

Scorpions Black widow Spider Rattlesnakes Bees Gila monster

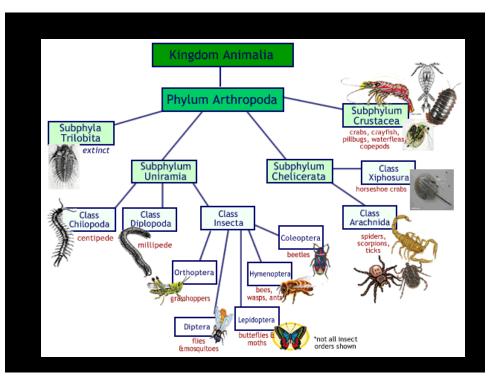
Objectives

Provide an overview of Envenomations encountered in the US

Outline pathophysiology of these injuries

Discuss optimal, initial care of patients with envenomations

3



Scorpions: Introduction

Phylum Arthropoda
Subphylum Chelicerata
Class Arachnida
Order Scorpionida



As many as 1400 species reported with ~ 30 capable of producing clinically significant envenomation

Buthidae largest / most dangerous family world-wide

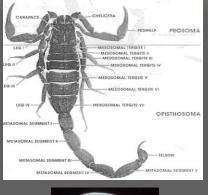
5

Scorpions: Introduction

Crablike body shape with 7 sets of paired appendages

Tail curves upward dorsally ending in terminal bulbous

Telson - contains paired venom glands and stinger





Scorpions: Introduction

Envenomation can result in distinct clinical syndromes

Most stings cause only local pain/inflammation

Some species in South America and North Africa can cause "autonomic storm"

Estimated 5000 deaths occur annually world-wide, 2nd only to snakes as sources of fatal envenomation

7

Scorpions: Venom

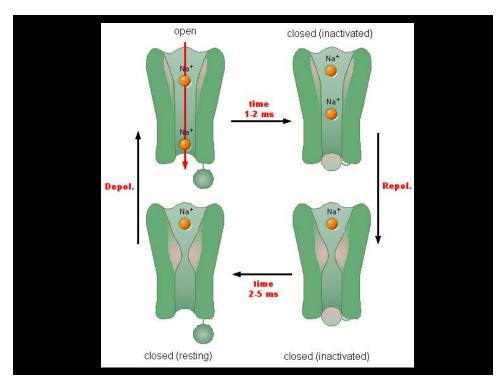
Contains several enzymes, neurotoxins, serotonin and histamine

Neurotoxin causes two effects:

Incomplete inactivation of sodium channels during depolarization Inward sodium current after repolarization

Net result: repetitive axonal firing, enhancing release of neurotransmitters at synapses/NM junctions

Net effect: excessive neuromuscular activity and autonomic dysfunction



Centruroides Sculpturatus

Of 40 species found in US, only *C. exilicauda* causes significant number of systemic reactions and is potentially fatal

Bark scorpion- resides in/near trees

Found statewide in Arizona, some areas Texas, New Mexico, northern Mexico, California

Accounts for ~ 10% of all calls to our poison center

Centruroides Sculpturatus:

Relatively small (5 cm)

Uniformly yellow/tan

Pincers/tail thin, streamlined



11

C. Sculpturatus: Envenomation

Grade I: Local pain and/or paresthesias at site of envenomation

Grade II: Pain and/or parasthesias remote from site of sting, in addition to local findings

Grade III: Cranial nerve dysfunction *or* somatic skeletal neuromuscular dysfunction

Grade IV: CN and skeletal neuromuscular dysfunction



Box 35-2 CENTRUROIDES EXILICAUDA ENVENOMATION, AS REPORTED BY AN INTENSIVE CARE SPECIALIST

Arriving home in the early evening, I decided to go for a run. My running shoes were in the kitchen area, where I had left them the day before. As usual, I would wear my shoes without socks. As I put my left foot into the shoe, I felt an intense burning pain on the dorsum of my first toe. I pulled my foot out of the shoe and along with it came a 1%- to 2-inch, clear-brown scorpion.

Having no idea what to do for a scorpion envenomation, I called the poison control center. I was informed that the systemic toxicity was usually mild for someone my age, and that if the pain was too severe, I should come in and be evaluated. As the minutes went by, I began to salivate and feel perioral paresthesias. As I walked, the paresthesias became more generalized, with a very noticeable paravertebral tingling with each step. After a few more minutes, I decided to call the poison control center to ask for advice. After dialing the number, I was unable to speak clearly because of severe dysarthria and excess salivation. The toe pain seemed to abate as other neurologic symptoms developed.

Since I was unable to talk on the phone, and no neighbors were home to drive me to the hospital, I decided to drive myself. The normal 10-minute drive took

45 minutes. I had coordination difficulties with the gas pedal, clutch, and gear shifting. It was also nighttime, and I could not process the multiple visual inputs of car lights, street lights, and road lines in a way that would allow me to drive more than 5 to 10 miles per hour. I not only had to stop frequently and close my eyes for a few seconds but also had difficulty keeping the car in my driving lane.

After arriving at the emergency department, I was ataxic, dysarthric, and drooling and had difficulty giving the admitting nurse a proper history. I'm certain that I was thought to be either mentally retarded or intoxicated. Examination by the ED physician revealed many abnormal cerebellar findings, continued salivation, inability to swallow liquids, continued symptomatic paresthesias, but no objective motor or sensory deficits. There were no physical signs of envenomation [at the sting site], but tapping the toe produced worsening pain. As my story became clearer to the ED physician, antivenom was ordered and administered. Within 20 minutes of finishing the infusion, all neurologic signs and symptoms were gone, except for toe pain.

Personal account of Dr. Thomas Bajo, Phoenix, Arizona.

13

C. Sculpturatus: : Treatment

Local: Observe for progression, symptomatic treatment

ABCs

Analgesia – fentanyl (1-2 mcg/kg IV)

Sedative Hypnotics - midazolam (0.05 - 0.1 mg/kg)

Continuous pulse oximetry and monitoring



Systemic progression ———— Antivenom

Historically

Goat-derived Antivenin (Phoenix)

Risks: Hypersensitivity, serum sickness

Anascorp[®]

Risks: Hypersensitivity

Benefits: Likely discharge from ED, rapid improvement

Made in Mexico, FDA approval in 2011

3 vials over 15 minutes; re-eval q30 minutes (max: 5 vials)

15

Black Widow: Introduction

Genus Latrodectus

Females 12-16mm thorax

Female shiny black with red hourglass on ventral abdomen

Habitate barns, garages, trash

heaps, outbuildings

Worldwide distribution

Every U.S. state except Alaska





Black Widow: Venom

Minimal local effects or inflammation



Neurotoxin, α -latrotoxin, releases neurotransmitter

Involves calcium and non-calcium mediated activities; membrane pore formation

Specific for presynaptic receptor of motor end plates; releasing acetylcholine and norepinephrine

17

Black Widow: Clinical Presentation

Latrodectism

Widespread, sustained muscle spasm following Latrodectus envenomation

Initial bite may be painful

Minimal, transient local reaction ("Target Lesion")

Small papule/punctum

Surrounding skin slight erythema/indurated

In most cases symptoms do not progress



Black Widow: Clinical Presentation

Neuromuscular signs/symptoms

Within 30-60 minutes

Involuntary spasm/rigidity of abdomen/limbs/back

'Acute abdomen'

Fasciculations

Weakness

Ptosis

Priapism

Respiratory muscle weakness

Black Widow: Clinical Presentation

Autonomic signs/symptoms

Salivation

Diaphoresis (can be localized)

Hypertension/hypertensive emergency

Fever

Bronchorrhea

Other: Pulmonary edema, uterine contractions, intractable crying (neonate), *Latrodectus* facies

21

Black Widow: Treatment

Pain/muscle spasm control

May remain severe for several days

Narcotics

Benzodiazepines

Calcium gluconate not helpful

Blood Pressure

Shorter acting, infusions, preferable easy on / off, only if analgesics / hypnotics don't work

Black Widow: Treatment

Antivenom

Indications: Uncontrolled pain, uncontrolled HTN, ACS, respiratory distress, seizures, pregnancy (?)

Old Antivenom

Single vial V over 30 minutes (100mL of NSS) No skin testing Have epinephrine at bedside

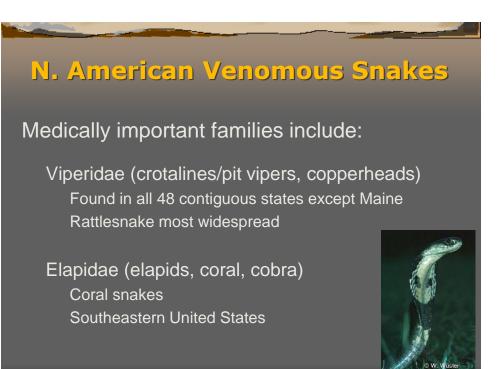
New Antivenom

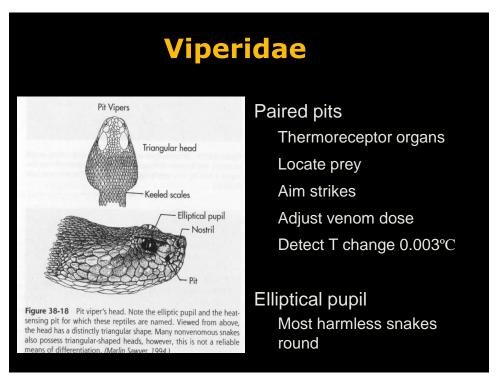
Experimental BioClon product Aracmyn PLUS®

23

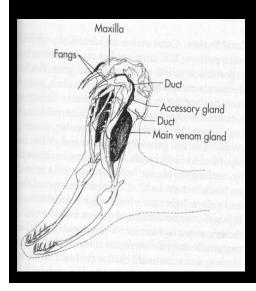


Good Samartian Regional Medical Center





Viperidae: Venom Delivery



Bilateral venom glands
Produce/store venom

Hollow fangs
Highly mobile

Voluntary control

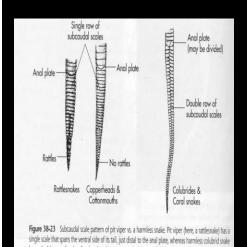
Brittle
Strike at 8 ft/second

Strike reach distances ½ body length away

1/4 bites "dry"

27

Rattlesnake: Shake, rattle, roll



has a double row of scales. Coral snakes also possess a double row of subcaudal scales. (Marlin Sawyer, 1994.)

Rattle

Loosely interlocked plates of keratin

Emits buzzing sound when vibrated

New segment added each shedding

Subcaudal scale pattern

Single Row = venomous

Double Row = nonvenomous

Viperidae: Regional Species

Timber Rattlesnake (*crotalus h. horridus*)

Canebrake

Eastern Massasauga (Sistrurus catenatus)

Copperhead (Agkistrodon contortrix)

Water Moccasin (Cottonmouth - A. piscivorus)

29

Viperidae: Venom

Complex mixture enzymes, metals

Proteolytic enzymes

Hyaluronidase

Phospholipase A₂

Thrombin-like enzymes

Collagenase

Rnase

Dnase



Viperidae: Venom Effects

Tissue injury

Most common complication

Enzymes breakdown tissue

Disrupt capillary endothelium

Necrosis of skeletal muscle



Coagulopathy/Thrombocytopenia

Fibrinolysins

Thrombin-like enzymes

Damage platelet membranes/initiate aggregation

31

Viperidae: Venom Effects

Cardiovascular toxicity

Hypotension- vomiting/hemorrhage

Myocardial depressor protein

Neurotoxicity

Mojave

Calcium-channel blockade in presynaptic neurons, inhibiting neurotransmitter release



Viperidae: Clinical Presentation Systemic effects GI: Nausea/vomiting CV: Hypotension, CV collapse, anaphylaxis Neurologic: Fasciculations, parasthesias, weakness, ptosis myokymia Hematologic: Thrombocytopenia, prolonged PT hypofibrinogenemia

Viperidae Bite: Management

Prehospital: Control bleeding

Elevate and immobilize effected limb (non-compressive splint)

NO ice/tourniquet/suction kits

Get to the antivenom

Analgesia/antiemetics/tetanus Fentanyl



35

Viperidae Bite: Management

Determine envenomation

Serial examination (progressive swelling/pain)
Baseline platelet, PT, fibrinogen (repeat 6 hours)

No evidence of envenomation in ED - discharge

Envenomation - admit

Leg bites: effects can be delayed - admit



Antivenom CroFab



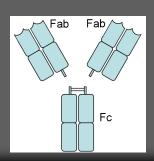
Fluid replacement/analgesia

Wound care

R/O Compartment syndrome

Simple debridement

Dermotomy / fasciotomy



37

Antivenom Adminsitration

CroFab

Indicated with significant envenomations

Progressive edema

Coagulopathy

Shock

Skin testing not routinely suggested

Low risk for anaphylaxis

CroFab Antivenom

'Safer' profile, apparently less effective (edema > coagulopathy)

Reconstitute 4 to 6 vials in 500 mL of NSS Initiate drip at 10 mL/hr and increase to 250 mL/hr

Evaluate for "Control" of envenomation

Recheck platelets, PT, fibrinogen and evidence of
edema progression

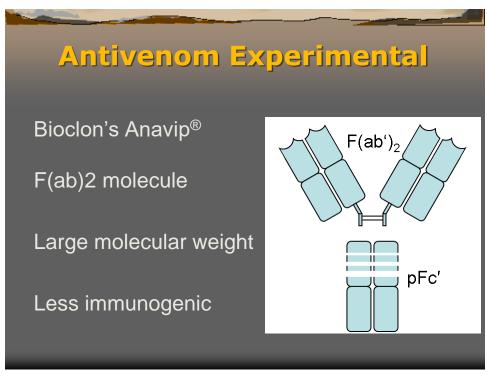
39

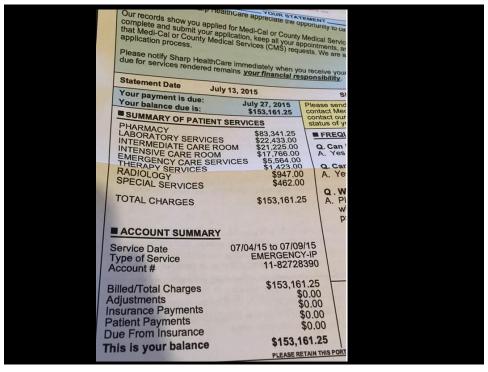
CroFab Antivenom

If "not controlled" re-administer 4 to 6 vials and then repeat testing

If "control" achieved:

2 vials every 6 hours for three doses
Each dose over one hour
Recheck labs within 4 hours after 3rd dose
Subsequent dosing based on P.E. and lab data







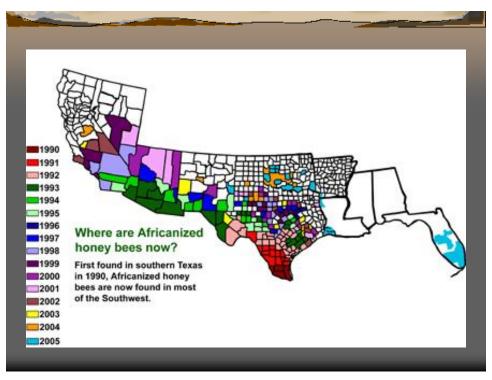
Africanized Bees

Apis mellifera scutellata/adansoni

More aggressive subspecies than native European bees of North/South America

Disease-resistant African bees imported in 1956 to Brazil and interbred with domestic honeybees (*Africanization*)

Africanized bees entered United States 1990





Africanized Bees: Venom

"Africanized" and domestic similar components, concentrations in venom sacs

Melittin

Major component

Inserts into phospholipid layer of cell membrane Causes breakdown of RBCs, WBCs, platelets, vascular endothelium

47

Africanized Bees: Venom

Phospholipase A₂
Increases capillary permeability

Morbidity associated with cumulative venom dose

>100 major systemic toxicity likely

Estimated human lethal dose ~ 20 stings/kg (~1500 stings in an adult)

Africanized Bees: Clinical Effects

Minor local reaction

Pain

Pruritis

Erythema

Urticaria

Major local reaction

Angioedema

Diffuse, widespread

edema

Major systemic reaction

N/V/D

Intestinal cramping

Bronchospasm/stridor

Shock

Delayed reactions (8-24hrs)

Hemolysis

Thrombocytopenia

Rhabdomyolysis

ARF

MI

49

Africanized bee attack killed man in Carefree

Agriculture Department warns public of hazards

By Robbie Sherwood

Tribune writer

The bees that attacked and killed a Carefree man were Africanized honey-bees—the so-called "killer" bees—an official at the State Department of Agri-

Africanized Bees: Management

Prehospital: Don't get swarmed; don't focus on removing stingers

ABCs

Local reactions: Analgesia, Cool compress, topical antihistamines

Systemic reactions: IVFs, antihistamines, steroids, epinephrine, bronchodilators

51

Africanized Bees: Management

<50 Stings

Baseline labs: CBC, CK, BMP, UA

Observe 6 hours

Asymptomatic, normal labs - Discharge Symptomatic, abnormal labs - Admit

>50 Stings

Baseline labs

Admit 24 hrs; observation for delayed effects

High risk: pediatrics, elderly, comorbidities



Gila Monster

Heloderma suspectum

Length ~ 50 cm



Massive jaw muscles with lancet-shaped, loosely-attached teeth

Venom delivery - pair of anterior multi-lobed glands that open into labial mucosa

Gila Monster

Agitation leads to salivation and venom flow

Chewing motion instills venom into wound by capillary action along grooves of teeth

Teeth and/or Gila monster may stay attached

Effective envenomation only 70% of bites

55

Gila Monster

Venom

Kallikrein-like substances

Hyaluronidase

Protease

Phospholipase A₂ Serotonin



Gila Monster: Clinical Effects

No fatalities, wound necrosis rare

Significant bleeding, local pain

Generalized weakness, nausea, vomiting, dizziness, parasthesias, tachycardia, hypotension, diaphoresis

Coagulopathy, thrombocytopenia, ECG abnormalities, MI reported

57

Gila Monster: Management

ABCs and Detach lizard!

Irrigate wound

Wound care, radiograph

Pain control, tetanus, antibiotics not routinely required unless evidence of infection

Scorpion - Treatment

Focus on the airway and airway secretions

Airway secretions (? Atropine)

Usually NOT an allergic reaction

Continuous pulse oximetry and monitoring

Pain medications or benzodiazepines

59

Blackwidow - Treatment

Consider the diagnosis (elderly and children)

Follow BP, ? ECG

Pain medications

Rattlesnake - Treatment

Immobilize Limb (straight, non-compressive splint)

No Tourniquet or Ice

IVFs (in non-effected limb)

61

Bees - Treatment

ABCs - Secure airway

IVFs

Anaphylaxis? (Epinephrine)

Don't Focus on the Stingers

Gila Monster - Treatment

Detach animal if its safe

Irrigate / Clean Wound

Pain medications

63

Regional Poison Center

Available 24 hours a day, 365 days a year

Can discuss case with a nurse or on call Medical Toxicologist

602-253-3334

1-800-222-1222

