

## Review Article

<https://doi.org/10.20546/ijcmas.2019.808.072>**Animal Bite: Rabies and Beyond Rabies; A Review**Nitasha Sambyal<sup>1\*</sup>, Ankush Proch<sup>2</sup>, Inderpal Singh<sup>3</sup> and Amit Challana<sup>4</sup>

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**ABSTRACT**

Increase in animal population has resulted in rise in animal bite cases and thus increase the potential for transmission of zoonotic diseases. That is why it is very important to consider some factors while considering the likelihood of disease transmission which includes species of animal, type of injury, severity of injury, vaccination status, interval between incident and treatment, and various attributes of the injured person (age, underlying health, etc.). It is also important to have knowledge about the natural oral flora of the animal. As mouth cavity of animals is rich in microflora, which sets infection when inoculated in skin of victim. Most frequently isolated bacteria in animal bite other than Rabies are *Pasturella* spp., *Streptococcus*, *Staphylococcus*, *Moraxella*, *Corynebacterium*, *Neisseria*, *Fusobacterium*, *Bacteroides*, *Posphuomonoa*, *Capnocytophagacanimorsus* and *Prevotella*. But there is lack of understanding and a very few data is available on natural oral flora of animal, mechanism of animal bite and pathogenic significance of isolated organisms from bite wounds. Even though the available literature in this field if present mostly focused on case report of rabies, neglecting other diseases which are evenly fatal if untreated. Thus, in the present study, data from various sources has been summarized to emphasis on various infection other than rabies which are transmitted via animal bite including human and mechanism of animal bite. So that the infection cause by bite wound can be efficiently diagnose by a physician for its proper management.

**Keywords**

Rabies, Animal bites, Dog, Microbiology of animal bites, Biting mechanism of animals

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**Introduction**

Animal bites continue to pose major public health challenges. The high risk group in animal bite cases comprises of veterinarians and animal owners, who should be more aware of bite risks and its consequences. Between 61%-68% of veterinarians suffer animal-related injury mainly dog and cat bites,

kicks, scratches etc. resulting in hospitalization and loss of work hours (Langley *et al.*, 1995; Gabel and Gerberich, 2002; Nienhaus *et al.*, 2005). Children are the next most vulnerable group to attacks by animals. Nearly 40% are below 15 years of age and 78.6% were males (Ichhpujani *et al.*, 2008). Children are typically bitten on the face, head and neck. Different locations for

animal bites are represented in Table 1. Injuries are complex in these body locations involving bony tissue injuries, intracranial complications and ophthalmologic lesions (Brogan *et al.*, 1995). As calvarium of children is thin therefore it gets crushed during dog bite cases (Iannelli and Lupi, 2005). The wound mostly results in fracture of skull as well as pneumothorax, stroke, spinal cord trans-section, and tracheal or oesophageal lacerations. Thus making animal bites an important community health problem requiring surveillance by public health agencies across the world.

Animal bite wounds get infected with pathogenic bacteria that include wide variety of fastidious aerobic bacteria and approximately 50-80% anaerobic bacteria. It has been scientifically proved that the bacteria isolated from bite wound reflect the oral flora of biting animal and the microbes of their ingested prey. Most of these bacteria cannot be isolated in labourites, because many of these species may phenotypically similar to each other and some grow slowly and may be missed unless plates are incubated for a long time (Goldstein *et al.*, 1978).

These wound get heal uneventfully and infectious complications are common. It is important that emergency physician should know about various diseases associated with animal bite and the tissue damage it caused. The severity of disease can be correlated with the biting mechanism. It has been reported that the inoculation of bacteria by cats are deeper than any other animals. If the fact is ignored than the anaerobic bacteria survives and condition becomes fatal.

India contributes the major share of animal bites as well as global deaths due to rabies. However, all these are preventable but for an effective prevention programme, it is important to understand the epidemiological behaviour of various animal bite diseases. As the data in this aspect is less thus, the present

study emphasis the mechanism of bite and transmission of bite related diseases. Even an attempt is made to find the bacteriological status of the bite wounds including human bite by reviewing the literature using computer assisted database, research references from cited articles and personal experiences.

## **Biting Status**

### **World**

In America it has been estimated that 1 out of 2 American's during his or her lifetime is bitten by an animal that accounts for 4.7 million bites per year and the bite wounds account for approximately 0.5 to 1% visits to hospital emergency rooms (Goldstein, 1992). In an Annual Report (2001) by City of Fort Worth Public Health Department, almost forty-percent (38.5%) of victims had high school education. More than 45% of bite victims were either the owner or acquainted with the owner. However, in most cases (73.7%), the owner was not present at the time of the incident. Even the report suggests that, the majority (78.6%) of bites were unprovoked. But in provoked cases, the situation was startling or awaking an animal (8.9%). It was further reported that 32% of biting animals were more than 6 years old.

The highest biting frequency was found on Saturdays, followed by Tuesdays and Fridays. And the month wise graph showed increase biting frequency during early spring which peaks in summer. Only 5% and 60% of all bite wounds are complicated by infection, among them 20% to 50% of cat bites, 10% to 50% of human bites, 3% to 20% of dog and 25% monkey (Edwards, 2004).

### **India**

In India 17.6 million annual animal bite cases are reported. Dog populations increased from 25.5 million in 1997 to 31.5 million in 2006,

mostly are ownerless or unimmunized against rabies (livestock census data 2006) and cause 2.28 million dog bite annually or 0.1 bite per dog. But according to NICD 2000 in South India there is annual incidence of 2.06 dog bites per 1000 of population. The household survey targeted 10 million population exposed to dog bites and rabies. It provides an estimate of 1.7 rabies cases per 100,000 and an estimate of 17,137 rabies cases in a one billion population, if paralytic form is 20% then the estimate increases to 20,565 or 2.06 cases per 100,000.

It was concluded that number of rabies cases per feral dog in India are in between 2 and 3 per 100,000 persons, or between 93 and 136 cases per 100,000 dogs (Sudharshan, 1994). The highest incidence of rabies was in 10–44 years of age (72%) (Sudharshan, 2005). But nothing can be said as data of rabies in animal are scanty, unreliable and controversial due to poor surveillance/reporting system. In U.S., the incidence of dog bite-related hospitalizations has been reported as 2.1/100,000 persons and 1.6 to 2.0/100,000 adults  $\geq$  20 years old. (Quinlan and Sacks, 1999)

Although a number of approaches are followed under evaluation to control rabies by managing dog population like dog movement restriction, reproduction and habitat control and dog removal. But one of such approaches, dog removal not only disrupts naturally established social organisation but also increases the contact rate, thus there is seen rise in population.

It was estimated that in India mortality due to rabies is 25,000 to 50,000 (Park, 1994), adults (64.7%) among which 71.1% were males and 87.6% were from poor/low income group. Over 95% are caused by dog bites, (Sudharshan, 1994; Park, 1994; Rehman, 1996; Sehgal, 1996) among them 39.5% are

by stray dogs and sites of bite were lower limb (56.2%), upper limb (20.9%) and hands (17%). 79% of rabies victims did not receive any anti-rabies vaccination and if taken then course is incomplete (Sudharshan, 1994)

According to the anti-rabies department, Government Medical College-Jammu (2010), it has been estimated that from April 2009 to March 2010 out of total 4357 animal bite cases recorded, the number of dog bite cases were 3703 (84.98%) followed by cat (2.24%), cow (3%) and 68 human bite cases.

### **Biting Mechanism**

Anatomy of the teeth and the strength of the jaws reflect in the nature of injuries caused by the biting animals which is directly proportional to its severity. On the basis of type of injuries caused by the biting animals, animal bites can be categorised into 3 types-avulsion (when skin is torn from underlying tissue and bone), laceration (most severe tear of underlying tissues with irregular edges) or puncture (teeth penetrating the skin and underlying structure) (Goldstein, 1992).

### **Canine**

Teeth of canines are large, broad and sharp. Jaws have powerful masseter-pterygoid complex muscles. This physical design of the mouth of the canine is associated with avulsion and laceration type of bites (Chambers and Payne, 1969). Therefore dog causes crushing injuries with direct structural damage to underlying vasculature, tendons, nerves, or joints (Benson *et al.*, 2006). Although all breeds of dog are capable of severe damage but the large brachiocephalic dogs have a greater biting force, compared with that of small dolichocephalic dogs. Breeds of dog which are more notorious for attack without provocation are pit bull breed, followed by Rottweilers, German Shepherd

Dogs and Chow Chows (Sacks *et al.*, 1996).

### Cat

The biting style of cat is different; it first bites and then holds their prey until they are motionless (Talan *et al.*, 1999). Because of their characteristic sharp, elongated, needle-like teeth (Bradley, 2002), the bite is typically deep puncture wound which pierce underlying structures, including ligaments, bone, joint capsule, tendon sheath, neurons, and vascular structures (Myers, 2003).

### Other animals

Bite by mice, rats and snakes tend to cause puncture wounds.

### Human

The bite resembles dog bite with abrasions and laceration being more common than punctures.

High risk animals are Bats, Raccoons, Skunks, Foxes, Bears, Opossums, Weasels, Wolves and Woodchucks. Whereas animals which pose less risk to human are Immunized dogs, cats, and ferrets, besides Squirrels, Hamsters, Rats, Mice, Rabbits, Hamsters, Chipmunks, Guinea pigs, Gerbils etc. pose little risk.

Among the various types of animal bite wound, the highest incidence of infection is seen in puncture wound as they get quickly sealed. Thus produce an environment for the growth of anaerobes. Wounds punctured by cats inoculate the microbes deep into the tissues which remain unnoticed and cause infection.

### Microbiology

Mammalian mouth contains approximately 100 million microbes per ml of saliva

belonging to over 200 species of facultative microbes and obligate anaerobes. Some recognized microbes are mentioned in Table 2.

### Dog and cat

Most frequently isolated aerobic bacteria are *Pasturella* spp. (50% *Pasturella canis* in dog bites and 75% *Pasturella multocida* subspecies *multocida* and *septic* in cat bites), next most commonly identified species are *Streptococcus* (46% cases), *Staphylococcus* (46% of dog bites and 35% of cat bites), *Moraxella*, *Corynebacterium*, and *Neisseria*. The most commonly identified anaerobes are species of *Fusobacterium*, *Bacteroides*, *Posphuomonoa*, *Capnocytophaga canimorsus* and *Prevotella* (Talan *et al.*, 1999).

### Human bites

They transfer more bacteria to his victim as compared to dog or cat bites as human mouth has higher population of resident bacteria (Smith *et al.*, 2000). The microbial difference of human bite in compare to dog and cat bite is that in human bite there is presence of *Eikenella corrodens*, the absence of *P. multocida*, and a higher incidence of  $\beta$ -lactamase-producing organisms. Rare cases of *Streptococcus pyogenes* necrotising fasciitis after human bites have been reported by Wienert *et al.*, (1999).

### Alligator

Its bite wound most frequently consist of *Aeromonas hydrophila* fallowed by *Enterobacter agglomerans*, *Citrobacter diversus*, *Proteus* spp., *Pseudomonas* spp., *Serratia* spp., and *Clostridium* spp. (Greenberg, 1999).

### Snakes

Wound caused by snakes have high rate of

infection by enteric gram-negative bacilli. The reason may be, the way snake eats the prey, the head of prey is swallowed first and the prey's fecal flora subsequently colonizes in the snake's mouth. Even though the venom of snake is sterile and after the bite causes extensive necrosis of the tissue. Thus providing an optimal environment for growth of inoculated organisms such as *Pseudomonas aeruginosa*, *Proteus* spp., *Salmonella arizonae*, *Aeromonas* spp., *Vibrio vulnificus*, *Bacteroides fragilis* and *Clostridium* spp. Gram-positive skin flora are also frequently isolated that include staphylococci (Hsieh and Babl, 1999).

### **Horse**

The bite infection by horses mostly caused by *S. anginosus* and *S. mutans* producing extensive gas in the tissues. Other isolated organisms are *Actinobacillus lignieresii*, *S. aureus*, *Neisseria* spp., *E. coli*, *Pasteurella* spp., *Bacteroides ureolyticus*, *B. fragilis*, *Prevotella melaninogenica*, *P. heparinolytica* (Goldstein, 1994; Abrahamian *et al.*, 2004).

### **Shark**

Their bite mostly contains *vibrio* spp. It also includes *A. hydrophila* and *S. aureus*.

### **Other marine animal**

The bite wounds are infected with *A. hydrophila*, *B. fragilis*, *Chromobacterium violaceum*, *Clostridium perfringes*, *E. rhusiopathiae*, *E. coli*, *Mycobacterium marinum*, *P. aeruginosa*, *Salmonella enteritidis*, *S. aureus*, streptococci, *Vibrio vulnificus*, *Vibrio carchariae*, *Vibrio damsela*, *Vibrio parahaemolyticus*, and *Peptostreptococcus* spp. (Brook, 2003; Murphey *et al.*, 1992). Infection caused by animal bites develops at the site of the wound. Microbes recovered from the adjacent tissue

of bite wounds suggests that the infection is transmitted from the oral flora of the biting animal (Iannelli and Lupi, 2005).

Although the severity of infection at the site of animal bite is not only depends upon the oral flora of the person or animal inoculating the wound. But it also depends upon the anatomic location and structures damaged by the animal bite, microbial contamination of the wound from the environment or skin, and the time elapsed since the injury.

### **Some important pathogens**

Although rabies is an important animal bite disease but there are many other diseases which must be considered as they are evenly fatal if untreated. An attempt is made to described some of these diseases.

### **Rabies**

It is the fatal neurological infection primarily of wild animals but also found in domestic animals from where it spread to humans by direct contact of saliva of rabid animal to mucous membrane, wounds or via bite. Even spread by inhalation of aerosol originating from bat caves.

It is caused by neurotropic, genus *Lyssa* virus. This genus includes the classic rabies virus, two European bat lyssa viruses, an Australian bat lyssa virus, and the African Duvenhage virus. They all produce a similar fatal encephalomyelitis in humans, known as rabies.

In 90% of the cases it is transmitted to human by the close association of domestic animals (Chhabra and Ichhpujani, 2003). There is no human to human transmission, with the exception of eight cases resulting from corneal graft implants and three from solid organ transplantation. There was one suspected case

where virus transmits via mother to a breastfed infant (Dutta, 1998). Transplacental infection occurs in animals but has not been reported in humans. It is endemic in many parts of the world. India reports around 30000 deaths due to rabies (Chomel, 1993) among them 30-50% were children reported by Rotivel *et al.*, (1998). But in the latest 2011 survey death toll reduces to 233 times due to public awareness and vaccination of the street dogs. Although in U.K. 1920 it was reported by pounder (2003) that rabies was eradicated but still in past 20 years 12 people died due to this.

Rabies virus cannot penetrate the intact skin and can be inactivated by ultraviolet rays and does not persist in environment. Therefore coming in contact with the environment around a rabid animal such as bedding or water bowls does not present a risk. It is not a true zoonosis as some infected animal die from infection (Sikes, 1962). In a survey done by Eng and Fishbein (1990), stated that rabid dogs die within 8 days of becoming infected.

### **Clinical manifestation**

Symptoms in the infected animal are characterized by increased aggression, ataxia, lethargy, or excess salivation; nocturnal animals may become active during the daytime. After bite the virus multiplies in the cells at the site of bite subsequently ascends centripetally to the CNS via peripheral nerves. In the neurons of brain it replicates and then transmitted centrifugally and deposit in body tissue including salivary glands via efferent nerves. Incubation period varies with the site of bite i.e. between 30-90 days even upto 7 years (may be 14 years exceptionally). It is shortest when bite is on head and neck (around 15 days) but prolonged when the bite is on extremities. First symptom is itching, pain or paraesthesia at the site of healed wound. Patients often complained of

encephalopathic syndrome that can be of two form- furious or paralytical form. Furious rabies is manifested by irritability, agitation, hyperaesthesia and autonomic disturbances (disorders of blood pressure, hypersalivation, and sweating). Hydrophobia is the pathognomonic symptom which is spasm of inspiratory muscle, painful laryngospasm and fear of swallowing. Initially it is while drinking water but later even on slightest stimulus or mention of water and end in aerophobia i.e. extension of the back and arms followed by generalised convulsion or cardiorespiratory arrest.

In untreated cases hydrophobic spasm is preceded by death or a generalized flaccid paralysis and death in a week. Paralytic form is seen in 20% of human cases and occurs after bites of vampire bats (Delpietro and Russo, 1996) or bite of vaccinated individual.

A flaccid paralysis develops with pain and fasciculation of the affected muscles, paraplegia and sphincter disturbances followed by paralysis of the respiratory and deglutive muscles. Hydrophobia is rare but spasms of the laryngeal muscles may occur in the terminal phase. Without treatment patients may survive for up to 30 days.

It can be misdiagnose with tetanus, as a wound on head and neck can cause cephalic tetanus causing trismus (facial stiffness and pharyngeal and laryngeal spasms) followed by asphyxia or aspiration. But can be distinguish as its incubation period is short (15 days), persistent muscle spasms and absence of meningoencephalitis.

According to the Association for Prevention and Control of Rabies in India (APCRI), India has the world's highest number of dog-bite cases resulting in 80 per cent of the world's rabies cases. They have even reported that, mainland of India is endemic and enzootic to

rabies and the islands of Andaman & Nicobar and Lakshadweep are rabies free and to ensure a Rabies Free India by 2020 a coordinated National Rabies Elimination Programme was launched (Sudarshan, 2005).

### ***Bergeyella zoohelcum***

It is a zoonotic pathogen, indigenous to dog and cat. It is reported in 38-90% nasal, gingival scrapings and oral fluid of dog (Decostere *et al.*, 2002; Reina and Borrell, 1992). It is transmitted to human after a dog or cat bite, but Kivinen *et al.*, (2003) also found its transmission via close contact with cats. It was referred by Centers for Disease Control and Prevention (CDC) as group IIj organism. In Holmes *et al.*, (1986) proposed its name as *Weeksella zoohelcum* but in 1994 Vandamme *et al.*, (1994) proposed a new genus, *Bergeyella zoohelcum*.

### **Morphology**

*B. zoohelcum* is a non-fermentative, Gram-negative, rod-shaped, non-spore-forming, non-motile aerobic bacterium that typically grows well on blood agar with characteristic colonies as circular, shiny, and smooth with entire edges (Holt *et al.*, 1994) but no growth on MacConkey's agar. *B. zoohelcum* is oxidase, catalase, and indole positive. At present, *B. zoohelcum* is the only representative of the genus *Bergeyella*.

Botha *et al.*, (1998) linked food as source of *Weeksella* and *Bergeyella* like bacteria. But on the other hand *W. virosa*, is a female urogenital pathogen and has been reported in healthy women (2%) and up to 15% in women with a high rate of sexual activity (Mardy and Holmes, 1988).

### **Clinical manifestation**

After exposure to dog bite it causes pneumonia, meningitis (Bracis *et al.*, 1979;

Grimault *et al.*, 1996), septicemia, leg abscess and tenosynovitis (Isotalo *et al.*, 2000; Reina and Borrell, 1992). Although it is an opportunistic pathogen with low virulence but to avoid complication of endocarditis the infection should be promptly treated (Kivinen *et al.*, 2003).

### **Treatment**

Goldstein *et al.*, (1999) reported that *W. zoohelcum* (*Bergeyella*) isolates are sensitive to gatifloxacin at 0.016g/ml and to the linezolid (2 g/ml)

*B. zoohelcum* has host diversity, Kroes *et al.*, (1999) reported it as commensal of human oral flora. Woo *et al.*, (2003) suggested that there could be misidentification of *B. zoohelcum* as *Riemerella anatipestifer* if the MicroSec 500 16S rDNA-based identification system (which uses the first 527 bases of the 16S rRNA gene) is used. Even the ecological habitat (regardless of the host) of *B. zoohelcum* is related to the oral environment. Thus in the past, there is possibility that fastidious strains of *B. zoohelcum* from animal bite wounds was not reported.

### ***Capnocytophaga***

It is from family flavobacteriaceae and is in the Flavobacterium– Cytophagar RNA homology group as described by Mandell *et al.*, (2005) and Vandamme *et al.*, (1996). Formerly *Capnocytophaga canimorsus* was originally isolated from the human oral cavity and described in 1956 by De Cadore *et al.*, (1956) it was isolated from the blood and spinal fluid of dog bitten patient and was known as dysgonic fermentor-2 or DF-2 (Bobo and Newton, 1976; Brenner *et al.*, 1989). But its current name *Capnocytophaga* (means “eater of Carbon dioxide”) *canimorsus* (means “dog bite”) was given in 1989 by Brenner *et al.*, (1989).

## **Morphology**

It is a fastidious, slow growing, an anaerobic, non-spore forming, Gram-negative, spindle shaped bacteria. It is catalase, oxidase, and indole negative. Many strains of the bacterium produce a yellow-brown pigment.

Being fastidious, other rapidly growing bacteria retard its growth, so colonies become apparent only after prolonged incubation of 48 to 72 h (Holt *et al.*, 1979; Leadbetter *et al.*, 1979; Socransky *et al.*, 1979). Although the organism grows slowly in blood cultures (6 days), a rapid interim diagnosis can be made by Gram staining the buffy-coat preparations (Abrahamian *et al.*, 2004).

It has been isolated from the oral cavity of both human and canine species and is the important etiology of localized juvenile periodontitis (Newman *et al.*, 1976). Valtonen *et al.*, (1995) have also reported few cases of *C. canimorsus* from cat bite infection.

## **Clinical manifestation**

Small proportion of untreated dog bite victims develops septicemia, subsequent multi-organ failure and ultimately death. But Brenner *et al.*, (1989) and Winkler (1977) suggested that the predisposing factors for severe infection are splenectomy, alcoholism, liver disease, corticosteroid therapy, lymphoma or leukaemia patient, chronic lung diseases and immune-compromised patient who sustain a dog/cat bite. They are the high risk groups which need more attention.

Symptoms after bite appear within 1 to 8 days (Hore, 2001). The patient becomes lethargic, tiredness, listlessness and subsequent fevers, rigors and fulminant sepsis with disseminated intravascular coagulation (DIC) with septicemia (34 to 36% of cases), meningitis, septic arthritis, endocarditis, renal failure,

acral gangrene (15% of cases), disseminated purpura (37% cases) with erythema and rare ocular infection (Brenner *et al.*, 1989; Lion *et al.*, 1996).

Even necrosis of the extremities followed by multi-system organ failure is also seen (Hore, 2001). Most of the patients also suffer from mucosal ulcerations and gingival bleeding.

It is not always important that infection occurred via bite; even the lick of dog at superficial burn can cause entry of the bacterium. Patients commonly associated with gastrointestinal complains including abdominal pain (26%), vomiting (31%), and diarrhea (26%) (Pers *et al.*, 1996).

Fischer *et al.*, (1995) reported that, although the bacterium is present in all dog wounds but still infection is rare. It is due to the fact that it is a fastidious bacterium with low virulence which is even susceptible to post bite prophylaxis antibiotic. Therefore most of the infection occurred in immune-compromised patients.

In a review, Lion *et al.*, (1996) listed that 33% of systemic infections occurred in asplenic patients, 24% in alcoholics, and 5% in immuno-compromised patients and 41% of infections in patients without any known risk factor. During systematic infection there is high mortality about 20%.

Keeping in view the risk group, it is important to inform the physicians about the history of splenectomy or other immune-compromising conditions of the patient after a dog bite as they are the predisposing conditions for *C. canimorsus* infection.

## **Treatment**

*C. canimorsus* infection can be treated by penicillins G (Jolivet-Gougeon *et al.*, 2000),

imipenem, erythromycin, vancomycin, clindamycin, third-generation cephalosporins, chloramphenicol, rifampicin, ofloxacin, ciprofloxacin, doxycycline and quinolones (Hore, 2001). But it is typically resistant to aztreonam, trimethoprim–sulfamethoxazole and aminoglycosides (Hore, 2001).

### ***Pasteurella species***

The bacterium has been reported by Woolfrey *et al.*, (1985) from the fang flora of 90% of large feline population. Human infection occurs after bite, scratch or direct contact with saliva of infected animals, more important in immuno-compromised human. *Pasteurella multocida* subspecies *multocida* and *septica* were isolated from systemic infections by O'Neill *et al.*, (2005), caused by a bite or licking of wounds by dogs and cats.

### **Morphology**

It is pleomorphic Gram negative, non-spore forming, cocobacillus. It is sugar fermentative, non-motile, facultative anaerobic bacteria. It shows Bipolar staining by the Giemsa, Wright or Romanowsky staining methods. It gives catalase, oxidase, nitrate reduction and indole positive and urease negative test. Selective colonies can be kept in BBL-soy-trypticase, Difco-tryptose-starch-Gibco-dextrosin. Colonies on Brain Heart Infusion (BHI) agar and Nutrient agar shows Mucoid, sticky convex colonies of 2-3 mm diameter. On blood agar colonies are translucent grayish or yellowish green without haemolysis and no growth on MacConkey agar.

### **Clinical manifestations**

Incubation period for infection is 24 hours with the appearance of erythema, swelling, tenderness and drainage at the site of the bite. Infection remains localized to the inoculated wound with cellulitis and abscesses, but direct

extension to surrounding tissues may occur resulting in lymphangitis, regional lymphadenopathy followed by bacteremia with metastatic foci causing osteomyelitis, arthritis, cirrhosis, diabetes, hematologic malignancies, neoplasms and immunosuppression (Smith *et al.*, 2000; Weber, 1984; Morris and McAllister, 1992).

Licking of face and ear by cat and dog is associated with rheumatoid tenosynovitis, sepsis and meningitis, while licking of ulcers of the owners legs can result in brain abscess, pneumonia and endocarditis (Weber, 1984). Infection via aerosols route causes tonsillitis, sinusitis, epiglottitis, under-mandible cellulitis (Ludwig's angina).

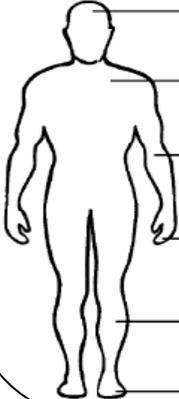
Early diagnosis is important especially for young and elderly people as well as in immuno-suppressed patients including the pregnant woman (Jordan *et al.*, 2006; Kaufman *et al.*, 2005). Although serological tests are not used frequently for diagnosis but still recent exposure can be indicated by higher titration (from 1/160 up to 1/1280 or higher ones) obtained through hemagglutination reaction.

### **Treatment**

Drug of choice is penicillin and ampicillin. Cefuroxime and cefpodoxime have better in-vitro activity than other cephalosporins. Nafcillin, dicloxacillin, cefazolin, cefaclor, cefadroxil, and cephalexin followed by Clindamycin and the aminoglycosides are resistant to *Pasteurella species* (Goldstein *et al.*, 1988).

Antibiotics treatment is used in patients undergoing steroids treatment or in patients with lesions on the face and hands, immuno-compromised, diabetic etc.

(Source for Table 1: City of Fort Worth Public Health Department, Animal bite prevention program: Annual Report-2001)



Location	Dog bite	Cat bite
Face, scalp, neck	16%	2%
Trunk	2%	0%
Shoulder, arm, forearm	12%	23%
Hand	50%	63%
Thigh, leg	16%	9%
Feet	4%	3%

Table.2 Some recognised microbes detected in various biting species

S. no.	Biting Species	Biting frequency	Biting force	Microbes
1.	Dog	80-90% (82.4%)	320 lbs (wolf-400 lbs), mastiff-500 lbs	<i>Pasturellacanis</i> , <i>Streptococcus</i> , <i>Staphylococcus</i> , <i>Moraxella</i> , <i>Corynebacterium</i> , <i>Neisseria</i> , <i>Fusobacterium</i> , <i>Bacteroides</i> , <i>Posphuomonoa</i> , <i>Capnocytophagacanimorsus</i> and <i>Prevotella</i> , <i>Leptospira</i> spp, <i>Bergeyellazoohelcum</i> , <i>CDC group EF-4b</i>
2.	Cat	5-15% (16.2%)	20 lbs	<i>Pasturellamultocida</i> subspecies <i>multocida</i> and <i>septica</i> , <i>Streptococcus</i> , <i>Staphylococcus</i> , <i>Moraxella</i> , <i>Corynebacterium</i> , <i>Neisseria</i> , <i>Fusobacterium</i> , <i>Bacteroides</i> , <i>Posphuomonoa</i> , <i>Capnocytophagacanimorsus</i> and <i>Prevotella</i> , <i>Bartonella henselae</i> , <i>Tularensis</i> , <i>Comamonas</i>
3.	Human	1%	120 lbs	<i>Eikenella corrodens</i> , <i>Streptococcus pyogenes</i> , <i>Porphyromonas</i> , hepatitis B
4.	Alligator	Rare	2500 lbs	<i>Aeromonas hydrophila</i> , <i>Enterobacter agglomerans</i> , <i>Citrobacter diversus</i> , <i>Proteus</i> spp., <i>Pseudomonas</i> spp., <i>Serratia</i> spp., and <i>Clostridium</i> spp.
5.	Snakes		Phyton: 200lbs Anaconda: 600 lbs	<i>Pseudomonas aeruginosa</i> , <i>Proteus</i> spp, <i>Salmonella arizonae</i> , <i>Aeromonas</i> spp., <i>Vibrio vulnificus</i> , <i>Bacteroides fragilis</i> and <i>Clostridium</i> spp.
6.	Horses			<i>S. anginosus</i> , <i>S. mutans</i> , <i>Actinobacilluslignieresii</i> , <i>S. aureus</i> , <i>Neisseria</i> spp., <i>E. coli</i> , <i>Pasteurella</i> spp., <i>Bacteroides ureolyticus</i> , <i>B. fragilis</i> , <i>Prevotellamelaninogenica</i> , <i>P. heparinolytica</i> .
7.	Sharks		600 lbs	<i>Vibrio</i> spp., <i>A. hydrophila</i> and <i>S. aureus</i> .
8.	Rat	2%	360 lbs	<i>Streptobacillus moniliformis</i> , <i>Spirillum minus</i>
9.	Other marine animals			<i>A. hydrophila</i> , <i>B. fragilis</i> , <i>Chromobacteriumviolaceum</i> , <i>Clostridium perfringes</i> , <i>E. rhusiopathiae</i> , <i>E. coli</i> , <i>Mycobacterium marinum</i> , <i>P. aeruginosa</i> , <i>Salmonella enteritidis</i> , <i>S. aureus</i> , <i>streptococci</i> , <i>V. vulnificus</i> , <i>Vibrio carchariae</i> , <i>Vibrio damsela</i> , <i>Vibrio parahaemolyticus</i> , and <i>Peptostreptococcus</i> spp.

Biting pressure of lion is 600 lbs which is less in comparison to biting pressure of hyenas (1000 lbs) and turtle (1000 lbs)  
Source- National geography Dr. Brady Barr's Bite Pressure Tests (Feb-2008)

## **Cat scratch fever**

It is also known as benign inoculation lymphoreticulosis, cat-scratch Syndrome, Cat adenitis, Cat-claw fever. It is mild to severe disease that affect human, although cat carries the bacteria but do not get sick. It only transmits the infection via bite or scratch. It is thought that cat flea (*Ctenocephalides felis*) can also transmit the bacteria to cats and can act as possible vectors in transmitting the bacteria directly to humans.

## **Etiology**

It is caused by *Bartonella henselae* bacterium (after A.L. Barton, a Peruvian physician who first described the germs). Earlier it was thought to be by *Afipia felis* and then a rickettsia *Rochalimaeahenselae*. It is a small, Gram-negative, pleomorphic with arrangement in the forms of Y-shape, chains or clumps, intracellular bacilli located in capillary walls, near follicular hyperplasia and inside micro-abscesses with Warthin-Starry silver stain.

## **Clinical manifestation**

The disease is seasonal in temperate climates, with most cases occurring during winter. Whereas no seasonal differences seen during hot climates. Approximately 75% of the cases occurred in children. Incubation period is between 7 to 20 days which is characterized by a papule or a cluster of papules at the site of inoculation, followed by regional lymphadenopathy without lymphangitis within 3-30 days. Primary lesions are seen at the point of inoculation i.e. partially healed ulcers surrounded by an erythematous area or erythematous papules, pustules, or vesicles. Lymphadenitis is generally unilateral appearing in the epitrochlear, axillary or cervical lymph nodes or in the femoral and inguinal lymph glands. Swelling in the lymph

glands is painful with suppurations which persist for few weeks to few months. It is also associated with pneumonia, pharyngitis, thyroiditis, encephalitis, hepatomegaly, splenomegaly, optic neuritis, cerebral arteritis and endocarditis. During systemic infection, fever is short-lived followed by chills, anorexia, malaise, generalized pain, vomiting, and stomach cramps. In rare occasion morbilliform cutaneous eruptions occur.

Although there is no sequel of the disease but in few cases complication do arise like Parinaud's oculoglandular syndrome, encephalitis, osteolytic lesions, and thrombocytopenic purpura. In Parinaud's oculoglandular syndrome, the route of entry is via conjunctiva or eyelids. It occurs when a person rubs his eyes after picking up infected cat. The conjunctival lesion heals in 1-3 weeks without causing damage to eye (August, 1988).

## **Treatment**

Gentamicin and doxycycline or oral administration of erythromycin proven successfully. It is recommended that immune-suppressed patients should avoid contact with kittens and young cats, if they do get bitten or scratched, they should seek medical attention as soon as possible.

## **Rat bite fever**

It is a rare systemic infection caused by *Streptobacillus moniliformis* present normally in the nasopharyngeal flora of rodents, gerbils, mice and other rodents and *Spirillum minus* (In Asia, known as sodoku) present in infected eyes of rats. In North America *Streptobacillus moniliformis* is also known as *Streptothrix murisratti*, *Nocardia muris*, *Actinomyces muris*, *Actinobacillus muris*, *Proactinomyces muris*, *Haverhillia multiformis* and *Asterococcus muris*.

## Morphology

*Streptobacillus moniliformis* is a non-motile, pleomorphic, microaerophilic bacillus commonly found in the oropharynx of wild rats. *Spirillum minus* is a small, thick, Gram-negative, motile spirochete with bipolar flagellar tufts (two or three turns).

The bacteria is spread through rat bite (10% cases), scratch, ingestion of food or water contaminated with rat feces and urine (known as Haverhill fever when organism is *Streptobacillus moniliformis*), ingestion of unpasteurized *S. moniliformis* contaminated milk or via handling rats (pet rats owners, laboratories) and also by aerosol. Cases have been reported that the infection occurred by bite of cats, dogs, ferrets, and weasels (may be they are originally infected by rodents).

## Clinical manifestation

Infection caused by *Streptobacillus moniliformis* is known as streptobacillary. Although incubation period is 3 to 10 days but before symptoms appear, bite wounds get healed without inflammation. Symptoms include fever, vomiting, headache, muscle pain, joint pain and rash. After 2 to 4 days followed by morbilliform petechial rash on extremities i.e. hands and feet (in 75% of patients) but in 50% of cases it is followed by migratory polyarthralgia or arthritis affecting the knees, shoulders, elbows, wrists, or hands which persist for years. Other serious complications including tenosynovitis, endocarditis, pericarditis, myocarditis, hepatitis, nephritis, meningitis, pneumonia, sepsis, and focal organ abscesses can also occur. Diarrhea is seen in infants and children causing weight loss.

Haverhill fever is associated with severe nausea, vomiting, and pharyngitis. Complications include endocarditis, abscesses

in the brain, tenosynovitis, pericarditis, myocarditis, hepatitis, nephritis, meningitis, pneumonia, sepsis, and focal organ abscesses. The mortality rate is 7% to 13% and 53% in endocarditis.

Spirillary is the infection caused by *Spirillum minus* with incubation period of 21 days. It is characterized by a relapsing fever, ulceration or eschar at the site of the bite wound, lymphadenopathy and lymphangitis. A roseolar-urticarial rash develops in 75% cases. Fever reappears in 2 to 4 cycles for four to eight weeks. Complication includes arthritis, endocarditis and nephritis. It rarely occurs in U.S., but is present in patient who had travelled to Asia.

Although rats are asymptomatic to *S. moniliformis* and *S. minus* infection, but can suffer from otitis media and can be present in subcutaneous abscesses as secondary invader. In mice infection develops polyarthritis, subcutaneous or hepatic abscesses, Conjunctivitis, photophobia, cyanosis, diarrhea, anemia, hemoglobinuria. Brown crusts can occur over the mammae of nursing females. Chronic arthritis, with swelling of the limbs or tail, may be a sequela. Deformation, ankylosis, or posterior paralysis, kyphosis, and priapism occur. Abortions and stillbirths have also been reported.

Guinea pig: *S. moniliformis* is associated with granulomatous pneumonia or cervical lymphangitis. Non-human primates described Septic arthritis and endocarditis. Turkeys and a tawny owl: Arthritis with infected feet was reported in the U.K.

## Treatment

Drug of choice is Penicillin given @ (400,000- 600,000 units IM bid), or penicillin G intravenously for seven to ten days. For uncomplicated cases, oral penicillin or

tetracycline may be effective therapy. Antibiotic for *S. moniliformis* include Cephalosporins and Vancomycin. Erythromycin has variable activity against *S. moniliformis*.

### **Porphyromonas**

It is present in the gingival pocket of human and animals (Dzink *et al.*, 1988) and thus can be isolated from bite wounds of 28% victims (Citron *et al.*, 1996). They were previously classified in the genus *Bacteroides* (Shah and Collins, 1988). The results indicate that vitamin K1 and hemin are important factors for the growth of most strains of *Porphyromonas* spp. Previous studies have indicated that these supplements are important for the growth of other anaerobes, including *P. melaninogenica* (Gibbons and MacDonald, 1960; Summanen *et al.*, 1993).

### **Morphology**

Three species *Porphyromonas gingivalis*, *Porphyromonas asaccharolytica* and *Porphyromonas endodontalis* are found in human (Shah and Collins, 1988). Periodontitis of animal consist of *Porphyromonas cangingivalis* and *Porphyromonas cansulci* (Collins *et al.*, 1994), *Porphyromonas gingivicanis* and *Porphyromonas crevioricanis* (Hirasawa and Takada, 1994), *Porphyromonas canoris* (Love *et al.*, 1994), *Porphyromonas gulae* sp. Nov (Allaker *et al.*, 1997a, b) and *P. gingivalis*.

It is strictly anaerobic, asaccharolytic or weakly saccharolytic, gram-negative, non-spore-forming and non-motile rod-shaped chemo-organotrophs.

On blood agar colonies shows brown to black pigments. All produce virulence factors such as collagenase, protease, and cytotoxin (Grenier and Mayrand, 1987).

### **Less commonly encountered microbes**

The bacteria present in animal bite are CDC group EF-4b (from dog and large feline bite) or *Comamonas* spp from large feline bite wounds (Griego *et al.*, 1995; Aghababian and Conte Jr., 1980; Goldstein, 1992).

CDC group EF-4b classified as eugonic fermenter group 4 (EF-4), is a non-fermentative, Gram negative coccoid to short rod bacteria (Schreckenberger and von Graevenitz, 1999). *Comamonas* spp are ubiquitous in environmental. It is gram negative bacilli that have generally been considered as non-pathogenic (Gilligan and Whittier, 1999).

In today world due to globalization and increasing trend of keeping exotic pets as companion to human, the chances of transmission of zoonotic diseases have increased. But major problem faced by public health workers is beyond this. There is tremendous rise in the population of elderly people and immune-compromised persons, who are at higher risk of getting infected. As such clinicians must be aware of potential of animal bite origin emerging microbial pathogens and its treatment. Thus more complete understanding of the circumstances of bites is needed in order to prevent and avoid infection in high risk groups.

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