Article/Artigo

Africanized honeybee stings: how to treat them

Picadas de abelhas africanizadas: como tratá-las?

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ABSTRACT

Introduction: In 1956, Africanized honeybees (AHB) migrated from Brazil to other regions of the Western Hemisphere, including South, Central, and North America, except for Canada. Despite being productive, they are highly aggressive and cause fatal accidents. This study aimed to evaluate patients at the Clinical Hospital of Botucatu Medical School (HC-FMB) and to propose treatment guidelines. **Methods:** From 2005 to 2006, the clinical and laboratorial aspects of 11 patients (7 male and 4 female) and the anatomopathological aspects of one patient who had died in 2003 were analyzed. **Results:** The age of the surviving patients varied from 5 to 87 years, with a mean of 42.5 years. The majority of accidents occurred in the afternoon, and the number of stings ranged from 20 to 500. The principal signs and symptoms were pain and local inflammatory signs, nausea, tachycardia, and vomiting. Biochemical findings presented increased levels of creatine phosphokinase, lactate dehydrogenase, and aspartate/alanine aminotransferase. An 11-year-old male patient died upon entering the attic of a two-storey building where he was attacked by a swarm, receiving more than 1,000 stings. He was sent to HC-FMB where he was treated, but he died 24h later. Observed at the autopsy were erythematous-purpuric skin lesions besides necrosis at the sting locations, rhabdomyolysis, focal myocardial necrosis, tubular hydropic degeneration and focal tubular acute necrosis of the kidneys, myoglobinuria, and centrolobular necrosis in the liver. **Conclusions:** Accidents caused by multiple AHB stings always constitute a medical emergency. As there is no specific antivenom, we have developed guidelines, including first aid, drugs, and the proper removal of stingers.

Keywords: Africanized honeybee stings. Treatment guidelines. *Apis mellifera.*

RESUMO

Introdução: As abelhas africanizadas (AHBs) migraram do Brasil em 1956 para todo o continente Americano. Apesar de produtivas, são agressivas causando acidentes fatais. O objetivo foi avaliar pacientes atendidos no Hospital das Clínicas da Faculdade de Medicina de Botucatu (HC-FMB) e propor um roteiro de tratamento. **Métodos:** Entre 2005 e 2006, foram analisados os aspectos clínicos e laboratoriais de 11 pacientes e anatomopatológicos de um que foi a óbito em 2003. **Resultados:** A idade dos pacientes variou entre 5 e 87 com média de 42,5 anos. Sete eram do sexo masculino e quatro do feminino. O número de picadas variou entre 20 e 500. Nove deles receberam mais de 50 picadas. Os principais sinais e sintomas foram dor local, náuseas, taquicardia e vômitos. Os exames hematológicos mostraram leucocitose, neutrofilia, anemia e desvio à esquerda escalonado. Os exames bioquímicos revelaram níveis elevados de creatinofosfoquinase, desidrogenase lática e aspartato/alanina aminotransferase. O paciente que foi a óbito 24h após o atendimento tinha 11 anos, era do sexo masculino e foi atacado ao adentrar um edifício de dois andares recebendo mais de 1.000 picadas. O exame anatomopatológico mostrou lesões eritemato-purpúricas, além de necrose nos locais das picadas. Apresentou também rabdomiólise, necroses focais do miocárdio, degeneração hidrópica acompanhada de necrose tubular renal aguda, mioglobinúria e necrose centrolobular no fígado. **Conclusões:** Os pacientes acometidos por múltiplas picadas necessitam de tratamento imediato e por não dispormos de um soro específico desenvolvemos um roteiro que inclui os primeiros socorros, as drogas a serem empregadas e a retirada dos ferrões corretamente.

Palavras-chaves: Picadas de abelhas africanizadas. Roteiro de tratamento. *Apis mellifera.*

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INTRODUCTION

At the outset of the 19th century, settlers from Europe brought the *Apis mellifera mellifera* bees to southern Brazil[1]. These bees were adapted to the European temperate climate, and upon arriving in Brazil, they presented low production of honey and its derivatives. This led the Brazilian government to implement a program to develop bees that would be more productive and better adapted to the tropical climate. Thus, in 1956, *Apis mellifera scutellata* queen bees were brought from Africa to Rio Claro, State of São Paulo, Brazil[2]. It was known at the time that the African bees were more productive and had greater swarming capacity but were much more aggressive. The objective of the project was to crossbreed the European bees already existent in Brazil with the African bees in an attempt to obtain a hybrid with the tameness of the European bees and the productivity of the African. By accident, some African queens escaped and initiated the natural Africanization of the bee group, initially in Brazil and subsequently throughout the Americas[3-6]. In 1990, the Africanized honeybees could be found in Texas and, today, are also distributed in Florida in the United States[7-15]. It is probable that in the near future, they will reach the vicinity of Canada, where they will face a harsh winter as their only natural barrier. On the one hand, pollination and honey productivity have increased substantially, making Brazil a major exporter of honey products today; on the other hand, the swarming capacity, aggressiveness, and attacks *en masse* have caused serious and fatal accidents in humans and other animals. Despite the small seasonal variations, the great difference between European and Africanized venoms is not in the quality but rather in the inoculated quantity resulting from massive attacks[16-18]. This causes serious envenomations and the development of an acute syndrome characterized by the release of a large quantity of pro-inflammatory mediators, including cytokines, thus causing a sharp inflammatory response that triggers disturbances in the immune system, heart, liver, kidneys, central nervous system, bone marrow, blood vessels, and skeletal muscles, among others[19-32]. As we do not yet have

a safe, efficacious antivenom, we have developed guidelines for the most appropriate care aimed at reducing the risk of death among these patients[32]. It must be emphasized that the Clinical Hospital of Botucatu Medical School (HC-FMB/UNESP) has been renowned in its region for over 40 years for the treatment of accidents by venomous animals. The objectives of this study were to evaluate the patients treated from 2005 to 2006 survived a massive attack of Africanized honeybees (AHB), to discuss the anatomopathological findings of one case that resulted to death in 2003, and to propose treatment guidelines that comprise emergency care, drugs, and the proper removal of stingers.

METHODS

A retrospective analysis of 11 accidents caused by multiple stings from AHB that occurred in the region of Botucatu, State of São Paulo, Brazil (48º 21'W, 22º 48' S) was carried out. These included eight adults and three children treated at the Tropical Diseases Unit of HC-FMB from 2005 to 2006. The epidemiological, clinical, and therapeutic data were collected by conducting a review of the respective patient charts, whereas the laboratory data were obtained by means of an informational system of the network of HC-FMB laboratories. The case of an 11-year-old boy from Botucatu, São Paulo, Brazil, who died in 2003 was presented. He was attacked by a swarm of AHB after he had entered the attic of a two-storey building located downtown to remove pigeon nests, unwittingly stimulating the beehive. Desperate, he jumped to the street from a height of approximately 10m. He was sent in critical condition to the HC-FMB but died after 24h.

Ethical considerations

This project was submitted to the Research Ethics Committee of Botucatu Medical School-UNESP and received approval on November 5, 2007 (Of.455/2007-CEP).

RESULTS

The 11 surviving patients ranged in age from 5 to 87 years, averaging 42.5 years. Seven (64%) were male; four were female. Four patients were attacked in May, three in October, and four in December. As to the period of the day, most (64%) of them were attacked in the afternoon and received medical care within 2h after the accident. The number of stings varied from 20 to 500, with 9 (82%) of the patients receiving more than 50 stings. None of the patients reported a previous attack by bees or said that the beehives were mechanically stimulated. The method adopted to remove the stingers was blade for three patients, tweezers for six, and unknown for two patients. **Table 1** summarizes these data.

Table 2 shows the clinical signs manifested by the patients. All patients reported local pain and local inflammatory signs (edema, erythema, itching). Nausea occurred in 73% of the cases, anemia in 64%, and tachycardia followed by vomiting in 55%. Hypertension and diarrhea, and generalized edema were detected in 45% and 36%, respectively. Dizziness, hypotension, fever, urticaria, weakness, palpebral edema, and wheezing lungs were also observed. Among these patients, arterial shock was not detectable.

Table 3 shows that leukocytosis with neutrophilia was presented by all patients, increased creatine phosphokinase (CPK) by 91%, neutrophilia with left shift and glucosuria by 55%, and increased lactate dehydrogenase (LDH) and aspartate aminotransferase (AST), hypoglycemia, and hematuria by 46%.

TABLE 1 - Identification of patients attacked by Africanized honeybees.

Parameters	Patients n	Patients %
Gender		
male	7	64.0
female	4	36.0
Month of the accident		
May	4	36.0
October	3	28.0
December	4	36.0
Period of the day		
morning	4	36.0
afternoon	7	64.0
Time until treatment (minutes)		
30	4	36.0
120	3	28.0
150	4	36.0
Stings (number estimated)		
20-30	2	18.0
50-60	2	18.0
100-150	2	18.0
200-300	4	36.0
300-500	1	10.0
Time to remove stingers (minutes)		
30-60	7	64.0
120-150	4	36.0

TABLE 2 - Clinical aspects of patients attacked by Africanized honeybees.

Clinical aspects	Present n	Present %	Absent n	Absent %
Local pain, edema, erythema, and itching	11	100.0	0	0.0
Nausea	8	73.0	3	27.0
Anemia	7	64.0	4	36.0
Tachycardia and vomiting	6	55.0	5	45.0
Hypertension and diarrhea	5	45.0	6	55.0
Generalized edema	4	36.0	7	64.0
Dizziness, hypotension, fever, urticaria, and weakness	3	27.0	8	73.0
Palpebral edema and wheezing lungs	2	18.0	9	82.0
Dry mouth, syncope, agitation, abdominal pain, laryngeal stridor, arthralgia, myalgia, anaphylaxis,and acute renal failure	1	9.0	10	91.0
Shock, arrhythmia, headache, convulsion, respiratory failure, oliguria, anuria, and central nervous system depression	0	0.0	11	100.0

TABLE 3 - Laboratorial tests.

Laboratorial tests	Increased		Normal		Not informed	
	n	%	n	%	n	%
Leukocytes and neutrophils	11	100.0	0	0.0	-	
Creatine phosphokinase (CPK)	10	91.0	-		1	9.0
Lactate dehydrogenase (LDH)	5	46.0	3	27.0	3	27.0
Aspartate aminotransferase (AST)	5	46.0	4	36.0	2	18.0
Erythrocyte sedimentation rate	2	18.0	2	18.0	7	64.0
Urea and creatinine	1	9.0	10	91.0	-	
Alanine aminotransferase (ALT)	1	9.0	8	73.0	2	18.0

Table 4 reveals the serum level of CPK observed over five days of hospitalization. On the first day CPK level did not exceed 353U/L, but it increased from the second day onward, reaching levels of 5.777U/L on the fourth day then decreasing from the fifth day onward.

TABLE 4 - Creatine phosphokinase* levels during hospitalization.

Patients	Hospitalization time				
	1st	2nd	3rd	4th	>5th
1	-	-	-	-	-
2	-	1,297	1,128	1,185	-
3	-	1,216	630	1,658	-
4	175	4,090	1,101	5,777	1,511
5	167	4,670	5,024	4,251	1,367
6	288	225	-	-	-
7	139	2,255	982	1,465	-
8	239	2,584	3,724	1,016	276
9	353	996	711	147	37
10	155	-	98	59	-
11	279	1,554	4,738	-	721

*CPK (U/L).

Treatment employed and clinical outcome

All the patients received stimulation to promote diuresis, principally by intravenous administrations of electrolytic solutions (100%), mannitol (91%), and/or loop diuretic (18%). Sixty-four percent received sodium bicarbonate by intravenous route for alkalinization of urine. Among the analgesics utilized, opioids (64%) were prominent. Finally, all received antihistamines, initially intramuscularly and subsequently orally. Only one patient did not receive intravenous corticotherapy. Non-opioid and diuretic analgesics were employed for five (45%) and two (18%) patients, respectively. The average time of hospitalization was 3.5 days, varying between 2 and 9 days, and all evolved to a complete cure without sequelae. The patient who received more than 1,000 bee stings arrived at the hospital unconscious, developed respiratory arrest, and subsequently died following a cardiac arrest.

Anatomopathological examination

The anatomopathological examination of the patient who died revealed the following: skin showing subepidermal vesicle with epidermal necrosis and mononuclear inflammatory infiltrate at the base associated with vasculitis, congestion, and edema in the adjacent dermis; skeletal muscle including rhabdomyolysis characterized by fiber eosinophilia, vacuolization, and absence of nuclei; heart showing necrotic cardiocytes characterized by fiber anucleation, eosinophilia, and lumpy cytoplasmic material, associated with discrete interstitial mononuclear infiltrate; kidney with tubular hydropic degeneration and myoglobinuria with eosinophilic amorphous material in Bowman's and intratubular space; and liver with centrilobular areas with hepatocytic necrosis and discrete steatosis.

DISCUSSION

Accidents by *Apis* bee stings present distinct clinical manifestations, depending on the sensitivity of the individual to venom and on the number of stings. The most frequent accident is that in which an individual who is not sensitized to the venom is affected by a few stings. In these cases, the clinical picture is limited to local inflammatory reaction with erythematous papules, pain, and heat. In most instances, this situation is resolved without the participation of a physician.

Another clinical presentation is that in which an individual previously sensitized to one or more venom components manifests immediate hypersensitivity. This can be triggered by only one sting and requires immediate medical intervention. In general, the clinical picture is manifested by laryngeal edema and bronchospasm accompanied by anaphylactic shock.

The third presentation is that of multiple stings. Generally, the accident occurs in the field and includes inoculation of a large quantity of venom caused by hundreds or thousands of stings[33-36].

The clinical picture of accidents always initiates on the skin, the location of a sting with venom inoculation. This event can evolve to necrosis, as shown in **Figure 1A**. This image shows a subepidermal vesicle with epidermal necrosis and mononuclear inflammatory infiltrate at the base associated with vasculitis. In addition, there was congestion and edema in the adjacent dermis. The presence of different molecules in the venom, including phospholipase A2, hyaluronidase, melittin, apamine[37-40], and, most recently, a serine protease-like protein studied by Lima et al.[41], may contribute to skin necrosis at the sting site. Such necrosis has frequently occurred and has been reported by most studies in Brazil[33-45].

The presence of rhabdomyolysis characterized by fiber eosinophilia, vacuolization, and absence of nuclei can be seen in **Figure 1B**. Rhabdomyolysis is a well-known cause of acute renal failure (ARF). Myoglobin toxicity has been related to renal vasoconstriction, intraluminal cast formation, and direct heme-protein cytotoxicity[46]. In a study conducted by Vanholder et al.[47], bee venom caused an early and significant increase in CPK, a reliable marker of the presence and intensity of muscle injury, and a massive tubular deposition of myoglobin. Other intramuscular cell enzymes like LDH and AST increased as well. Experimental bee venom-induced rhabdomyolysis with enzyme elevations was previously described by Azevedo-Marques and colleagues[44]. The main factor responsible for rhabdomyolysis in bee venom is most likely melittin[48]. This substance inserts itself into the phospholipid bilayer of cell membranes, causing hydrolysis and cell disruption[49]. Phospholipase A2 may also damage the cell membrane and may play a role in rhabdomyolysis[48]. Clinically, a number of cases of ARF induced by bee venom presented rhabdomyolysis that was evidenced by increased enzymes or myoglobinemia and myoglobinuria[33,36,50]. In the present study, the maximum levels of CPK were observed on the fourth day of hospitalization, in agreement with the literature[33-36].

The heart can also be affected in these accidents. Accordingly, **Figure 1C** shows necrotic cardiocytes demonstrated by fiber

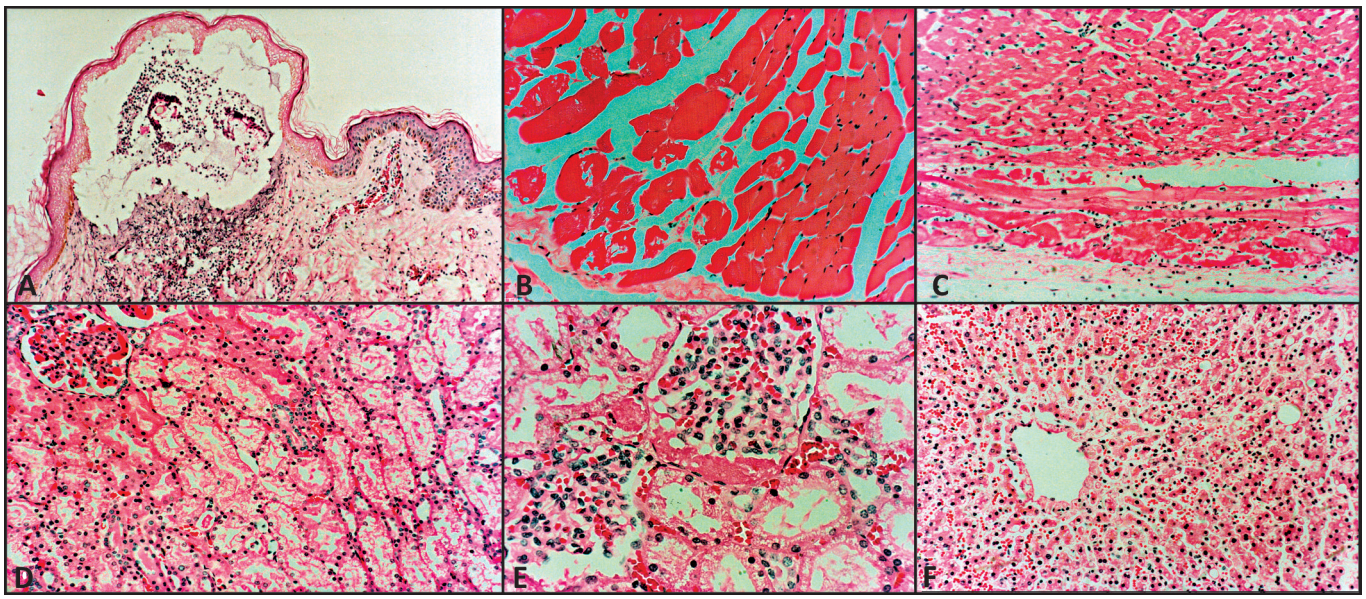

FIGURE 1 - A Skin showing subepidermal vesicle with epidermal necrosis and mononuclear inflammatory infiltrate at the base associated with vasculitis. Congestion and edema in the adjacent dermis HE (40X). B Skeletal muscle including rhabdomyolysis characterized by fiber eosinophilia, vacuolization, and absence of nuclei HE (400X). C Heart showing necrotic cardiocytes characterized by fiber anucleation, eosinophilia, and lumpy cytoplasmic material, associated with discrete interstitial mononuclear infiltrate HE (200X). D Kidney with tubular hydropic degeneration: cytoplasmic vacuolization with regular nuclei. Spots of acute tubular necrosis: nuclear pyknosis and cytoplasmic eosinophilia HE (200X). E Myoglobinuria: eosinophilic amorphous material in Bowman's space and intratubular space HE (400X). F Liver: Detail of centrilobular areas with hepatocytic necrosis: hepatocyte rarefaction, nuclear pyknosis, cytoplasmic eosinophilia, sinusoidal congestion, and edema of Disse's space. Discrete steatosis HE (400X).

anucleation, eosinophilia, and lumpy cytoplasmic material, associated with mild interstitial mononuclear infiltrate. The venom provokes rhabdomyolitic activity in both experimental animals and humans[44]. In severe cases, there are areas of myocardial necrosis similar to microinfarctions. There might be direct toxicity of the venom or only microinfarctions resulting from thrombosis of the microcirculation associated with the propagation of intravascular clotting[36,44-45]. Ferreira et al.[45,51] demonstrated enzymatic changes and morphological lesion of the acute myocardial infarction type, showing a possible direct toxic action of the venom on cardiac muscle.

On the other hand, the cardiac changes previously demonstrated in Wistar rats suggest that the components of the venom themselves or even substances released in the organism play some role in peripheral arteriolar resistance and may contribute to the changes in mean arterial pressure. In addition to presenting several vasoactive components, AHB venom contains melittin, a polypeptide toxin known to mobilize arachidonic acid from the cell membrane. Melittin evokes endothelium-derived hyperpolarizing factor-type relaxation by activating endothelial Ca2+-dependent phospholipase A2 followed by the transmission of a chemical and/or electrical signal via myoendothelial gap junctions. This vasorelaxation mechanism may be negatively regulated by nitric oxide[44]. Recent studies conducted by Guimarães et al.[43] concluded that the fall in mean arterial pressure is probably due to several factors, in addition to the cardiac changes already demonstrated in Wistar rats; it is possible that the venom components themselves or even substances released in the organism play some role in peripheral arteriolar resistance and may contribute to the changes in mean arterial pressure.

The kidneys require special attention in these accidents because hemolysis, rhabdomyolysis, and other direct effects of the venom contribute to acute tubular necrosis. **Figure 1D** shows a kidney with tubular hydropic degeneration: cytoplasmic vacuolization with regular nuclei and spots of acute tubular necrosis. Acute renal failure, which occurs after massive attacks by AHB, results from toxic and ischemic mechanisms accompanied by hypovolemic and/or anaphylactic shock associated with tubular lesion caused by pigments released by muscular lesion (myoglobinuria), by hemolysis (hemoglobinuria), and by the direct toxic effect of the venom itself[33-35]. This type of accident is followed by complications such as hypotension, hemolysis, rhabdomyolysis, clotting disturbances, and hepatic involvement[34,52]. According to Gabriel et al.[53], four hypotheses must be considered to explain the pathogenesis of acute tubular necrosis: I) a direct toxic effect of AHB venom on renal tubules, especially proximal tubules; II) a toxic effect of myoglobin and hemoglobin on the tubules (**Figure 1E**); III) an ischemic effect mediated by AHB or by substances released in the organism, or by a fall in cardiac output due to acute myocardial infarction-like lesions with a consequent fall in mean arterial pressure, reducing renal plasma flow; and iv) a stress-potentiating effect acting, for example, on nicotinamide adenine dinucleotide release in the myocardium, aggravating the cardiac ischemic component and consequently renal perfusion.

The hepatic changes shown in Figure 1F are similar to those described by Barraviera et al.[52,54] and reported in ophidian accidents. These alterations were recently confirmed by other authors[55-56]. These are unspecific lesions that may occur in the acute envenoming syndrome[19-20,32] previously described and in situations of shock. Initially, there is mitochondrial edema followed by Na+/K+ pump failure, hydropic degeneration, and hepatic necrosis[19-20,52,54]. In addition, lesions due to the venom itself may worsen necroses observed in severe cases[57]. Recently, Fighera et al.[57] studied six fatal cases of dogs attacked by the bee *Apis mellifera*. At necropsy, five of them had jaundice, red-orange liver, intensively blackish kidneys, and reddish urine. At histology, there was centrilobular hepatic necrosis, hemoglobinuric nephrosis, and lesions typical of intravascular hemolytic crisis. Based on these data, we can hypothesize that the liver may be affected by the direct toxic action of the venom, by the auto-immune reaction of the patient, and by the action of anaphylactic shock due to massive envenoming.

Finally, the accidents caused by multiple AHB stings always constitute a medical emergency. Early treatment, the removal of stingers, and precautions in relation to the status of hydration and renal function, in addition to the use of antihistamines, corticosteroids, non-opioid analgesics and diuretics, when indicated at appropriate doses, can be lifesavers in this type of accident.

Concluding remarks and recommendations

The treatment of patients stung by AHB should be approached as described below. First, we classify the case as an allergic or a toxic reaction. Next, we observe whether there is a local or a systemic affliction. The treatment must be conducted on a case-by-case basis, such as:

Allergic reactions: a) local (one or more stings): edema >10cm, progression up to 48h, duration in days, serous blister; b) systemic (one or more stings): reactions of hypersensitivity from degrees I to IV, including anaphylactic shock.

Toxic reactions: a) local (few stings): pain, erythema, low-intensity local edema, duration in hours; b) systemic (from 50 to 100 stings): pruritus, flushing, urticaria, sweating, fever, hypotension, headache, nausea or vomiting, abdominal cramps, bronchospasm, shock, respiratory failure, rhabdomyolysis, hemolysis, and ARF.

Allergic reactions

Accidents involving bee stings have different clinical manifestations depending on the individual's sensitivity to the venom and the number of stings. The most frequent accident is that in which an individual not sensitized to the venom is affected by a few stings. The clinical signs in these cases are limited to local inflammatory reaction, including erythematous papules, pain, and heat, and this situation is usually solved without medical assistance. When medical intervention is required, however, systemic antihistamines and topical corticosteroids are recommended. Dextrochlorpheniramine maleate at a dose of 2 to 6mg, administered orally every 6h for adults, and 0.15-0.30mg/kg weight up to a maximum of 5mg for children has been the drug of choice. This dosage must be maintained for at least 3 to 5 days depending on the case. In addition, we can use topical corticosteroids either alone or associated with menthol at 0.5%.

Another clinical presentation involves an individual previously sensitized to one or more venom components who manifests an immediate hypersensitivity reaction. This is a severe case that may be triggered by only one sting, and it requires immediate medical intervention. The general clinical signs include laryngeal edema and bronchospasm followed by anaphylactic shock. This patient requires hospitalization and immediate medical care. A thick vein from the patient's forearm should be catheterized for liquid and drug infusion. The latter should be maintained with glucose solution at 5% until the patient presents total hemodynamic stabilization. To promote hemodynamic stabilization in case of shock, it is necessary to offer 20ml/kg of saline solution (0.9%) or lactate ringer intravenously as fast as possible (less than 5min). This volume must be repeated until the signals of shock have disappeared. In case of anaphylactic shock, the drugs should be injected in the following order. First is liquid adrenaline at 1:1,000 dilution. It is the only effective and immediate measure and should be used subcutaneously or intramuscularly at the dose of 0.3 to 1ml (0.01ml/kg weight). In cases of cardiac arrest, intravenous and/or endotracheal routes should be used at 1:10,000 dilution. Second is promethazine: one to two ampoules by intramuscular or intravenous route. For children, use 0.5 to 1.0mg/kg weight. This drug has antihistaminic effect. We can also use diphenidramine in an intravenous route at a dose of 5mg/kg every 6h. In case there is no response, we must use anti-H2 antihistaminics. Third is aminophylline: This is a bronchodilator drug indicated for bronchospasm. Use 5mg/kg weight (0.3ml/kg weight). For children, we prefer to use inhalatory short-acting beta-2 agonists. Concomitantly, oxygen must be offered in a maximum concentration to maintain pulse oximetry above 92%. Fourth drug is hydrocortisone: This is a corticosteroid-type drug that inhibits the action of inflammation mediators. Use 5-10mg/kg body weight diluted in 100ml glucose solution at 5%. Administer repeated intravenous doses every 6h.

Toxic reactions

Multiple stings are the third presentation of this type of accident. In this case, treatment is always a medical emergency. Unfortunately, a specific antivenom is not yet available, although many researches have been conducted. Based on the experience of other Brazilian authors[33-36] and on the protocol used at the Department of Tropical Diseases, Botucatu Medical School, UNESP, the following measures should be taken as soon as the patient arrives at the hospital. A) Inject, by intramuscular route, an ampoule of promethazine; for children, use 0.5-1.0 mg/kg body weight. B) Inject, by intramuscular route, an ampoule of meperidine-type hypnoanalgesic; for children, administer 1.5mg/kg weight/day. C) If the patient is in shock, inject subcutaneously or intramuscularly from 0.5 to 1 ampoule of liquid adrenaline 1:1,000; for children, use 0.01mg/kg body weight. D) In case of bronchospasm inject, by intramuscular route, 1 ampoule of aminophylline; for children, preferentially, use short-acting beta-2 agonist. E) Catheterize a central vein, and then install central venous pressure; in case of shock, use intraosseous route if necessary. F) Administer intravenously 1g hydrocortisone; for children, use 5-10mg/kg body weight. This scheme should be maintained for at least three to five days, according to the clinical evolution. G) The patient must be well hydrated with colloids and crystalloids; then, osmotic diuresis should be induced intravenously with 20% mannitol, at a dose of 100ml, every 6h for adults and from 10 to 12.5ml/kg body weight for children. Mannitol administration should continue for at least five days. However, special attention should be paid to avoid iatrogenic dehydration. For patients presenting anuria, mannitol is contraindicated. H) Alkalinize the urine with sodium bicarbonate solution at a dose of 1 to 2mEq/kg weight/dose every 6h to prevent renal lesions caused by Hemoglobinuria and myoglobinuria.

Acid urine pH favors the establishment of lesions. I) Remove stingers one by one, paying attention to avoid inoculating the venom they contain. It must be emphasized that during stinging, only one-third of the venom contained in the stinger is inoculated into the victim. The remainder stays in the inoculation apparatus located in the proximal end of the stinger. Incorrectly removing stingers may lead to the compression of this apparatus. Consequently, inoculation of a large quantity of venom will follow. To remove stingers, use a razor close to the skin. J) Perform vesical and nasogastric catheterization. K) Apply potassium permanganate at 1:40,000 dilution for antisepsis of the stung areas. L) Accomplish enteral feeding of around 2,000 calories per day. M) Maintain the hydroelectrolytic and acid-base balance. N) Perform tracheostomy and/or orotracheal intubation, with assisted ventilation when indicated. O) Conduct peritoneal dialysis or hemodialysis, when there is ARF. P) Prevent the formation of decubitus sores. Q) Avoid secondary respiratory infections.

Finally, regarding such injuries, treatment choice is always challenging and must be assessed case by case, especially when the patient is a child or an adult over 60 years. In these situations age, weight, and possible associated morbid conditions should be considered when choosing the best therapy to avoid unfavorable clinical outcomes.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

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