatus) stings are associated with major neurologic toxicity, especially in young children. In Mexico, multiple toxic species exist, and annual mortality due to scorpion envenomation is ten times higher than that due to snakebite.

The majority of scorpion envenomations in the USA or Mexico cause only local or no pain with minimal to no inflammation. But, *C. exilicauda* (sculpturatus) and *C. suffusus* often produce significant neuromuscular effects of life-threatening. *C. vittatus* stings are less likely to cause systemic effects.

Clinical pictures after *C. exilicauda* enven-

Clinical pictures after *C. exilicauda* envenomation are divided into 4 clinical grades of envenomation. Envenomation by *C. suffusus* is similar. After *C. exilicauda* (sculpturatus) envenomation, symptoms may begin immediately and typically progress to maximum severity within five hours. Infants can reach the highest severity of envenomation in as quickly as 15 to 30 minutes after being stung. Although adults appear to be envenomed more often, children are more likely to develop severe illness requiring intensive supportive care.

Laboratory studies are not indicated in patients with mild (Grade I to II) envenomation. Severe effected patient (Grade III to IV) must undergo assessment: 1- Serum electrolytes, 2- Liver enzymes (AST & ALT), 3- Blood urea nitrogen and serum creatinine, 4- Lipase or amylase, 5- Serum creatine kinase, & 6-Urinalysis. Also, CBC and coagulation studies depend upon the clinical scenario.

Mild envenomation management: 1- Pain management with oral medications, cleansing of the sting site, and tetanus prophylaxis typically suffice in these patients. Patients should be observed for four hours to ensure no further progression of symptoms, & 2- Prior to discharge patient must tolerate oral intake, have no progression of symptoms, and their pain should be adequately controlled with oral medication.

Severe envenomation management: Victims with severe systemic symptoms (e.g., restlessness, muscle fasciculations, hypersalivation, cranial nerve dysfunction, roving eye movements) are at heightened risk for respiratory compromise, myocardial infarction (adults with coronary artery disease), hyperthermia, rhabdomyolysis, and multiple organ failure and warrant close monitoring for these complications.

Key supportive interventions in these patients encompass: 1- Frequent suctioning of oral secretions, 2- Endotracheal intubation in patients unable to maintain their airway or who develop pulmonary edema, 3- Intrave-

nous administration of fentanyl 1 mcg/kg. Fentanyl is preferred if antivenom administration is planned as, unlike morphine, fentanyl does not cause histamine release, 4- IV administration of benzodiazepines (as midazolam infusion titrated to effect) for sedation and to treat muscle spasticity. The benzodiazepines should be limited or avoided if antivenom administration is planned, & 5- Patients in the United States or Mexico with Grade III or IV symptoms after scorpion envenomation (skeletal muscle and/or cranial nerve dysfunction) receive intravenous scorpion-specific F(ab')₂ equine antivenom (Grade 2B).

References

- Abd El-Aziz, FEA, El Shehaby, DM, Elghazally, SA, Hetta, HF, 2019:** Toxicological and epidemiological studies of scorpion sting cases and morphological characterization of scorpions (*Leiurus quinquestriatus* and *Androctonus crassicauda*) in Luxor, Egypt. *Toxicol Rep.* 6:329-335
- Abdel-Rahman, MA, Omran, MA, Abdel-Nabi, IM, Nassier, OA, Schemerhorn, BJ, 2010:** Neurotoxic and cytotoxic effects of venom from different populations of the Egyptian *Scorpio maurus palmatus*. *Toxicon* 55, 2/3:298-306
- Abdel-Rahman, RZ, Mohamad, HM, Morsy, ATA, Morsy, TA, 2015:** Allergic reactions caused by venom of hymenopterous stinging insects and the role of health care workers. *J. Egypt. Soc. Parasitol.* 45, 2:403-12
- Abdel-Rahman, RZ, Morsy, ATA, Morsy, T A, 2016:** Aseptic meningitis in adults caused by virus, bacteria, drug with special references to zoonotic parasites. *J. Egypt. Soc. Parasitol.* 46, 2:329-350.
- Abroug, F, Ouanes-Besbes, L, Tilouche, N, Elatrous, S, 2020:** Scorpion envenomation: State of the art. *Intensive Care Med.* 46, 3:401-10.
- Agrawal, A, Kumar, A, Consul, S, Yadav, A, 2015:** Scorpion bite, a sting to the heart! *Indian J. Crit. Care Med.* 19, 4:233-6.
- Al-Agroudi, MA, Ahmed, SAM, Morsy, TA, 2016:** Intervention program for nursing staff regarding approach to a patient with spider phobia and/or bite. *J. Egypt. Soc. Parasitol.* 46, 1: 167-78.
- Al-Asmari, AK, Al-Saif, AA, 2004:** Scorpion sting syndrome in a general hospital in Saudi Arabia. *Saudi Med. J.* 25, 1:64-70.
- Alhamoud, MA, Al Fehaid, MS, Alhamoud, MA, Alkhalifah, AA, Alzoayed, MH, et al, 2021:** Scorpion stings in Saudi Arabia: An overview. *Acta Biomed.* 92, 4:e2021273.
- Amaral, CF, Rezende, NA, 1997:** Both cardiogenic and non-cardiogenic factors are involved in the pathogenesis of pulmonary edema after scorpion envenoming. *Toxicon* 35:997-9.
- Amr, ZS, Abu Baker, MA, Al-Sarairah, M, Warrell, DA, 2021:** Scorpions and scorpion sting envenoming (scorpionism) in the Arab Countries of the Middle East. *Toxicon* 191:83-103.
- Annobil, SH, 1993:** Scorpion stings in children in the Asir Province of Saudi Arabia. *J. Wilderness Med.* 4:241-8.
- Arie-Saadia, G, Sofer, S, Zlotkin, E, Shainberg, A, 1996:** Effect of *Leiurus quinquestriatus hebreus* venom on calcium & deoxyglucose uptake in cultured cardiac cells. *Toxicon* 34:435-40.
- Ates, S, Karahan, MA, Altay, N, Akelci, K, Ikiz, N, et al, 2018:** Approach to scorpion stings in pregnancy: A retrospective case series and literature review. *Taiwan J. Obstet. Gynecol.* 57, 5:692-5
- Badr, AM, 2020:** Organophosphate toxicity: updates of malathion potential toxic effects in mammals and potential treatments. *Environ. Sci. Pollut. Res. Int.* 27, 21:26036-57.
- Balint, B, Mencacci, NE, Valente, EM, Pisani, A, Rothwell, J, et al, 2018:** Dystonia. *Nat. Rev. Dis. Primers.* Sep. 20; 4(1):25. doi:10.1038/s41572-018-0023-6.
- Bardin, PG, van Eeden, SF, Moolman, JA, Foden, AP, Joubert, JR, 1994:** Organophosphate and carbamate poisoning. *Arch. Intern. Med.* 154, 13:1433-41.
- Bell, J, Wijegunasinghe, D, Samarakoon, S, Palipana, H, Gunasekera, S, et al, 2010:** Neurophysiological findings in patients 1 year after snake bite induced neurotoxicity in Sri Lanka. *Trans. R. Soc. Trop. Med. Hyg.* 104, 5:351-6.
- Berg, RA, Tarantino, MD, 1991:** Envenomation by the scorpion *Centruroides exilicauda* (*C. sculptratus*): Severe and unusual manifestations. *Pediatrics* 87:930-4.
- Bergman, H, Soares-Weiser, K, 2018:** Anticholinergic medication for antipsychotic-induced tardive dyskinesia. *Cochr. Database Syst. Rev.* 2Jan 17; 1(1):CD000204.
- Binford, GJ, Wells, MA, 2003:** The phylogenetic distribution of sphingomyelinase D activity in venoms of Haplogyne spiders. *Comp. Biochem. Physiol. B Biochem. Mol. Biol.* 135:25-32.

- Boo, SJ, Kim, HU, 2018:** Esophageal foreign body: Treatment and complications. *Korean J Gastroenterol.* 72, 1:1-5.
- Bond, GR, 1992:** Antivenin administration for *Centruroides* scorpion sting: risks and benefits. *Ann. Emerg. Med.* 21, 7:788-91.
- Bouaziz, M, Bahloul, M, Kallel, H, Samet, M, Ksibi, H, et al, 2008:** Epidemiological, clinical characteristics and outcome of severe scorpion envenomation in South Tunisia: Multivariate analysis of 951 cases. *Toxicon* 52:918-26.
- Boyer, L, Heubner, K, McNally, J, Buchanan, P, 2001:** Death from *Centruroides* scorpion sting allergy. *J. Toxicol. Clin. Toxicol.* 39:561-9.
- Boyer, LV, Theodorou, AA, Berg, RA, et al, 2009:** Antivenom for critically ill children with neurotoxicity from scorpion stings. *N. Engl. J. Med.* 360:2090-9.
- Boyer, LV, Theodorou, AA, Chase, PB, Os-naya, N, Berg, M, et al, 2013:** Effectiveness of *Centruroides* scorpion antivenom compared to historical controls. *Toxicon* 76:377-85
- Buysse, DJ, 2013:** Insomnia. *JAMA* 309, 7:706-16.
- Bronstein, AC, Spyker, DA, Cantilena, L RJr, et al, 2008:** Annual report of the American Association of Poison Control Centers' National Poison Data System (NPDS): 25th Ann. Rept. *Clin. Toxicol. (Phila)* 46:927-32.
- CDC, 2019a:** Botulism: Division of Foodborne, Waterborne, and Environmental Diseases.
- CDC, 2019b:** Tetanus: Symptoms and Complications.
- Chippaux, JP, Goyffon, M, 2008:** Epidemiology of scorpionism: A global appraisal. *Acta Trop.* 107: 71-9.
- Clark, RF, Selden, BS, Kunkel, DB, Frost, M D, 1991:** Abnormal eye movements encountered following severe envenomations by *Centruroides sculpturatus*. *Neurology* 41:604-6.
- Cohen, J, Bush, S, 2005:** Case report: Compartment syndrome after a suspected black widow spider bite. *Ann. Emerg. Med.* 45:414-20.
- Corneille, MG, Larson, S, Stewart, RM, Dent, D, Myers, JG, et al, 2006:** A large single-center experience with treatment of patients with crotalid envenomations: Outcomes with and evolution of anti-venin therapy. *Am. J. Surg.* 192, 6: 848-52.
- Cowart, BJ, 2011:** Taste dysfunction: A practical guide for oral medicine. *Oral Dis.* 17, 1:2-6.
- Cupo, P, 2015:** Clinical update on scorpion envenoming. *Rev. Soc. Bras. Med. Trop.* 48, 6:642-9.
- Curry, SC, Vance, MV, Ryan, PJ, et al, 1983:** Envenomation by the scorpion *Centruroides sculpturatus*. *J. Toxicol. Clin. Toxicol.* 21:417-21.
- Dehghani, R, Kamiabi, F, Mohammadi, M, 2018:** Scorpionism by *Hemiscorpius* spp. in Iran: A review. *J. Venom Anim. Toxins Incl. Trop. Dis.* 24:8-16.
- Dehesa-Dávila, M, Possani, LD, 1994:** Scorpionism and serotherapy in Mexico. *Toxicon* 32: 1015-20.
- Duplantier, AJ, Kane, CD, Bavari, S, 2016:** Searching for therapeutics against botulinum neurotoxins: A true challenge for drug discovery. *Curr. Top. Med. Chem.* 16, 21:2330-49
- Ebrahimi, V, Hamdami, E, Moemenbellah-Fard, MD, Ezzatzadegan Jahromi, S, 2017:** Predictive determinants of scorpion stings in a tropical zone of south Iran: Use of mixed seasonal autoregressive moving average model. *J. Venom Anim. Toxins Incl. Trop Dis.* 23:39-42
- El-Bahnasawy, MM, Aly, NZ, Abdel-Fattah, MA, Morsy, TA, 2014:** Botulism as a food poisoning: What is it? *J. Egypt. Soc. Parasitol.* 44, 1:211-20.
- El-Bahnasawy, MM, Mohammad, AE, Morsy, TA, 2014:** Hydrocarbon insecticides: Their risks for environment and human health. *J. Egypt. Soc. Parasitol.* 44, 2:361-72
- Erickson, TB, Cheema, N, 2017:** Arthropod envenomation in North America. *Emerg. Med. Clin. North Am.* 35, 2:355-75.
- Francke, OF, 2013:** Biodiversidad de Arthropoda (Chelicerata: Arachnida ex. Acari) en México. *Rev. Mex. Biodivers.* 85:S408-18.
- Gambhir, IS, Singh, DS, Pattnaik, DN, 1998:** Stroke in a young woman. *Postgrad. Med. J.* 74:555-9.
- Garcia, ML, Hanner, M, Kaczorowski, GJ, 1998:** Scorpion toxins: Tools for studying K⁺ channels. *Toxicon* 36:1641-8.
- Gateau, T, Bloom, M, Clark, R, 1994:** Response to specific *Centruroides sculpturatus* antivenom in 151 cases of scorpion stings. *J. Toxicol. Clin. Toxicol.* 32, 2:165-71.
- Hmimou, R, Soulaymani, A, Mokhtari, A, Arfaoui, A, Eloufir, G, et al, 2008:** Risk factors caused by scorpion stings and envenomations in the province of Kelâa Des Sraghna (Morocco) *J. Venom Anim. Toxi. Incl. Trop. Dis.* 14:628-40.
- Hutt, MJ, Houghton, PJ, 1998:** A survey from the literature of plants used to treat the scorpion stings. *J. Ethnopharmacol.* 60:97-9.

- Isbister, GK, Bawaskar, HS, 2014:** Scorpion envenomation. *N. Engl. J. Med.* 371:457-60.
- Kallel, H, Mayence, C, Guegueniat P, Didier, H, 2016:** Acute necrotizing pancreatitis after *Tityus obscurus* scorpion envenomation in French Guiana: Case report. *J. Clin. Toxicol.* 6:6-10.
- Karacnji, IB, 2005:** Facts about nicotine toxicity. *Arh. Hig. Rada Toksikol.* 56, 4:363-71.
- Kent, R, 2004:** Poisoning & Drug Overdose. Copyright © 2004, by the McGraw-Hill Co, Inc.
- Khoury, A, Cahill, JD, 2020:** Tetanus vaccination 2020 and collateral protections against pertussis and diphtheria. *R I. Med. J.* 103, 6:38-40.
- Klauber, M, 1997:** Rattlesnakes: Their Habits, Life Histories, and Influence on Mankind, Vol. 1, University of California Press, Berkeley.
- Klotz, SA, Yates, S, Smith, SL, Dudley, S Jr, Schmidt, JO, et al, 2021:** Scorpion stings and anti-venom use in Arizona. *Am. J. Med.* 134, 8: 1034-8.
- Kolecki, P, 1998:** Inadvertent methamphetamine poisoning in pediatric patients. *Pediatr. Emerg. Care* 14:385-90.
- Krifi, MN, el Ayeb, M, Ben Lasfar, Z, et al, 1992:** Improvement and standardization of anti-venoms sera. *Arch. Inst. Past. Tunis* 69:253-8.
- Lewander, WJ, Aleguas, A, 2007:** Petroleum distillates and plant hydrocarbons. In: Haddad and Winchester's Clinical Management of the Poisoning and Drug Overdose, 4th ed., Shannon, MW, Borron, SW, Burns, MJ (Eds.), Saunders Elsevier, Philadelphia.
- LoVecchio, F, McBride, C, 2003:** Scorpion envenomations in young children in central Arizona. *J. Toxicol. Clin. Toxicol.* 41:937042.
- LoVecchio, F, Welch, S, Klemens, J, et al, 1999:** Incidence of immediate and delayed hypersensitivity to *Centruroides antivenom*. *Ann. Emerg. Med.* 34:615-9.
- Morsy, TA, Khater, MKA, Khalifa, AKE, 2021:** Principle management of snake bites with reference to Egypt. *J. Egypt. Soc. Parasitol.* 52, 2:333-42.
- Najafian, M, Ghorbani, A, Zargar, M, Baradaran, M, Baradaran, N, 2020:** Scorpion stings in pregnancy: an analysis of outcomes in 66 envenomed pregnant patients in Iran. *J. Ven. Anim. Toxins Incl. Trop. Dis.* Apr 30; 26:e20190039.
- Nijssen, J, Comley, LH, Hedlund, E, 2017:** Motor neuron vulnerability, and resistance in amyotrophic lateral sclerosis. *Acta Neuropathol.* 133, 6:863-85.
- O'Connor, AD, Padilla-Jones, A, Ruha, AM, 2018:** Severe bark scorpion envenomation in adults<sup/> Clin. Toxicol. (Phila). 56, 3:170-4.
- Oh, SY, Kim, JS, Dieterich, M, 2019:** Update on opsoclonus-myoclonus syndrome in adults. *J. Neurol.* 266, 6:1541-8.
- Pang, KK, de Sousa, C, Lang, B, Pike, MG, 2010:** A prospective study of the presentation and management of dancing eye syndrome/opsoclonus myoclonus syndrome in the United Kingdom. *Eur. J. Paediatr. Neurol.* 14:156-61.
- Pickett, J, 2003:** Toxic and metabolic disorders of the neuromuscular junction. In: Office Practice of Neurology, Encyclopedia of Toxicology (Second Edition).
- Putz, K, Hayani, K, Zar, FA, 2013:** Meningitis. *Prim. Care* 40, 3:707-26
- Ramsey, JM, Salgado, L, Cruz, A, et al, 2002:** Domestic scorpion control with pyrethroid insecticides in Mexico. *Med. Vet. Ento.* 16:356-60.
- Rimsza, ME, Zimmerman, DR, Bergeson, PS, 1980:** Scorpion envenomation. *Pediatrics* 66, 2: 298-302.
- Russell, FE, 1991:** Venomous arthropods. *Vet. Hum. Toxicol.* 33:505-10.
- Sharma, PP, 2018:** Chelicerates. *Curr. Biol.* 28, 14:R774-8.
- Sheila, SR, Mani, PJ, 2004:** Opsoclonus myoclonus syndrome: Response to plasmapheresis. *Indian Pediatr.* 41:499-502.
- Shellhaas, RA, 2019:** Seizure Classification, Etiology, and Management. *Handb. Clin. Neurol.* 162: 347-61
- Sicherer, SH, Simons, FER, 2017:** Epinephrine for first-aid management of anaphylaxis: Section on allergy and immunology. *Pediatrics* 139, 3:e20164006
- Sofer S, 1995:** Scorpion envenomation. *Intensive Care Med.* 21:626-30.
- Smith, BA, 1990:** Strychnine poisoning. *J. Emerg. Med.* 8, 3:321-5.
- Stipetic, ME, Lugo, A, Brown, B, et al, 1998:** A prospective analysis of 558 common striped scorpion (*Centruroides vittatus*) envenomations in Texas during 1997 (meeting abstract). *J. Toxicol. Clin. Toxicol.* 36:461-9.
- Tomlin, CDS, 2006:** The Pesticide Manual: A World Compendium, 14th ed.; British Crop Protection Council: Hampshire, UK.
- Ulahannan, T, Hardern, RD, Hamer, DW, 1996:** Do accident and emergency senior house officers know the British Guidelines on the man-

agement of acute asthma? Postgrad. Med. J. 72: 162-3.

Vatanpour, H, Rowan, EG, Harvey, AL, 1993: Effects of scorpion (*Buthus tamulus*) venom on neuromuscular transmission in vitro. Toxicon 31:1373-8.

Wainger, BJ, Kiskinis, E, Mellin, C, Wiskow, O, Han, S, et al, 2014: Intrinsic membrane hyperexcitability of amyotrophic lateral sclerosis pa-

tient-derived motor neurons. Cell Rep. 7, 1:1-9.

Wang, GS, Hoyte, C, 2019: novel drugs of abuse. Pediatr. Rev. 40, 2:71-8

Wexler, P, 2014: Encyclopedia of Toxicology, English, 3rd Ed., Amsterdam Academic Press.

Zourgui, L, Maammar, M, Emetris, R, 2008: Taxonomical and geographical occurrence of Libyan scorpions. Arch. Inst. Pasteur Tunis 85, 1/4:81-9.

Table 1: Rapid overview: Emergent management of anaphylaxis in infants and children

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|----------------------------|---|
| Clinical diagnosis | Commonest signs and symptoms are cutaneous (as sudden onset of generalized urticaria, angioedema, flushing, pruritus) but, 10 to 20% without skin findings. |
| Danger signs: | Rapid symptoms progression, respiratory distress (as stridor, wheezing, dyspnea, increased work of breathing, retractions, persistent cough, cyanosis), poor perfusion, dysrhythmia, hypotension, collapse. |
| Acute management | Urgent therapy epinephrine. NO absolute contraindications to epinephrine in anaphylaxis setting. |
| Airway: | Immediate intubation if evidence of impending airway obstruction from angioedema; delay may lead to complete obstruction; intubation difficult but, done by the experienced clinician available; cricothyrotomy may be necessary. |
| Epinephrine | 0.01mg/kg IM (max 0.5mg), better in mid-antrolateral thigh, can repeat every 5 to 15 minutes as needed. If signs of poor perfusion or no epinephrine respond, prepare IV epinephrine for infusion |
| place | Patient in recumbent position, if tolerated, and elevate lower extremities. |
| Oxygen: | Give 6 to 8 liters/ minute via face mask, or up to 100% oxygen as needed. |
| Normal saline rapid bolus: | Treat poor perfusion with rapid infusion of 20ml/kg; reevaluate and repeat fluid boluses (2 ml/kg) as needed; massive fluid shifts with severe loss of intravascular volume occur; monitor urine output. |
| Albuterol: | Bronchospasm resistant to IM epinephrine, give albuterol 0.15mg/kg (mini 2.5mg) in 3 ml saline inhaled via nebulizer; repeat as needed. |
| H1 antihistamine: | Consider giving diphenhydramine 1mg/kg (max 40 mg) IV |
| H2 antihistamine: | Consider giving ranitidine 1mg/kg (max 50 mg) IV |
| Glucocorticoid: | Consider giving methylprednisolone 1mg/kg (max 125 mg) IV. |
| Monitoring: | Continuous noninvasive hemodynamic monitoring and pulse oximetry monitoring must be performed; urine output should be monitored in patients receiving IV fluid resuscitation for severe hypotension or shock |
| Refractory Symptoms | |
| Epinephrine infusion | Patients without response to IM epinephrine and IV saline, give epinephrine continuous infusion at 0.1 to 1 mcg/kg/minute, titrated to effect. |
| Vasopressors: | Patients may require large amounts of IV crystalloid to maintain blood pressure; if response to epinephrine and saline is inadequate, dopamine (5 to 20mcg/kg/minute) can be given as continuous infusion, titrated to effect. |

Table 2: Rapid overview: Emergent management of anaphylaxis in adults

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|------------------------------------|--|
| Diagnosis clinically | Commonest signs and symptoms cutaneous (sudden onset of generalized urticaria, angioedema, flushing, pruritus). But, 10 to 20% without skin findings. |
| Danger signs: | Rapid progression of symptoms, respiratory distress (stridor, wheezing, dyspnea, increased work of breathing, persistent cough, cyanosis), hypotension, dysrhythmia, chest pain, collapse. |
| Acute management: | Urgent therapy epinephrine for anaphylaxis. NO absolute contraindications to epinephrine in setting of anaphylaxis. |
| Airway: | Immediate intubation if evidence of impending airway obstruction from angioedema; delay may lead to complete obstruction; intubation difficult and must be done by experienced clinician; cricothyrotomy may be necessary. |
| Promptly and simultaneously, give: | IM epinephrine (1mg/ml preparation): Give epinephrine 0.3 to 0.5mg IM, better in mid anterolateral thigh; repeat every 5 to 15 minutes as needed. If not responding to epinephrine injections, prepare IV epinephrine for infusion. |
| Place: | Patient in recumbent position, if tolerated, and elevate lower extremities. |
| Oxygen: | Give 6 to 8 liters/minute via face mask, or up to 100% oxygen as needed. |
| Normal saline rapid bolus: | Treat hypotension with rapid infusion of 1 to 2 liters IV; repeat as needed; massive fluid shifts with severe loss of intravascular volume can occur. |
| Also consider administration of | |
| Albuterol | For bronchospasm resistant to IM epinephrine, give 2.5 to 5mg in 3ml saline via nebulizer; repeat as needed. |
| H1 antihistamine: | Consider giving diphenhydramine 25 to 50mg IV (to relief urticaria and itching only). |
| H2 antihistamine: | Consider giving ranitidine 50mg IV. |
| Glucocorticoid: | Consider giving methylprednisolone 125mg IV. |
| Monitoring: | Continuous noninvasive hemodynamic monitoring and pulse oximetry monitoring must be performed; urine output should be monitored in patients receiving IV fluid resuscitation for severe hypotension or shock. |
| Treatment of refractory symptoms: | |
| Epinephrine infusion* | Patients without response to IM epinephrine and IV saline, give epinephrine continuous infusion, 2 to 10mcg/ minute by infusion pump. Total dose continuously according to blood pressure, cardiac rate and function, and oxygenation. |
| Vasopressors* | Some patients require a second vasopressor (added to epinephrine). All vasopressors should be given by infusion pump, with the doses titrated continuously according to blood pressure, cardiac rate and function, and oxygenation. |
| Glucagon: | Patients on beta-blockers not respond to epinephrine, must give glucagon 1 to 5mg IV over 5 minutes, followed by infusion of 5 to 15micrograms per minute. |

* receiving an infusion of epinephrine and/or another vasopressor require continuous noninvasive monitoring of blood pressure, heart rate and function, and oxygen saturation.