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CAT BITE TRANSMISSION OF *YERSINIA PESTIS* INFECTION TO MAN

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SUMMARY

The transmission of bubonic plague from the kitten of a domestic cat to a man by means of a bite on a finger is described. The human case was complicated by the development of a secondary meningitis, followed, after specific therapy, by protracted recovery. The kitten showed swollen lymph nodes of the head and neck, frothing at the mouth and nostrils, and signs of an acute infectious disease which had a fatal termination. *Yersinia pestis* was isolated on about the 8th day from the cerebrospinal fluid of the man. The foster mother of the kitten exhibited signs of spinal and cerebral meningitis but recovered following treatment; her serum contained plague antibody levels of 1:512 and 1:1024 on the 22nd and 34th days respectively after the first evidence of illness. Three litter mates of the kitten also died. The outbreak occurred on a farm in the Graaff-Reinet district of the eastern Cape Province, which is situated about 160 km from the nearest known natural plague focus.

INTRODUCTION

Plague is a specific infectious disease caused by the plague bacillus, *Yersinia pestis* (formerly known as *Pasteurella pestis*). The illness in man takes the form of an acute fever which may be rapidly fatal before characteristic features appear or may cause swelling and suppuration of regional lymph nodes. These typical swellings usually result from infected flea bites and are called buboes, hence the common form of the disease is referred to as bubonic plague[9]. Transmission of bubonic plague to man by means other than the bites of infected fleas is less common. A small proportion of cases of bubonic plague may be traced to entry of the bacilli through abraded skin of the feet from infected floors, or of the hands in the performance of an autopsy, or in handling or skinning infected animals. Primary septicaemic plague may

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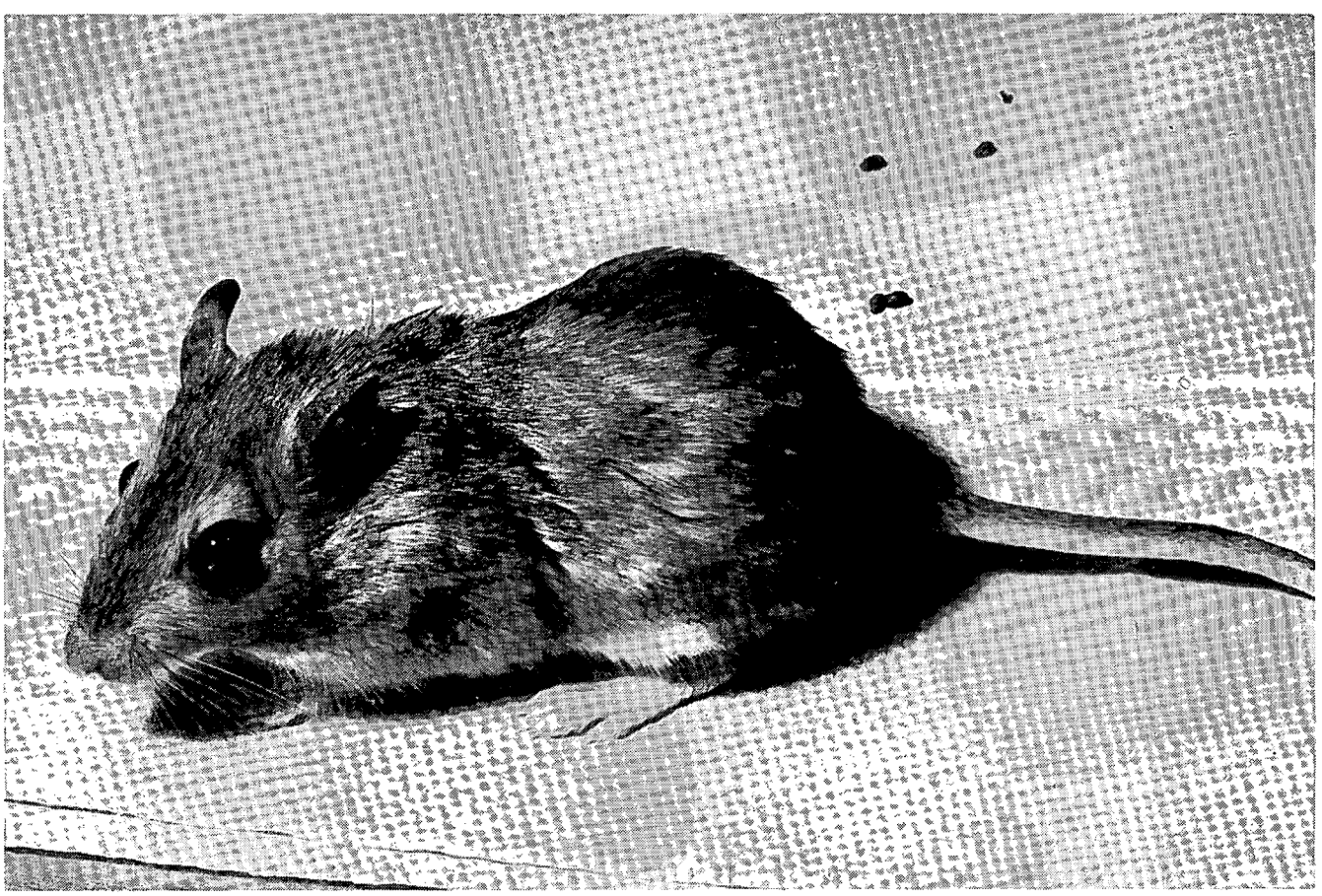

Fig. 1: The gerbil, *Tatera brantsii*, one of the small wild-living rodents responsible for the maintenance of natural plague foci in parts of South Africa.

Fig. 2: The gerbil, *Desmodillus auricularis* also plays an important role in the formation of permanent wild rodent reservoirs of infection in this country.

also result from the entry of the organisms through mucous membranes, especially those of the mouth, throat and conjunctiva[4]. Primary pneumonic plague occurs rarely and is transmitted directly from man to man by droplet infection. Plague is primarily a disease of rodents although several other mammals, including cats, are susceptible[2, 3], and is a typical zoonosis. In rodents it exists in two forms: wild rodent plague existing in nature independent of human populations and their activities; and domestic plague, intimately associated with man and the rodents living with man, with a definite potential for producing epidemics in man.

The WHO Expert Committee on Plague[11] recently emphasized that the permanent character of wild rodent plague in numerous natural foci in various parts of the world still requires the constant attention of health authorities. In the Republic of South Africa the Public Health Act 36 of 1919 and its regulations list plague as one of the six formidable infectious diseases which must be immediately reported, even if only suspected in man, rodents, cats, dogs or other animals. In a natural focus ecological conditions ensure the persistence of the aetiological agent for considerable periods of time, and epizootics and periods of quiescence alternate without the introduction of infection from outside. In natural foci the infection is maintained permanently by hosts, termed permanent reservoir hosts, which are relatively resistant. They pass the infection to less resistant animal hosts and cause epizootics (murine plague) which affect some domestic rodents and thus cause outbreaks of plague in man[10] particularly in times of famine or unusual meteorological conditions[1]. Pollitzer[7], however, contends that the presence of such reduced susceptibility of the host colony is a *sine qua non* for the continuation of infection.

Several known natural and temporary or probable plague foci exist in southern Africa[11]. In South Africa the gerbils *Tatera brantsii* and *Desmodillus auricularis* associated with the fleas *Xenopsylla philoxera* and *X. piriei* respectively form the permanent wild rodent reservoirs, although other species and subspecies of small mammals including rodents are at risk of infection and other species of fleas have to be regarded as actual or potential vectors[7]. Human infection in this country has in most cases not been directly derived from the wild rodents or through their fleas, but from the semi-domestic *Praomys natalensis* (the multimammate mouse) with a mixed flea fauna derived from wild and domestic sources and the domestic *Rattus rattus* (the black rat) which lives in close contact with man in his dwellings and its associated fleas acting as intermediaries between the primary gerbil reservoirs and man[7]. Plague first gained a foothold in South Africa in 1900 when large amounts of forage were imported from South America during the South African War, and Pollitzer[7] states that there is no doubt that the natural foci of infection became established here through a spread of infection from the commensal or domestic to the wild species of rodents during the present century.

It is the purpose of this paper to record the transmission of plague from a cat to a human by means of a bite.

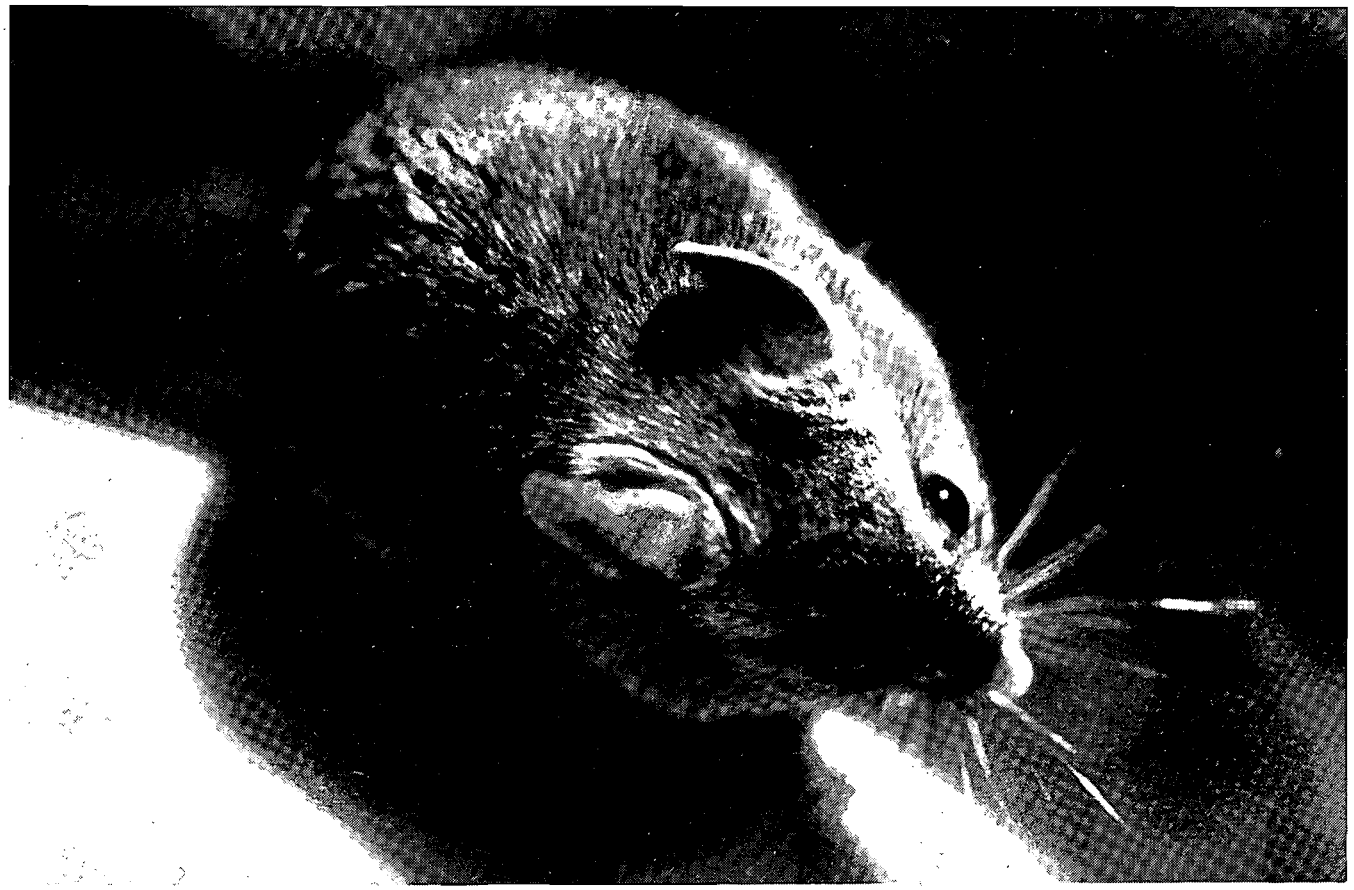

Fig. 3: The semi-domestic multimammate mouse, *Praomys natalensis* forms an important link in the transference of fleas and infection from wild rodents living in natural plague foci to "domestic" rodents and man.

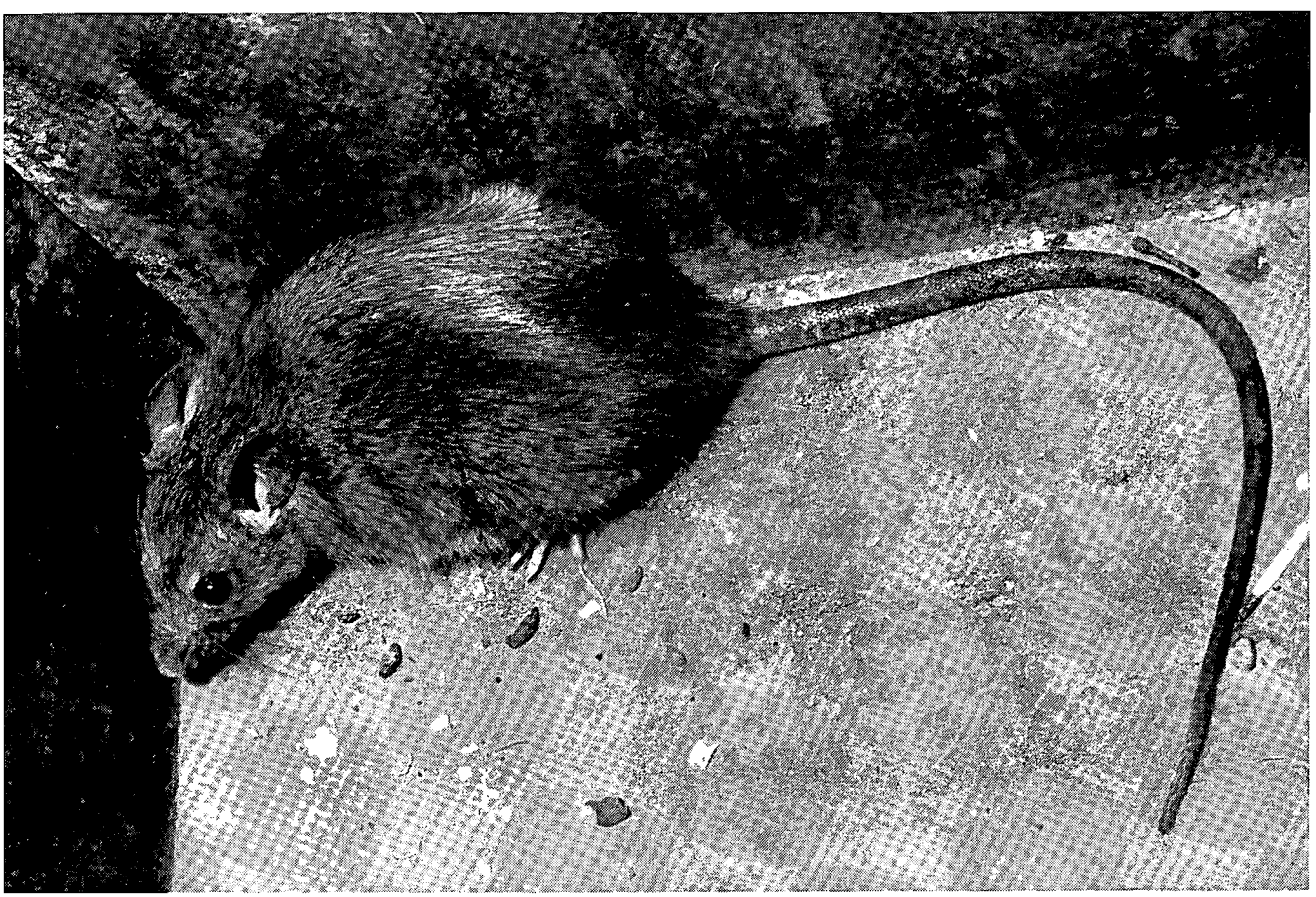

Fig. 4: The black rat, *Rattus rattus* lives in close contact with man in his dwellings.

CASE REPORT

On a farm situated in the Graaff-Reinet district of the eastern Cape Province a two-year-old female domestic cat was seen on approximately 9.11.72 to have a changed habitus; she was dull, inactive, refused to eat and for most of the day lay with her nose resting on the ground. This animal, subsequently named "Pestie", was at that time suckling her own kitten of about 4 weeks of age and had partly adopted four other slightly older kittens from another cat which had also partly adopted Pestie's offspring, i.e., two lactating females were feeding five kittens between them. During the next 2 days the clinical signs shown by Pestie had progressed to a quadriplegia and although she remained inappetent until evidence of recovery on the sixth day of illness was noticed when she appeared brighter and ate some food, she continued to suckle the kittens during the whole period.

On 11.11.72 the four older kittens were noticed to be ill. Two of these died 4 days later and the other two on the 7th day of the disease but none of these sick kittens was examined by us. The farmer and his wife reported that apart from the more general signs associated with an acute infectious disease such as malaise and inappetence, the head and upper part of the neck of the kittens appeared swollen. This appearance may have been the result of enlarged regional lymph nodes. The two kittens which died first had haemorrhaged from the mouth and nose, while the other two which had suffered from the more protracted disease showed frothing from the mouth and nostrils from which strings of mucous were suspended for several days before death. Pestie's own kitten and the other adult cat did not contract the disease. The cats had the run of the house and slept in the kitchen until a week before human illness. Thereafter they slept on the veranda. Of four other cats belonging to labourers on the farm one died during this period.

Veterinary advice was sought and given telephonically soon after Pestie was noticed to be ill. The owners feared that she and the sick kittens might have been suffering from rabies and wished to know whether they could be treated with sulphadiazine administered orally in tablet form. Pestie and the sick kittens were thereafter treated daily with this sulphonamide; in addition some erythromycin syrup was given. It was during the process of administration of a sulphonamide tablet to one of the kittens which had shown frothing from the mouth and nostrils, that the left thumb of the 58 years old farmer sustained superficial laceration from the animal's teeth. The wound healed within a few days. The farmer was bitten on 11.11.72 and examined by one of us (B.J. Te W.N.P.) on 20.11.72 after he had been ill for 3 to 4 days, i.e., the length of the incubation period was somewhere between 3 and 7 days. The condition was characterized by sudden onset with few prodromal signs and symptoms which were of short duration. The latter consisted of malaise, headache, nausea and painful left axillary lymphadenopathy. On the day of the initial examination the bite wound had healed and apart from a fever of 39,5°C the most prominent clinical sign was the presence of a painful swelling about the size of a hen's egg in the anterior region of the left axilla which was unlike that of a secondary lymphadenitis following a wound infection in that it was surrounded by extensive oedema. Treatment with tetracycline was prescribed and instituted but during the course of the following few days the patient manifested signs of mental confusion.

After hospitalization a lumbar puncture was performed on 25.11.72 and a specimen of the cerebrospinal fluid was taken for pathological and bacteriological examination. *Yersinia pestis* was isolated from this fluid. More specific antibiotic and other treatment was instituted and the patient made a very slow and protracted recovery during the course of the following 8-10 weeks[5].

In order to determine the source of the infection Pestie was clinically examined by one of us (D.J.T.) on 1.12.72 and 5 ml blood was taken for serological and bacteriological examination. Apart from being weak and in poor condition she appeared healthy on this day, and her temperature was within normal limits. Serologically, however, she had a titre of 1:512 for plague antibodies but the bacteriological culture was negative. A throat swab taken a few days later also proved negative when examined for the presence of *Y. pestis*. A second serological examination on 12.12.72 revealed a rise in plague antibody titre to 1:1024. She was not bled again and this may or may not have been the final titre.

Once a definite diagnosis of plague had been established all the necessary public health measures, including rodent control, were carried out on the farm by the appropriate authorities. Pestie was placed in isolation and was treated with streptomycin administered intramuscularly while the other cats and the dogs on the farm were regularly dusted with insecticidal powder to control flea infestation. No further animal or human cases were diagnosed on the farm or in its vicinity.

DISCUSSION

Pollitzer[7] discusses in some detail the role played by a variety of animals as hosts of infection to the plague bacillus and quotes the experience of many authors in this respect. He states that while cats may suffer from plague under natural conditions it has been debated whether they can contract infection by feeding, and whether their infection occurs frequently enough to be of importance for the spread of the disease. Various authors are quoted who had reported that under natural conditions cats could contract infection by feeding only if their buccal or intestinal mucosa was traumatized by bone fragments. Cats were also not believed, in general, to be very susceptible to experimental infection with *Y. pestis*, although in various South American countries high mortalities have been noted in cats during plague outbreaks in humans. Unequivocal proof that these animals were suffering from *Y. pestis* infection, however, seems to be lacking. In Argentina instances were often noted where the presence of plague in cats seemed to be responsible for human cases[7].

It is of interest to note that during the "Black Death" of London in 1665 when the recorded human deaths from plague in the city and suburbs exceeded 7000 per week, and the total plague mortality for the year was about 20% of the population, one of the measures promulgated by the Lord Mayor and

Aldermen of the City of London to prevent spread of infection was that "the streets are to be kept clean and no hogs, dogs or cats, or tame pigeons, or conies (rabbits) be suffered to be kept within any part of the city"[9]. Recently Sloan considered that the decision to destroy the dogs and cats was an ill-judged measure and probably led to an increase in the rat population[9]. In view of the apparent lack of resistance of at least five of eight cats described in this paper (if it may be assumed that they had all suffered from plague though there is little doubt that at least two of them were infected) as well as the recent studies by Rust, Cavanaugh, O'Shita and Marshall[8] who have shown that cats and dogs are quite susceptible to experimental plague infection, one wonders whether the edict was indeed ill-judged at that time. Moreover, cats, like dogs, are apt to be dangerous as far as the transmission of plague is concerned as they may bring not only plague — infected rodent fleas into the houses[7] but some have the habit of bringing their actual prey caught elsewhere into dwellings (no doubt diseased rats are easier to capture). While cats are useful in keeping dwellings free from mice their value for purposes of rat control has been questioned[7]. Both the cat and the dog fleas (*Ctenocephalides felis* and *C. canis* respectively) may carry the plague bacillus although they have proved rather inefficient plague vectors under experimental conditions. They occur not only on their specific hosts but also to some extent on other species of mammals including rodents and are prone to attack man[7].

In view of the persistence of infection in natural plague foci in southern Africa in which diverse biotic and abiotic factors condition the ecology of local rodent reservoirs and flea vectors and cause distinct seasonal appearances of epizootic plague, sporadic human cases will continue to occur.

The farm on which the present isolated outbreak occurred lies approximately 160 km from the nearest natural focus of plague although the disease was shown in 1925/6 to have spread among spring hares (*Pedetes capensis*) to within 48 km east of Graaff-Reinet[6]. It was not determined how the cats in the small localized outbreak under review initially contracted the infection but various possibilities do exist. One is that the disease was contracted from wild rodents although no unusual outbreaks of mortality had been observed in these at the time. The Rodent Control Group, however, were able to determine that there had been mortalities from 6 months to a year previously. The Namaqua gerbil (*D. auricularis*) and the semi-domestic multimammate mouse *P. natalensis* as well as *Otomys (Myotomys) unisulcatus* (karoo rat), *Rhabdomys pumilio* (striped mouse) and *Mus musculus* were trapped on the farm shortly after plague was diagnosed. Wild rodent nests were found very near the farm house. It is possible that the disease could have been present in wild form in the district and remained undetected since the early 1900's although no infection of fleas or rodents was found on the farm. Another possibility is that infected rodents could have been introduced onto the farm in 1970 when a quantity of maize for sheep feed was imported from the Orange Free State, parts of which are enzootic areas. A third possibility is that wild carnivores, *e.g.* red lynx, and rodents may have migrated into the area carrying the infection with them.

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