Wild Animal Injury in Taiwan

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Abstract

Taiwan's varied landforms contribute to its abundant wild life species. Despite the decreasing number of wild animals caused by the cultivation of forest zone and over hunting on the island, cases of wild animal bite are still reported each year. The focus of care for these cases includes wound care, tetanus immunization, and human-animal communicable diseases prevention and treatment. It is also an important part of wildness medicine. Along with the frequent interactions among Taiwan and other rabies epidemic areas and the rampant smuggling of wild animal in black market, the threat of rabies needs to be critically considered and recognized in Taiwan.*(Ann Disaster Med. 2004;3 Suppl 1:S18-S29)*

Key words: Wild Animal Injury; Wild Animal Bite; Rabies

Introduction

Due to the varied landforms and the subtropical climate, there are 64 species of mammals in Taiwan (Figure) (Table 1).[1,2] Although the number of some wild animal has decreased due to the development of circumstances and hunting, sporadic animal injury events are reported every year.

Wild animal injuries include bites or claw wounds from a wild animal, the crush injuries caused by large animals and the diseases transmitted by wild animals through wounds or by close contact. Due to the limited species of large mammalian animals including the Formosan black bear, boar , and wild deer in Taiwan, and the decreasing number of these species caused by unrestricted hunting and cultivation of land, and the environmental change of their natural habitat, only very few cases of large animal injury are reported in Taiwan every year. The more commonly seen cases are biting wounds caused by smaller mammalian animals such as Formosan macaques.

The most concern of animal bite transmitted diseases is Rabies worldwide. No rabies case has been reported in Taiwan since 1959. However, due to the frequent interflow and smuggling with Mainland China and other countries with rabies-epidemic, rabies is becoming an emerging problem. A rabies case was identified in Taiwan and reported in July, 2002. As reported, the victim was first bitten by a dog in Mainland China, then starting feeling ill and eventually died in Taiwan.[3] As evidenced, Taiwan is affected and threatened by rabies. In addition to rabies, other important and ever-

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reported diseases transmitted by animal injury in Taiwan also include: Tetanus infection, rat-bite fever and cat scratch fever.

Mammalian Bite Wound and Mauling

The biting wounds caused by wild mammalian can be various. The severity of wounds may span from a simple scratch to more severe

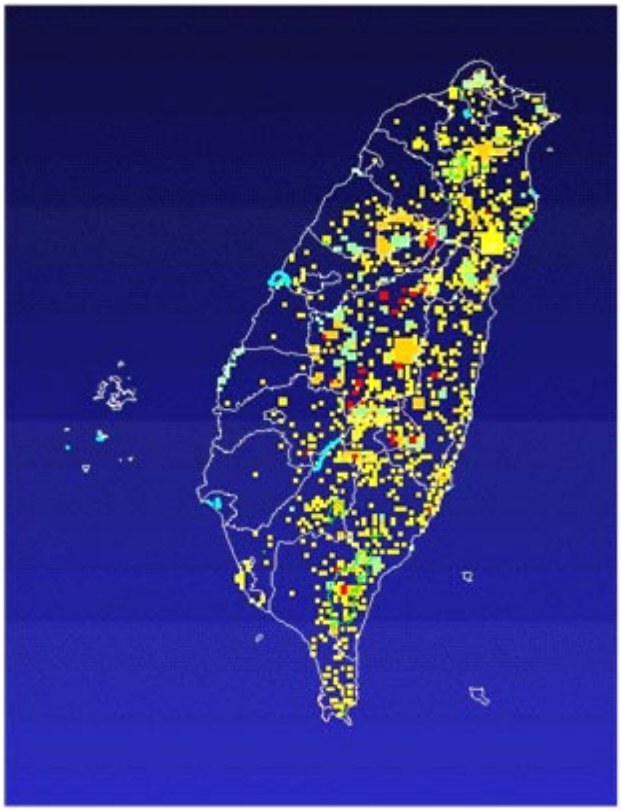

Figure. Distribution of mammals in Taiwan

Table 1. Wild mammal species in Taiwan

Formosan masaque 臺灣獼猴、Taiwan Redbellied Tree Squirrels 赤腹松鼠、Formosan giant flying squirrel 大赤鼯鼠、White-faced flying squirrel 白面鼯鼠、Formosan weasel 黃鼠狼、Formosan ferret-badger 鼬獾、Formosan gem-faced civet 白鼻心、Formosan wild boar 臺灣野豬、Formosan Barking Deer 山羌、Formosan Serow 臺灣長鬃山羊 Asiatic water shrew 水鼩、Kuroda's shrew 臺灣小麝鼩、Tada's shrew 蘭嶼小麝鼩、Hosletti's shrew 臺灣白足鼩鼱、Taiwan long-tailed shrew 臺灣煙尖鼠、House shrew 家鼩、Formosan flying fox 臺灣狐蝠、Formosan greater horseshoe bat 臺灣大蹄鼻蝠、Formosan lesser horseshoe bat 臺灣小蹄鼻蝠、Formosan leaf-nosed bat 臺灣葉鼻蝠、Formosan tailles leaf-nosed bat 臺灣無尾葉鼻蝠、Watase's orange whiskered bat 渡賴氏鼠耳蝠、Broad-muzzled whiskered bat 寬吻鼠耳蝠、Formosan whiskered bat 臺灣鼠耳蝠、Horikawa's bat 堀川氏棕蝠、Bent-winged bat 褶翅蝙蝠、Hairnan lesser-hellow bat 高頭蝙蝠、Formosan tube-nosed bat 管鼻蝠、Vespertilo orientalis 東方食蟲蝠、Formosan striped field mouse 赤背條鼠、House mouse 家鼷鼠、Formosan black-bellied vole 臺灣黑腹絨鼠、Formosan mountain field vole 臺灣高山田鼠、Chinese otterr 水獺、Formosan clouded leopard 臺灣雲豹、梅花鹿 Cervus nippon taiwanus

punctures, lacerations or avulsions . All can result in significant damage, regardless of the amount of bleeding present. Larger mammalians are able to deliver a bite force of greater than 450 pounds per square inch,[4-6] capable of perforating light sheet metal.(Table 2) This type of crush injury is common and may result in tearing and tissue devitalization, which enhances the risk for infection. Lacerations or, less commonly, puncture wounds and avulsions may also occur. Among the various wound types, puncture wounds are touted to have the highest incidence of infection, whereas injuries involving the hand or joint carry the greatest risk of infection and disfigurement. Other potential complications that should be considered include osteomyelitis, septic arthritis, tenosynovitis, local abscesses and, rarely, endocarditis, meningitis, brain abscess and sepsis.[7]

Complications of Bites

The most common complication of bite is infection. Bleeding is another common complication especially when there are vessel injuries. Mammalian teeth may result in a deep laceration, thus creating a route for microorganisms transmission. Osteomyelitis, septic arthritis, tendinitis or tenosynovitis may be seen with tooth penetration into a bone or joint[8]. Bites of the cranium may yield central nervous system infections such as brain abscess.[9] Endocarditis, lymphangitis, meningitis and sepsis with disseminated intravascular coagulation have also been reported. Other infectious complications of mammalian bites include cat-scratch disease (Afipia felis), rat-bite fever (Streptobacillus moniliformis), rabies, tularemia (Francisca tularensis) and tetanus.[10]

Treatment

Emergency care should be sought immediately if there are serious injuries, the person is suffering from severe blood loss, significant flesh loss, or there are many bites. It is also important to seek emergency care if the person has been bitten by a strange animal.

Wound Management

1. Open wounds should be irrigated with copious volumes of normal saline as soon as possible. The preferred way is to use high pressure irrigation with normal saline or lactated ringers via a 19 gauge needle and large syringe.
2. The police should be notified of the incident.

Table 2. Mechanism of large animal injury (19)

Animal Type (No. Patients)	Fall/Thrown n (%)	Kicked n (%)	Stepped on or Trampled n (%)	Gored n (%)	Crushed[a] n (%)	Other[b] n (%)
Equine (n = 79)	45 (57)	16 (20)	3 (4)	—	11 (14)	4 (5)
Bull (n = 47)	14 (30)	5 (10)	21 (45)	6 (13)	—	1 (2)
Cow (n = 16)	—	6 (38)	4 (25)	1 (6)	—	5 (31
Wild game (n = 3)	—	—	—	2 (67)	—	1 (33
p value	0.003	0.04	<0.001			

[a] Denotes full weight of animal falling on or pinning the rider.
[b] Denotes individuals who were butted, knocked to the ground, bitten, or injured during roping activities.

Adapted from Norwood.S. Mechanisms and Patterns of Injuries Related to Large Animals. Journal of Trauma 2000;48(4),740-744

3. When possible, tetanus toxoid and rabies vaccination status should be determined for all animal bites. (The rabies vaccination is not recommended in Taiwan)
4. Assess the extent of damage to the tissues including joint involvement, consider a roentgenogram to rule out a fracture if suspected, determine the need for cosmetic repair, stratify the patient for risk factors of infection, obtain wound cultures if infection is suspected (i.e. fever, erythema at injury sites, and observe necrosis or purulent wound drainage).[11] (Table 3)

Microbiology

Infections caused by animal bites are often polymicrobial, with an average of three to five bacterial species isolated per wound culture.

Staphylococcus spp., Streptococcus spp., Corynebacterium and various gram-negative enteric pathogens are the most commonly identified aerobes. Bacteroides spp., Fusobacterium, Peptostreptococcus, Actinomyces, Veillonella parvula and Eubacterium are commonly identified anaerobic organisms (11)

Antimicrobial Therapy

Prophylactic treatment

It has been suggested that antibiotic serum concentrations should be therapeutic within 3 hours after the injury. The recommended duration of prophylactic therapy with oral agents is a period of 5-7 days.

Those with more severe wounds, diabetes, vascular disorders, or who are immunocompromised may require a more aggressive approach with oral or parenteral antibiotics. The selection of an antibiotic regimen should follow the same criteria of empiric therapy of an infected bite wound.

Table 3. Evaluation and care of bite wound

1. Complete history of injury
   a. Time of bite
   b. Information of biting animal
   c. Injury circumstances –attack provoked? Precipitating events?
   d. Patient health history – allergies, tetanus immunization status?
2. Physical examination of the wound
   a. Measure and classify
   b. Range of motion, neurovascular examination, and tendon function
3. Baseline X-rays if hand or if area is over bone
4. Cultures (anaerobic and aerobic) and gram stains if signs of infection
5. Copious wound irrigation with soap and water and/or saline
6. Evaluation for closure or surgical referral
7. Wound immobilization and elevation
8. Tetanus immunization evaluation
9. Rabies prophylaxis evaluation
10. Antibiotic therapy: prophylactic or therapeutic if indicated
11. WBC test and blood cultures if systemic infection is suspected
12. Patient teaching for wound care and risk of infection
13. Follow up in 1or 2 days if not hospitalized

Empiric antibiotic therapy of infected wounds

An appropriate empiric antibiotic regimen must direct at the pathogens most likely to cause infection, including both aerobic and anaerobic bacteria. Therapy should target organisms from both the oral cavity of the animal, as well as potential pathogens from the skin flora of the victim.

Indications for hospital admission include signs of sepsis, fever, significant edema or crush injuries, spreading cellulitis, involvement of a bone, joint, tendon or nerve, failed outpatient therapy, and immunocompromised hosts. Intravenous therapy may also be necessary in patients with known histories of medication noncompliance.

The most common agent for mammalian bites is monotherapy with amoxicillin/clavulanate owing to the additional anaerobic coverage offered by the [beta]-lactamase inhibitor. Alternative regimens include clindamycin plus ciprofloxacin, dicloxacillin plus penicillin, tetracyclines, trimethoprim/sulfamethoxazole, and second- or third-generation cephalosporins such as cefuroxime.[11] (Table 4)

Rabies

Several countries, most of which are islands, are rabies free, including the British Isles, New Zealand, Japan, Taiwan, many of the Caribbean islands, Sweden, Norway, and Spain. The fact that these countries remain free of rabies is a tribute to the stringency of their quarantine laws for imported animals. Australia was at one time believed to be rabies free, but bat-transmitted rabies is now endemic there.

Although no rabies case was identified in Taiwan since 1959, due to close interactions and black market animal smuggling among Taiwan and other rabies plagued countries, the diagnosis of rabies should considered while a history of animal bites is presented. At present, rabies vaccine is not required for human as a preventive measure when biting by animasl in Taiwan. Even a rabies case was identified in 2002 in HuaLiang (花蓮), the bite took place by a dog in Mainland China instead of Taiwan. Furthermore rabies is not yet detected among local wild species in Taiwan for the time being. However, preventive rabies vaccine is required

Table 4. Selected antibiotic regimens for therapy of bite wounds

Oral regimens	Parenteral regimens
Amoxicillin/clavulanate	Ampicillin/sulbactam
Ciprofloxacin + clindamycin	Ciprofloxacin + clindamycin
Azithromycin	Azithromycin
Penicillin + dicloxacillin	Penicillin + nafcillin
TMP/SMX + clindamycin or metronidazole	TMP/SMX + clindamycin or metronidazole
Cefuroxime	Cefuroxime or ceftriaxone
Doxycycline ± dicloxacillin or cephalexin	Doxycycline ± nafcillin or cefazolin
Levofloxacin	Levofloxacin
Trovafloxacin	Alatrofloxacin
Sparfloxacin	

by the Council of Agriculture for all domestic animals in order to prevent the spread of the disease through animal smuggling from nearby rabies epidemic areas.

Rabies is a fatal viral encephalitis that causes 30,000 to 70,000 deaths worldwide each year. Prevention is the primary approach to the disease. Rabies is transmitted usually by saliva from infected animal bites. However, recent findings that rabies can be transmitted from bats to humans by relatively casual contact have resulted in dramatic changes in guidelines from the Centers for Disease Control and Prevention for postexposure prophylaxis.

Pathogenesis

Rabies is caused by a bullet-shaped RNA rhabdovirus that is a member of the Rhabdoviridae family, genus Lyssavirus. Rabies is transmitted by saliva from infected animal bites but may also be transmitted by scratches, secretions that contaminate mucus membranes, aerosolized virus that enters the respiratory tract, and corneal transplants. In recent years, it was noted that rabies could be transmitted from bats to humans by relatively casual contact. The rabies virus has a predilection for nerve tissue and spreads along peripheral nerves and possibly muscle fibers from the contact site to the central nervous system (CNS), causing encephalomyelitis.

Clinical Features

Rabies is a fatal disease once clinical symptoms manifest. Only 6 documented cases survived after onset of clinical rabies. All these patients had received either preexposure prophylaxis or expeditious postexposure prophylaxis after the rabid contact and before the patients had established clinical disease. Rabies presents with 1 of 2 clinical features. Encephalitic (furious) rabies (80%-85% of cases) has the classic presentation with hydrophobia, pharyngeal spasms, and hyperactivity leading to paralysis, coma, and death. The paralytic form is much less common. Incubation periods range from 10 days to 1 year (average, 20-60 days). Prodrome occurs 2 to 10 days after exposure and lasts 1 day to 2 weeks. This stage is characterized by nonspecific flu-like symptoms such as malaise, anorexia, irritability, low-grade fever, headache, nausea, vomiting; paresthesia, pain, or numbness at the bite site.

Acute neurologic syndrome occur 2 to 7 days after the prodromes. This syndrome includes dysarthria, dysphagia, excessive salivation, diplopia, vertigo, nystagmus, restlessness, agitation, visual or auditory hallucinations, manic behavior alternating with lethargy, hydrophobia secondary to painful contractions of pharyngeal muscles, polyneuritis; hyperactive deep tendon reflexes with positive Babinski signs and nuchal rigidity often are present.

Coma occurs 7 to 10 days after onset of acute neurologic syndrome. This stage is characterized by hydrophobia, prolonged apnea, and generalized flaccid paralysis similar to Guillain-Barré syndrome, seizures, coma, and ultimate respiratory and vascular collapse. Death may follow 2 to 3 days after onset of paralysis but may be delayed by life-support equipments. Recovery is rare.

Unfortunately, once the patient is symptomatic, use of antirabies vaccine or rabies immune globulin (RIG) does not improve prognosis, and treatment consists entirely of supportive care.

Animal Reservoirs

Domestic animals that transmit rabies (dogs, cats, cattle) account for only 10% of human exposures, whereas wild animals account for the other 90%, with skunks, foxes, raccoons, and bats being the most prominent. Dogs are the primary reservoir in undeveloped countries.

Prevention

Rabies cannot be treated. Therefore, efforts must focuse on prevention.

1. Rabies immunization for domestic animals
2. Education about avoiding contact with wild animals is an important public health issue on reducing risk of rabies exposure. Raccoons, skunks, foxes, and coyotes can be enjoyed from a distance, but they should not be attracted intentionally or adopted.

Pre-exposure Prophylaxis

Pre-exposure prophylactic immunization is recommended for people who are likely exposed to rabid animals. Veterinarians, animal handlers, and laboratory personnel should consider routine immunization. Also, people traveling to areas where dog rabies is endemic and who will not have easy access to medical care should consider immunization before traveling. A person who was previously immunized and who has had a potential rabies exposure should receive 2 intramuscular doses of vaccine. Give the first dose as soon as possible after exposure and the other 3 days later.

Post-exposure Treatment

Passive Vaccination---RIG is a solution of globulins dried from the plasma or serum of selected adult human donors who have been immunized with rabies vaccine and have developed high titers of rabies antibody

RIG is administered to previously unimmunized people so that passive antibodies are present until the person begins making ac-

Table 5. Indication for rabies immunization

Animal type	Evaluation and disposition of animal	Postexposure prophylaxis and recommendations
Dog, cat, ferret	Healthy and available for 10-day observation	Use prophylaxis only if animal develops signs of rabies
	Rabid or suspected of being rabid	Treat immediately with rabies vaccine and RIG
	Unknown or escaped	Consult public health officials
Bat, skunk, raccoon, fox, and most other carnivores, wood shuck	Regard as rabid unless geographic area is known to be rabies free or until animal is proved rabies free by laboratory tests	Treat immediately with rabies vaccine and RIG
Livestock, rodents, lagomorphs (rabbits and hares)	Consider individually	Consult public health officials

tive antibodies to the vaccine. RIG should not be given more than 7 days after initiation of the vaccine because RIG may decrease a person's own antibody response. RIG is given in a single dose of 20 IU/kg body weight. After the initial vaccine is given, the 4 subsequent doses should be given on days 3, 7, 14, and 28. The production of specific antibodies against rabies virus requires about 7 to 10 days to develop.

Active Vaccination---Three rabies vaccines are currently available in the United States: rabies vaccine adsorbed, human diploid cell rabies vaccine, and purified chick embryo cell vaccine; all 3 are inactivated virus vaccines.

Patients who are taking corticosteroids or are immunosuppressed may not develop active immunity with the vaccine. Antibody titers may need to be monitored

No evidence has shown that RIG or the rabies vaccine causes fetal abnormalities. Therefore, pregnancy is not a contraindication to appropriate rabies prophylaxis. RIG and the rabies vaccine have been used without problems in infants.[13, 14, 15] (Table 5)

Tetanus

The highest incidence rate of such disease in Taiwan took place in 1956 with the number of one thousand and four identified cases. Ever since the beginning of toxoid vaccination in 1972, the case number has gradually decreased to less than 100 cases. Since 1981, the number of annual reported Tetanus cases has been maintained under 20 cases per year, and the mortality rate has also decreased to single digit number (several cases).

Epidemiology

Approximately, 50% of cases of tetanus in the United States occur after injuries. Infected wounds (both traumatic and surgical) and abscesses, surgical wounds, parenteral drug abuse, major trauma, and animal-related injuries account for 25% of the tetanus-associated injuries. Because immunization is effective in preventing tetanus, the disease is most frequently noted in countries or in ethnic groups whose effective immunization is less likely to be accomplished.

Etiology and Pathophysiology

The tetanus bacillus is a Gram-positive, anaerobic rod that may develop a terminal spore, giving it a drumstick appearance. They can survive in soil for years and may be found in house dust, soil, salt, fresh water, and the feces of many animal species.

After into tissues, spores convert to vegetative forms, multiply, and elaborate tetanospasmin. Tetanospasmin enters the peripheral nerve at the site of entry and travels to the central nervous system (CNS) through the nerves or is transferred by the lymphocytes to the CNS. The toxin binds to gangliosides at the presynaptic nerve ending in the neuronal membrane, prevents release of neurotransmitters, and affects polarization of postsynaptic membranes in complex polysynaptic reflexes. The lack of inhibitory impulses that results is manifested in the characteristic spasms, seizures, and sympathetic overactivity of tetanus. The toxin has no effect on the mental status, so consciousness is not impaired directly by this illness.

The length and the course of the illness are determined by the location and amount of the bound toxin. The complete course of tetanus takes usually from 2 to 4 weeks and is in-

fluenced by patient age and the development of complications.

Clinical Manifestation

In addition to neonatal tetanus, tetanus can present in 1 of 3 clinical forms: localized, generalized, or cephalic. We just introduce generalized tetanus here:

Generalized tetanus

It is the most common form of clinical tetanus. It may occur after relatively minor injuries and often follows tetanus-prone wounds. The typical initial findings of trismus due to spasm of the parapharyngeal and masseter muscles are seen in 50% of cases.

Common complaints are pain, swallowing difficulty, and unilateral or bilateral stiffness of the neck and other muscle groups, such as those of the abdomen or thorax.[17] Persistent trismus accounts for the risus sardonicus that is considered a classic finding of tetanus. As the illness progress, additional muscle groups become involved. One of the most striking findings occurs with spasm of the paraspinal musculature, which may result in severe opisthotonos. All voluntary muscles can be affected and the disease may involve the larynx, which can be fatal. Minor stimuli including light, drafts, noises or voices, and light touch may trigger spasms.

The effect of tetanospasmin on the autonomic nervous system can induce cardiovascular instability. Labile hypertension and episodes of tachycardia or other tachyarrhythmias are common. The sympathetic overactivity or superinfections, such as pneumonia can cause fever. Spasms and cardiovascular complications occur most commonly during the first week and resolve slowly during the ensuing 2 to 4 weeks.

Complications

Complications of tetanus include direct toxic effect (laryngeal and phrenic nerves palsy, and cardiomyopathy), and spasm-related sequels (respiratory compromise, rhabdomyolysis, myositis ossificans circumscripta, and vertebral compressed fracture), respiratory compromise (hypoxic cerebral injury), and rhabdomyolysis (acute renal failure), as well as the psychologic impact.

Diagnosis and Differential Diagnosis

The classic presenting complaints in tetanus consist of muscle spasms, trismus, stiffness, pain with dysphagia and cranial nerve weakness. These can be seen in other conditions.

The lack of altered consciousness in tetanus is an important point of differentiation from CNS infections, and a parapharyngeal inflammation can be diagnosed by clinical examination and/or radiographs of the airway.

Specific diagnosis of tetanus by routine laboratory tests is difficult. Gram stains and anaerobic cultures of wounds reveal the characteristic Gram-positive bacilli with terminal spores in as many as one third of tetanus patients. Although a positive wound culture can support the clinical diagnosis, a positive culture in the absence of symptoms does not indicate that tetanus intoxication will develop.

Management and Prognosis

Appropriate treatment based on the clinical diagnosis is warranted even without specific confirmatory laboratory tests. The goals of therapy are eradicating C. tetani, neutralizing its toxin, and providing appropriate supportive care. Specific therapy includes intramuscular administra-

tion of tetanus immune globulin (TIG) to neutralize circulating toxin before it binds to neuronal cell membranes. Early administration of antitoxin may prevent spread of the toxin within the CNS. The recommended dosage of TIG ranges from 500 to 3000 U. Additionally, specific therapy includes antimicrobial agents for C. tetani such as penicillin G administered as 200,000 U/kg/d in 4 divided intravenous doses for 10 days. Alternatives for those allergic to penicillin include oral tetracycline (40 mg/kg/d; maximum of 2 g) or intravenous vancomycin (30–40 mg/kg/d).

Local wound care, including surgical debridement, is essential. Foreign bodies should be removed and wounds irrigated well and left open.

Patients should be managed in an intensive care setting of a tertiary-care center whenever possible. Facilities and equipments should be available including a quiet darkened room, suction equipment and oxygen, cardiac and respiratory monitors, a ventilator, and tracheostomy equipment. Neuromuscular blockade can be achieved with curariform drugs. The agents used most often are pancuronium and vecuronium.

The hypertension that results from sympathetic overactivity may require treatment. Beta-blocking agents are the most useful. Propranolol is administered most commonly.

Maintaining adequate nutrition and hydration is of outmost importance. Parenteral nutrition is usually required because of the likely length of the disease and the undesirability of oral or nasogastric feedings. Tracheostomy may be required to prevent laryngospasm.

Mortality

The worldwide mortality rate for generalized tetanus ranges from 45% to 55%. Although survivors generally do not experience neurologic sequelae, prolong convalescence with residual muscle rigidity is seen for several months.
The main predictors of prognosis are the rapidity of symptom onset and the rate of progression from trismus to severe spasms. Poor outcome is predicted by an interval between injury and trismus shorter than 7 days.

Prevention

Active immunization with tetanus toxoid is the most effective mean of protection. The primary series of tetanus toxoid, administered as DTP vaccine at 2, 4, and 6 months and a booster 12 months later, ensures protection in childhood. Additional boosters of tetanus toxoid should be given each decade throughout life.[18]

Rat-Bite Fever

Rat bite fever is a systemic illness classically characterized by abrupt onset of fever, rash, and arthralgias, which carries a mortality rate of 13% if left untreated

Epidemiology

In Asia the causative organism is usually Spirillum minus, a small, spiral gram-negative organism. Children account for more than 50% of rat bite fever cases. In the United States rat bite fever is caused primarily by transmission of Streptobacillus moniliformis, a pleomorphic gram-negative rod .
Rat bite fever can be divided into 3 clinical syndromes. 1. Rat bite fever caused by Streptobacillus moniliformis infection, which is the predominant form seen in the United States. 2. “Soduku” caused by Spirillum minus, seen pri-

marily in Asia. 3. When the clinical syndrome follows S moniliformis ingestion via contaminated food it is called "Haverhill fever. "

Clinical Manifestations

Rat bite fever is a systemic illness characterized by abrupt onset of fever and chills.

Spirillum Moniliformis

Fevers begin abruptly and usually resolve in 3 to 5 days but can relapse. The rash occurs in roughly 75% of patients and may be maculopapular, petechial, or purpuric. Hemorrhagic pustules or vesicles may also be seen. These usually appear 2 to 4 days after the fever resolves and may last up to 3 weeks. These often involve the extremities, especially the hands and feet. Approximately 20% of rashes due to S. moniliformis will desquamate. Other commonly associated symptoms include severe myalgias, headache, nausea, and vomiting. Within the first week, more than 50% of patients develop a non-suppurative polyarticular or migratory polyarthritis. The joint involvement may affect either large joints or the small joints of the hands and feet. Migratory arthritis may persist for years despite appropriate treatment.

Spirillum Minus

The incubation period averages 14 to 18 days, with a range of 1 to 36 days. If there is a bite, infection is usually heralded by an indurated lesion at the site as symptoms become obvious. The lesions may ulcerate and there may be regional lymphadenopathy. The fevers have regular relapses separated by afebrile periods lasting 3 to 7 days. Approximately 50% of patients develop a violaceous red-brown rash that usually consists of large macules with occasional erythematous plaques or urticarial-type lesions. The joint manifestations are rare. The mortality with S minus infection is slightly lower than that seen with S moniliformis and approaches 6.5%. If infection with either organism goes unrecognized, there can be serious sequelae, including arthritis, endocarditis, myocarditis, pericarditis, pericardial effusion, hepatitis, nephritis, and meningitis.

Diagnosis

Blood culture isolation is the gold standard.

Treatment

Penicillin is the treatment of choice despite rare reports of penicillin-resistant strains. Current recommended treatment is 5 to 7 days of intravenous penicillin at doses of 20,000 to 50,000 units/kg/day followed by 7 days of oral penicillin.[19]

Conclusion

As we protect wild-animal species and acknowledge their right to share territory, interactions—and possibly attacks—are likely to increase. Awareness, education, knowledge and prevention, rather than the elimination of animal populations, may be the best way to control wild-animal attacks on humans in the future.

References

1. 李培芬、林曜松。臺灣地區國家公園動物生態資料庫報告。內政部營建署，1994，台北
2. 李培芬、戴永禔、林曜松。臺灣野生動物資源之多媒體資料庫系統。臺灣動物資源資料庫建立研討會論文集(吳金洌、陳章波主編)，1992，台北。

3. 東森新聞報。花蓮發現40年來首例境外移入---狂犬病人體病例 2002/07/12 21:15
4. Lewis KT, Stiles M. Management of cat and dog bites. Am Fam Physician 1995; 52:479-85
5. Presutti RJ. Bite wounds early treatment and prophylaxis against infectious complications. Postgrad Med 1997;101: 243-52
6. Garcia VF. Animal bites and pasturella infections. Pediatr Rev 1997;18:127-30
7. Goldstein EJC, Citron DM, Finegold SM. Dog bite wounds and infection: a prospective clinical study. Ann Emerg Med 1980; 9:508-512
8. Brogan TV, Bratton SL, Dowd MD, Hegenbarth MA. Severe dog bites in children. Pediatrics 1995;96:947-950
9. Agahbabian RV, Conte JE. Mammalian bite wounds. Ann Emerg Med 1980;9: 79-82.
10. Goldstein EJC. Bite wounds and infection. Clin Infect Dis 1992;14:633-640
11. Smith PF. Treating mammalian biting wound, Journal of clinical Pharmacy and Therapeutics. 2000;25:85-99
12. Hankins DG. Rosekrans JA. Overview, prevention, and treatment of rabies Majo Clin Proceedings 2004;79: 671-76
13. Weir E. Putting the bite on rabies. CMAJ 2002;167:781
14. Jackson AC. Update on rabies. Curr Opin Neurol 2002;15:327-31
15. Kaeser HE, Sauer A. Tetanus toxin: a neuromuscular blocking agent. Nature 1969; 223:842
16. Einstein L. Tetanus. N Engl J Med. 1973; 289:293–96
17. Itzhak B. Tetanus in children. Ped Emerg Care 2004;20,48-51
18. Freels LK, Elliott SP. Rat bite fever: three case reports and a literature review. Clin Ped 2004;43:291-5
19. Norwood.S. Mechanisms and Patterns of Injuries Related to Large Animals. J Trauma 2000;48:740-744