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# Venomous Snakebites

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***Key words:*** *venomous snakebites; first aid; treatment.*

***Summary.*** *More than 5 million people are bitten by venomous snakes annually and more than 100 000 of them die. In Europe, one person dies due to envenomation every 3 years. There is only one venomous snake species in Lithuania – the common adder (Vipera berus) – which belongs to the Viperidae family; however, there are some exotic poisonous snakes in the zoos and private collections, such as those belonging to the Elapidae family (cobras, mambas, coral snakes, etc.) and the Crotalidae subfamily of the Viperidae family (pit vipers, such as rattlesnakes). Snake venom can be classified into hemotoxic, neurotoxic, necrotoxic, cardiotoxic, and nephrotoxic according to the different predominant effects depending on the family (i.e., venom of Crotalidae and Viperidae snakes is more hemotoxic and necrotoxic, whereas venom of Elapidae family is mainly neurotoxic). The intoxication degree is estimated according to the appearance of these symptoms: 1) no intoxication ("dry" bite); 2) mild intoxication (local edema and pain); 3) moderate intoxication (pain, edema spreading out of the bite zone, and systemic signs); 4) severe intoxication (shock, severe coagulopathy, and massive edemas). This topic is relevant because people tend to make major mistakes providing first aid (e.g., mouth suction, wound incision, and application of ice or heat). Therefore, this article presents the essential tips on how first aid should be performed properly according to the "Guidelines for the Management of Snake-Bites" by the World Health Organization (2010). Firstly, the victim should be reassured. Rings or other things must be removed preventing constriction of the swelling limb. Airway/breathing must be maintained. The bitten limb should be immobilized and kept below heart level to prevent venom absorption and systemic spread. Usage of pressure bandage is controversial since people usually apply it improperly. Incision, mouth suction, or excision should not be performed; neither a tourniquet nor ice or heat should be applied. A doctor must monitor respiratory rate, blood pressure, heart rate, renal function, fluid balance, and coagulation status. The only specific treatment method is antivenin – serum with antibodies against antigens of snake venom. Antivenins against pit vipers used in the United States are Antivenin Crotalidae Polyvalent (ACP) and a more purified and hence causing less adverse reactions – Crotalidae Polyvalent Immune Fab (CroFab). In Europe, a polyvalent antiserum against Viperidae family snakes (including the common adder) can be used. Antivenins often may cause severe hypersensitivity reactions because of their protein nature. The bite of the common adder (the only poisonous snake in such countries as Lithuania and Great Britain) relatively rarely results in death; thus, considering the risk of dangerous reactions the antivenin causes itself, the usage of it is recommended to be limited only to life-threatening conditions.*

## Introduction

Though European (and at the same time Lithuanian) wild nature is not as rich in the variety of venomous snakes as Asia or the United States, people still get bitten and some of them die. The problem of snake envenomation is becoming more relevant because of developing tourism to exotic countries, where the risk of encountering a poisonous snake increases. What is more, people working in the zoos or owning private collections of poisonous snakes are at constant risk of being bitten even in the countries, where these snakes are naturally nonexistent, e.g., Lithuania (North Europe). Therefore, the aim of this article was to update the medical staff with the basic knowledge on snake envenomation and change the popular old-fashioned approach to first aid because people still think that special actions, such as mouth suction, application of a tourniquet, ice, or heat are helpful; however, they can harm and must be avoided. What is more, antivenin is the only

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specific treatment with serum containing antibodies against venom antigens, and it helps to save lives of many who encounter highly poisonous snakes. However, it is related to high incidence rate of hypersensitivity reactions leading to death and, therefore, should be avoided in cases of less dangerous bites of the common adder occurring in Lithuania, unless the victim's life is threatened (1, 2).

**Epidemiology**

More than 5 million people are bitten by venomous snakes annually and more than 100 000 of them die, mostly in Asia. Every year, 6000–8000 envenomations occur in the United States, and 15–50 of them result in death. In Europe, one person dies due to envenomation every three years. More than 100 bites by the common adder (syn. European adder, *Vipera berus*) occur annually in Great Britain; 70% of patients have local reactions only (pain and swelling); however, 14 people have died over the past 100 years. There is only one venomous snake species in Lithuania – the common adder, which used to be found in the outer forest areas and by the bogs more often than it is nowadays. However, there are some exotic poisonous snakes in the zoos and private collections, such as the *Elapidae* family (cobras, mambas, coral snakes, etc.) and the *Crotalidae* subfamily of the *Viperidae* family (pit vipers, such as rattlesnakes) (1–4).

People at risk of being bitten by a snake are the agricultural workers, fishermen, hunters, tourists, and those working with venomous snakes. Alcohol consumption may be a risk factor as it diminishes attention. The limbs below elbows or knees are the most commonly bitten body area (90%). Even dead snakes can bite due to posthumous reflex, which makes oral muscles contract (5, 6).

**Pathogenesis**

Snake venom can be classified into hemotoxic, neurotoxic, necrotoxic, cardiotoxic, and nephrotoxic since snake venom has different predominant effects depending on the family (i.e., venom of *Crotalidae* and *Viperidae* snakes is more hemotoxic and necrotoxic, whereas venom of *Elapidae* subfamily is mainly neurotoxic). On the other hand, such classification is rather superficial as one kind of venom usually affects multiple organs and systems. Snake venom is a mix of toxic proteins and enzymes (2, 7–10).

After a snakebite, the venom is activated by body temperature and tissue pH. The enzyme hyaluronidase catalyzes the hydrolysis of the main interstitial constituent, thus, increasing tissue permeability and helping the venom spread. Proteolytic enzymes destruct the endothelium and basal membrane of capillaries (cytotoxic effect); thus, the capillary permeability increases, albumins leave to the perivascular spaces, and the tissue oncotic pressure increases, while plasma oncotic pressure decreases and edema occurs. Phosphatases hydrolyze phosphorous junctions of nucleotides. All of this tissue destruction promotes further spread of the venom; moreover, another component of the venom – antibactericidin – paralyzes the phagocytic activity of leukocytes and maintains the process of suppuration. Phospholipase A acts directly on erythrocyte membranes or indirectly through the production of lysolecithin and decreases osmotic erythrocyte resistance, which leads to intravascular hemolysis. Erythrocyte count decreases, and hemoglobinuria appears. Phospholipase A is not neurotoxic; however, it facilitates the access of active neuropharmacological poison components to nerve tissue. The snake venom also contains low-molecular-weight toxic peptides, kinins, leukotrienes, histamine, serotonin, acetylcholine, metalloproteins, glycoproteins, lipids, etc. The poisons together with partly hydrolyzed tissue products quickly get into the systemic circulation and cause hypotension, nausea, vomiting, tachycardia, and anuria (2, 4, 5, 9).

In case of severe envenomation, some snake venom can release autopharmacological vasoactive substances (bradykinin, histamine, and serotonin). Plasma kininogens release bradykinin, which in turn causes vasodilation. Such kininogen-induced vasodilation and hypotension as well as increased capillary permeability due to proteolytic enzymes cause hypovolemia, collapse, and shock. A myocardial depressant factor from the venom reduces myocardial contractility; thus, the cardiac output decreases and hypotension is potentiated. Signs of myocardial ischemia can occur. Acetylcholine directly affects the heart and neuromuscular junctions as well as facilitates the spread of toxic venom components. Unidentified neurotoxins, acting like curare, inhibit impulse transmission in neuromuscular junctions in presynaptic as well as postsynaptic membranes. Cholinesterase maintains the impulse blockage in neuromuscular junctions (2, 5, 9).

Cell detritus, hemoglobinuria, myoglobinuria, and venom-induced consumptive coagulopathy (VICC) damage renal tubules; besides, the venom can directly kill parenchymal cells and tubular epithelium and, together with systemic hypotension, cause acute renal failure (5, 9, 11, 12).

Snake venom causes hemorrhagic syndrome by activating the coagulation system in several different ways. Some of them are serine proteases, which can cleave fibrinopeptide A or B, or both from fibrinogen, thus, causing coagulation and secondary fibrinolysis. Some kinds of venom (metalloendoproteases possessing zinc in their structure) directly degrade fibrinogen, whereas others activate pro-

thrombin or factor X or directly aggregate platelets. Defibrination syndrome occurs.

In rare cases, especially after repeated snakebite, anaphylaxis can occur as a reaction to the same antigen or protein similar to it.

The intoxication degree is estimated according to the appearance of the following symptoms: 1) no intoxication ("dry" bite); 2) mild intoxication (local edema and pain); 3) moderate intoxication (pain, edema spreading out of the bite zone, and systemic signs); and 4) severe intoxication (shock, severe coagulopathy, and massive edemas) (3, 5, 9, 11, 13).

The severity and outcome of a snakebite depend on these factors: 1) bite localization; 2) snake's size, condition of glands and teeth, bite angle, and bite duration; 3) microflora of the snake's mouth and patient's skin; 4) patient's age, weight, and health status; 5) patient's activity after a bite (influencing the spread of the venom).

Snakebite to the limbs and adipose tissue is milder than that to the trunk, face, or directly to a vessel. If a snakebite is intracutaneous, the venom slowly spreads through lymphatic and superficial venous vessels, but there has to be a sufficient venom concentration to reach systemic circulation and cause systemic reactions in a few hours (only 50% of snakebites cause systemic intoxication). The venom in derma directly causes dermal tissue necrosis and increases capillary permeability, which leads to local edema formation with subsequent pressure and ischemia of the surrounding tissues, which in turn stimulate the progression of edema and tissue destruction. In rare cases, snakebites are intramuscular, thus, causing myonecrosis as well as compartment syndrome and ischemia. Snakebites are intravascular extremely rarely, and systemic symptoms appear in several minutes; thus, bites to well-vascularized regions (head and neck) are more dangerous. In case of a "dry" snakebite, no venom gets to the body (2, 3, 5, 9, 11, 13, 14).

**Signs and Symptoms**

Snake venom causes local and systemic body reactions. Local signs are puncture wounds inflicted by snake's fangs; there can be one, two, or in rare cases a few of them. A large snake's bite is indicated by a 15-mm space between punctures, whereas a space of less than 8 mm indicates a bite by a small snake. The wound is oozing with serohemorrhagic fluid and noncoagulant blood. If the bite wound is not bleeding or the blood is coagulating, it probably shows infliction by an insect, not by a snake. Local edema usually develops in 10 minutes (no longer than in 30 minutes) and spreading proximally can cover a whole limb or trunk; for children, a generalized edema may occur within 24 hours. One should bear in mind that the size of edema does not necessarily correlate with the severity of envenomation. There are redness and pain; however, it depends on the species of snakes. The venom of *Crotalidae* and *Viperidae* (the latter being found in Lithuania) snakes is characterized by dominating cytotoxic effect; therefore, the pain is severe and burning. On the other hand, the venom of *Elapidae* snake is predominantly neurotoxic; thus, the pain may be noncontinuous, mild, or there might be no pain at all. Petechiae, ecchymoses, and in rare cases vesicles (which fill up with hemorrhagic fluid within 8 hours) as well as local tissue necrosis appear rapidly (2, 7, 10, 14).

Systemic signs and symptoms are generalized, hematologic, and neuromuscular.

General symptoms are weakness, fatigue, anxiety, tachycardia, weak pulse, bradycardia, tachypnea, sweating, nausea and vomiting, diarrhea and acute abdominal pain, hypothermia or chills and fever, regional lymphadenopathy, lymphangitis (developing in 1–2 days), metal taste in mouth, headache, thirstiness, pulmonary edema, heart failure, hypotension, collapse, shock, cerebral anoxia (sleepiness, slurred speech, disorientation, delirium, loss of consciousness), and anaphylactic reaction to venom proteins (facial, tongue, and epiglottic angioedema, which causes respiratory tract obstruction). If hypotension develops in 2 hours, it is one of the main signs of intoxication. Hypotension may be of different duration: short (ceasing spontaneously in 2 hours), prolonged, or progressive and deadly. Renal damage manifests as proteinuria, hemoglobinuria, myoglobinuria, azotemia, and anuria. A convulsive syndrome may occur due to cerebral anoxia caused by hypotension and anemia, and not due to direct venom neurotoxicity. Electrocardiogram (ECG) may show nonspecific changes of ST segment and T wave, and episodes of atrial fibrillation (2, 9, 14).

Hematological changes appear as coagulopathy and hemorrhagic syndrome ranging from local bleeding to profuse blood loss: bleeding from gums, nose, hematemesis, melena, hematuria, petechiae, and ecchymoses in the snakebite area. Bleeding may occur from the kidneys, lungs, peritoneum, rectum, vagina, endometrium, and pathological sites such as peptic ulcers. There might be a slightly noticeable icterus. Laboratory tests show prolonged prothrombin and activated partial thromboplastin (APTT) time, decreased fibrinogen, plasminogen, factor XIII, factor V, antithrombin III, protein C levels, thrombocytopenia, elevated D-dimer level (a product of fibrin degradation), morphological erythrocyte changes and anemia (due to blood extravasation to the bite area), as well as neutrophilic leukocytosis.

Neurological symptoms are characteristic of snakebites of the *Elapidae* family and appear after a latent period of no more than 8 hours. Neuromus-

cular damage manifests as paresthesias in perioral region and fingers, muscular fasciculations, spasms, weakness, diplopia, hypersalivation, ptosis, myosis, dysphonia, dysphagia, trismus, respiratory dysfunction, and paralysis.

**Complications**

The causes of death are shock, intracranial hemorrhage into hypophysis, alimentary tract hemorrhage, and renal tubular necrosis (2, 3, 5, 11, 14).

**First aid**

It is essential to calm the patient down: hyperdynamic status may provoke the development of shock and worsen the condition due to the spread of venom. One must remove rings and other things that might constrict the swelling body part. The wound should not be washed or cleaned. Incision and suction should not be done because the incision in the bite area causes unnecessary tendon and nerve damage, and additional microflora enters the wound during suction by mouth. Even in case of perfectly performed incision and suction, only 20% of venom is removed. Besides, since snake fangs are curved, the collection of venom is not directly under the bite marks; therefore, only a very deep incision would reach it. Commercial extractors can be used to remove venom as they create negative pressure of 1 atm. Excisions must not be done at all (1, 4–8, 10, 11, 13, 14).

The patient should be laid down in a comfortable position, and the bitten limb should be immobilized using a splint or sling keeping it below the heart level. This is done in order to restrain any movements, which increase the absorption of venom into the bloodstream and lymphatics. If the patient's status is stable, the bitten limb may be elevated to reduce the edema (1, 6–8, 13).

Pressure-immobilization may be considered in cases of bites by *Elapidae* family snakes, since their venom is neurotoxic, and pressure might limit the spread of the venom through lymphatics; however, it is not recommended for viper bites as their venom acts mostly cytotoxically and causes local damage. In general, performing an efficient and safe pressure-immobilization requires skills and equipment, which is usually not available in a field situation and, therefore, should not be done (15, 16). The tourniquet must not be used in any situation (1, 6, 7, 10).

Ice applications are contraindicated as well. Though ice reduces pain and drainage of lymphatics, it causes additional ischemia, which in turn increases the damage.

It is very important to monitor and assure the respiratory function. To correct hypotension, crystalloid infusions are recommended. No alcohol or stimulants should be given. Pain may be relieved by giving analgesics.

Antivenom is not used for first aid due to high risks of adverse reactions.

Quick hospitalization of the victim is essential (1, 6, 11, 13).

**Treatment**

A doctor must assess and monitor the patient's blood pressure, heart rate, ECG, respiratory rate (since venom of coral snakes and rattlesnakes is neurotoxic and can impair breathing), renal function, fluid balance, leukocyte count, platelet count, and coagulation tests, as well as determine blood group and Rh status. Periodic measurement (every 15 minutes) of the injured limb circumference should be done in order to evaluate the dynamics of swelling and to prevent the compartment syndrome (17).

Antivenin is the only specific method of treatment; thus, in case of severe envenomation, it may be vitally important (1, 3, 4, 6, 8, 10, 11, 14).

In the United States, 2 types of antivenins against pit vipers (i.e., rattlesnakes) are currently used: Crotalidae Polyvalent Fab snake antivenin (CroFab) and Antivenin Crotalidae Polyvalent (ACP). Both antivenins are made by immunizing animals (CroFab – sheep, ACP – horses) with venom of different pit vipers. ACP is mostly comprised of horse IgG and albumins. CroFab is a more purified antivenin: ovine IgG molecules are degraded into Fc and Fab fragments. Fc fragments are removed since they cause anaphylactoid reactions to antivenin, whereas Fab (fragment antigen binding) molecules are saved because they bind the antigens of snake venom. It is thought that the use of ACP will be discontinued in the future.

Antivenins effectively correct clinical and laboratory signs; however, they are not necessarily effective as far as thrombocytopenia and rhabdomyolysis are concerned. Besides, protection from local tissue damage has never been proven (1, 4, 11, 13).

Administration of CroFab (for bites by pit vipers): the starting dose should be diluted in 250 mL of normal saline. The starting dose is 4 vials for mild bites, 4–6 vials for moderate bites, and 6+ vials for severe bites. Antivenom should be administered intravenously at a slow rate with epinephrine ready in case of an acute anaphylactoid reaction. If tolerated, the rate of infusion should be increased to get the entire dose administered in 1 hour. If symptoms of envenomation progress, the starting dose is repeated and this sequence is continued until stable; usually no more than 2 starting doses are needed. After stabilization, repeated dosing of CroFab should follow at 2 vials every 6 hours for 3 additional doses (11).

The administration of ACP is as follows: a skin test for possible allergy, though recommended by a manufacturer, is very insensitive, cannot accurately predict patients who will develop acute anaphylac-

toid reactions, wastes time and, therefore, should be omitted. Pretreatment with H1- and H2-blocking antihistamines in standard doses to mitigate possible acute hypersensitivity reactions should be considered. The starting dose should be diluted in 1000 mL of normal saline. The starting dose is 0–5 vials for mild bites, 10 vials for moderate bites, and 15 vials for severe bites. Antivenom should be administered intravenously at a slow rate with epinephrine at hand. If tolerated, after approximately 10 minutes, the rate of infusion should be increased to get the entire dose administered in 1–2 hours. ACP is then given as needed for continuous toxicity (in 5–10-vial increments with further dilution).

Adverse effects of antivenins are as follows: acute type I anaphylactic reactions due to circulating IgE reacting to horse proteins; anaphylactoid reactions due to direct degranulation of mast cells caused by horse proteins; serum sickness (a late-phase allergic reaction, caused by immune complexes, combined of patient's immunoglobulins against horse proteins – antigens). Anaphylactoid reactions to ACP occur in 25%–50% of the cases, whereas reactions to CroFab occur in 15% of the cases. They can present as hives, stridor, laryngeal edema, abdominal pain, vomiting, diarrhea, and hypotension. As many as 85% of the patients treated with ACP, but only 3% of those treated with CroFab, suffer from serum sickness, which manifests as fever, hives, myalgia, arthralgia, renal dysfunction, and neuropathy within 1–2 weeks after the administration of antivenin; it is though easily treated with oral steroids (i.e., prednisolone, 1–2 mg/kg per day orally) until the symptoms disappear. Oral antihistaminics can also relieve the symptoms (1, 9, 11, 13, 14, 17).

The snakes of the *Viperidae* family account for most of the envenomations in Europe; therefore, a specific antiserum is used in cases of snakebites by the common adder (European adder). This polyvalent antiserum of European viper venom (equine) neutralizes the absorbed venom of snakes of 6 species of the *Viperidae* family preventing or diminishing the effects of venom. If a patient has not previously received horse proteins, the complete dose can be administered at once, except in patients with allergic diathesis. In patients who had previously received horse proteins and had not developed an allergic reaction, an initial 0.2-mL dose of antiserum is administered subcutaneously, and if no allergic reaction occurs after 30 minutes, the remaining dose can be administered intramuscularly (preferably in the largest muscle mass such as the gluteal muscle). On the contrary, in patients who had previously received the equine antiserum and developed local or general reactions, as in individuals with allergy, antiserum of another animal should be given. However, if the administration of horse proteins is unavoidable, desensitization should be attempted by subcutaneous injection of 0.2-mL antiserum diluted in physiological solution at a ratio of 1:10 followed by injection of 0.2-mL undiluted antiserum after 30 minutes. If no reaction occurs within the next 30 minutes, the remaining quantity of undiluted antiserum can be administered intramuscularly. In case of a hypersensitivity reaction (anaphylactic reaction or serum sickness), an adequate treatment should be applied (1).

Taking into consideration the adverse effects of antivenins, they should be given when a systemic effect of venom appears (moderate or severe envenomation) (5). However, if a patient has been bitten by an *Elapidae* snake, the antivenin should be administered before the systemic symptoms develop as this kind of venom has a long latent period. Spreading local edema is not an indication for antivenin treatment, but rather demands monitoring. Antivenin must not be administered intramuscularly or to the site of the snakebite. The dose depends on the severity of envenomation.

The compartment syndrome should be assessed objectively by measuring the pressure in the limb. When the pressure exceeds 30–40 mm Hg, fasciotomy should be performed. The early use of antivenin can relieve the symptoms of limb ischemia as well as the destruction caused by venom. Usually, fasciotomy is not very efficient and can never substitute antivenin.

Tetanus prophylaxis should be done as the spores might enter the body during the snakebite (1, 11).

Prophylactic administration of antibiotics should not be done, unless it is known that the wound incision or suction by mouth had been previously done. In these cases, broader-spectrum antibiotics at standard doses can be given. If the wound becomes infected, antibiotics are given according to the flora cultured from the wound secretions; in 90% of the cases, the following bacteria are found: *Enterobacter* spp., *Pseudomonas* spp., and *Clostridium perfringens*.

For symptomatic treatment, antiemetics and antihistaminics are given; following hemorrhage, fresh plasma, cryoprecipitate, and platelet mass are used; hypotension is corrected by crystalloids and vasopressors; pain is relieved by nonsteroidal anti-inflammatory drugs; and muscle spasms or seizures are treated with calcium gluconate. Adequate respiration must always be maintained.

Envenomation by snakes might be complicated by acute renal failure due to acute renal tubular necrosis.

One of the main mistakes when treating snakebites is an inadequate dose of antivenin leading to major tissue necrosis, dissection, and vasculitis.

Later on, fibrosis may cause contractures reducing movement amplitude (1, 6, 11, 13).

Prognosis is worsened by very young or old age, debility, cardiovascular diseases, hypertension, diabetes mellitus, and coagulation disorders. The risk of bleeding is higher for those using anticoagulants, having coagulation disorders, peptic ulcer, endometriosis, and menstruations. The absence of microhematuria is a good prognostic sign showing that envenomation is not severe.

Insufficiently treated hypotension is the main cause of death in patients who suffered a snakebite.

Patients using beta-blockers are at higher risk of developing anaphylaxis to antivenin.

The damage to a fetus during pregnancy can be caused by uterine arterial hypotension and hypoxia, hemorrhagic complications, and placental abruption by venom-induced uterine contractions. The fetus might be unaffected as well.

There is no such thing as active immunization against snake venom; thus, bites in the future may cause envenomation again (2, 6, 9, 11).

When traveling in endemic areas, appropriate clothing and shoes, avoiding contact with the snakes (which are more likely to retreat than attack), and traveling with companions are the means for prophylaxis of snakebites (1, 3, 4, 8).

**Conclusions**

Although there is only one poisonous snake species in Lithuania (common adder or *Vipera berus)*, one should have knowledge of proper first aid and treatment of envenomation of other poisonous snakes as well, since they may be encountered in zoos, private collections, or during exotic trips. The clinical presentation of envenomation depends on the effect of different venom of specific snakes. The first aid measures are the following: the victim must avoid physical activity; and constricting items (e.g., rings) must be removed from the limb, which should be immobilized below heart level. Nonprofessionals should not apply a pressure bandage. Incision, excision, or mouth suction should not be performed; neither a tourniquet nor ice or heat should be applied. Vital signs and coagulation status must be monitored in order to provide efficient management. In Europe, the polyvalent antivenin against *Viperidae* (common adder) is recommended to be used in cases of only severe envenomation since the antivenin itself may cause dangerous hypersensitivity reactions rather often.

**Statement of Conflict of Interest**

The authors state no conflict of interest.

## Nuodingųjų gyvačių įkandimai

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**Raktažodžiai:** nuodingųjų gyvačių įkandimai, pirmoji pagalba, gydymas.

**Santrauka.** *Epidemiologija.* Pasaulyje kasmet nuodingosios gyvatės įkanda apie 5 mln. žmonių, iš kurių apie 100 tūkst. miršta. Europoje pasitaiko vienas mirties atvejis per kelerius metus. Lietuvoje esama tik vienos *Viperidae* šeimos nuodingųjų gyvačių rūšies – paprastoji angis (*Vipera berus*), tačiau zoologijos soduose bei privačiose kolekcijose gali būti laikomos ir egzotiškos nuodingosios gyvatės, priklausančios *Elapidae* šeimai (kobros, mambos, koralinės gyvatės) arba *Viperidae* šeimos *Crotalidae* pošeimiui (duobagalvės gyvatės, pvz., barškuolės).

*Patogenezė.* Gyvačių nuodus galima klasifikuoti į kategorijas: hematoksinai, neurotoksinai, nekrotoksinai, kardiotoksinai, nefrotoksinai, nes skirtingų šeimų gyvačių nuodai turi tam tikrą vyraujantį nuodų poveikį (pvz., *Viperidae* šeimos gyvačių nuodams būdingas labiau hematoksinis, nekrotoksinis, o *Elapidae* – neurotoksinis nuodų poveikis).

*Klinika.* Intoksikacijos laipsnis vertinamas pagal simptomų pasireiškimą: 1) nėra intoksikacijos („sausasis“ įkandimas); 2) lengva intoksikacija (lokali edema, skausmas); 3) vidutinė (skausmas, edema, išplintanti už įkandimo ribų, sisteminiai požymiai); 4) sunki (šokas, sunki koaguliopatija, masyvios edemos). Ši tema aktuali tuo, jog žmonės, teikdami pirmąją pagalbą, dažnai padaro šiurkščių klaidų (pvz., nuodų išsiurbimas burna, žaizdos incizija, ledo arba šalčio aplikacijos). Todėl, remiantis Gyvačių įkandimų gydymo gairėmis pagal PSO (2010), straipsnyje aprašomi pagrindiniai būdai, kaip teisingai suteikti pagalbą tokiais atvejais.

*Pirmoji pagalba.* Patyrus gyvatės įkandimą būtina ramybė. Nuo galūnės pašalinami daiktai, galintys veržti prasidėjus tinimui. Turi būti užtikrinta kvėpavimo funkcija. Pažeista galūnė imobilizuojama ir laikoma žemiau širdies lygio siekiant apriboti nuodų absorbciją ir sisteminį išplitimą. Dėl spaudžiamojo tvarsčio nuomonė prieštaringa, nes žmonėms dažniausiai pritrūksta įgūdžių teisingai jį uždėti. Negalima daryti incizijos, išsiurbimo burna, ekscizijos, negalima dėti turniketo, ledo, kaitinti.

*Gydymas*. Būtina stebėti paciento kvėpavimo dažnį, kraujospūdį, pulsą, inkstų funkciją, skysčių balansą, krešėjimo rodiklius. Vienintelis specifinis gydymo metodas – antiveninas. JAV vartojami dviejų rūšių antiveninai nuo duobagalvių gyvačių: ACP (Antivenin Crotalidae Polyvalent) ir labiau išgrynintas, todėl mažiau nepageidaujamų reiškinių sukeliantis CroFab (Crotalidae Polyvalent Immune Fab). Europoje vartojamas polivalentinis priešnuodis nuo *Viperidae* šeimos gyvačių įskaitant paprastąją angį. Antiveninai gali sukelti alergines (anafilaksiją, seruminę ligą) ir anafilaktoidines reakcijas. Kadangi angies (vienintelės nuodingosios gyvatės tokiose šalyse, kaip Lietuva ir Didžioji Britanija) įkandimas retai baigiasi mirtimi, todėl antiveniną dėl jo paties sukeliamų pavojingų reakcijų rizikos rekomenduojama vartoti tik išsivysčius gyvybei grėsmingai būklei.

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