RICKETTSIAL DISEASES OF ANIMALS
TRANSMISSIBLE TO CHILDREN

By Lauri Luoto, D.V.M., M.P.H.

U. S. Department of Health, Education and Welfare, Public Health Service, National Institutes of Health, National Institute of Allergy and Infectious Diseases, Rocky Mountain Laboratory, Hamilton, Montana

Because rickettsial infections occur sporadically, pediatricians may not be as thoroughly familiar with them as they are with common diseases of children that are characterized by rash. Hence, the rickettsias are infrequently considered in the etiology of febrile exanthemas or fevers of undetermined origin. Rickettsial diseases of primary interest in this country are Rocky Mountain spotted fever, murine typhus, rickettsialpox and Q fever. The agents causing these diseases exist as latent infections in arthropods and animals, and they are transmitted accidentally to man through well-defined channels of spread. An awareness of the epizootiology of rickettsial diseases in nature and of the means by which these agents are transmitted to man is helpful in establishing a diagnosis of a rickettsial disease. Therefore, salient features of the natural cycles of infection which may suggest a clinical diagnosis will be stressed, and characteristics of these diseases in children will be reviewed briefly.

Rocky Mountain spotted fever is usually contracted during the spring or summer through the bite of an infected tick although infection also may be acquired by dermal contact with tissues of a crushed or improperly removed tick. The wood tick, Dermacentor andersoni, the dog tick, D. variabilis, and the lone-star tick, Amblyomma americanum, which are found, respectively, in the western, eastern, and southern United States are responsible for transmitting the organism to man. Natural infection with Rickettsia rickettsii in these species is maintained by transovarial passage of the agent and by the feeding of immature stages on infected small mammals. Environmental factors such as rainfall, temperature, food supply of small mammals, and prevalence of predators indirectly affect the abundance of ticks. The
percentage of ticks carrying rickettsiae varies in different areas, but usually it is low. Hence, the likelihood of acquiring the disease from a given tick bite is actually rather slight.

That man is only an accidental host and that he is not involved in the natural cycle of Rocky Mountain spotted fever should be emphasized. When tick-infested areas are visited, certain precautions can be taken to preclude man’s accidental entrance into the cycle. The use of tick repellents or the wearing of suitable clothing will impede ticks attempting to reach body surfaces. Since transmission by D. andersoni may not occur until several hours after attaching, clothes and body surfaces should be inspected carefully at least twice daily. Attached ticks should be removed intact by gentle traction with a forceps or fingers. In the eastern United States, D. variabilis is frequently carried into the house by the family dog. Thus, periodic treatment of the dog with acaricides when ticks are prevalent and disposal of ticks found in the home are important precautions.

Murine typhus occurs principally in the southeastern United States. The natural cycle of this disease involves the common rat and its siphonapteran parasite, Xenopsylla cheopis. Consequently, the majority of cases in children appear during summer and fall among families of low income whose living quarters are rat-infested. The flea, which remains infectious for several months, bites man only when natural hosts are not available. Contamination of the wound with feces of the flea is the probable mode of transmission. During the past decade, the elimination of slum areas in larger cities, and the extensive use of the newer insecticides and rodenticides have drastically reduced the incidence of murine typhus in this country.

Rickettsialpox is caused by an organism antigenically related to the rickettsia that causes spotted fever, but the infection in natural hosts is entirely dissimilar. The natural cycle of infection of R. akari, the causative agent, involves the common house mouse and its parasitizing mite, Allodermanysus sanguineus. Like the flea which transmits murine typhus, the mite prefers its natural hosts and will bite man only when such hosts are not convenient. To date, outbreaks of this disease have been recognized chiefly in large cities in the northeastern United States. It is well known that the Norwegian rat will not share its domain with the common house mouse. Hence, unlike murine typhus which affects low-income families, rickettsialpox occurs chiefly among higher-income families living in mouse-infected apartments or other multiple-housing units.

Q fever differs from the three previously mentioned rickettsioses because the causative organism, Coxiella burnetti, has a natural cycle of infection in small wild mammals and their arthropod parasites and another independent cycle in large domestic animals. The characteristics of the cycle in nature are similar to those described for Rocky Mountain spotted fever. Man is rarely involved in this cycle.

Q fever is known to be endemic among livestock in southwestern United States and the Intermountain area.¹ Recent detection of human cases and foci of infection in cattle in Ohio, Wisconsin and Iowa suggests that the disease probably is disseminated throughout the United States. Chronically infected cattle, sheep, and goats contaminate their environment by the elimination of C. burnetti in placentas and lacteal secretions. Since C. burnetii is extremely resistant to desiccation and since the disease is spread by airborne dust, such a contaminated environment remains a persistent source of infection. Under these conditions livestock presumably acquire the disease by inhalation of infectious air-borne particles. Contact with infected animals during occupational exposure, residence near infected premises, and the household use of raw infected dairy products are factors associated with a high incidence in man.² Although Q fever occurs primarily in males engaged in occupations associated with domestic livestock, cases in children have
been recognized among families residing on or near infected premises and in house-
holds in which raw dairy products are used. Epidemiologic evidence suggests that man
contracts the disease by inhalation of infec-
tious dust.

Since textbooks contain excellent de-
scriptions of the symptomatology of these
diseases, only brief reference will be made
to features peculiar to these rickettsioses. Early symptoms, such as headache, fever,
chills, and myalgia are, of course, common
to these diseases and other febrile illnesses.
With the exception of Q fever, each of the
rickettsioses is characterized by a distinc-
tive rash. In patients affected with Rocky
Mountain spotted fever, a rose-red maculo-
papular rash appears first on the wrists and
ankles and then spreads to the extremities,
trunk, face, and usually to the palms and
soles. Later these lesions become petechial
and in severe cases they may form confluent
purpuric areas. The rash observed in pa-
tients with murine typhus is somewhat
similar in appearance but differs in distribu-
tion. The rash appears first on the trunk and
then spreads to the rest of the body except
the face, soles and palms. In the southern
United States where both diseases occur,
spotted fever often is misdiagnosed as
murine typhus. A distinguishing feature of
rickettsialpox is a red papule which appears
during the prefebrile period at the site of a
mite bite. The papule later becomes vesic-
ular, then dries to form a black eschar.
The early rash of this disease is more papular
than macular and lesions occur at any site
except the soles and palms. Later, vesicles
form in the summit of papules; lesions eventu-
ally desiccate to form black crusts which
fall off without producing scars. Before this
disease was established as a distinct entity,
cases often were diagnosed as chickenpox.

Patients affected by Q fever present only
generalized signs that may suggest a diag-
nosis of influenza. A transient pneumonitis
that may accompany severe cases has been
confused with atypical pneumonia.

The diagnosis of a rickettsial disease
should be confirmed by laboratory tests.
Isolation of the responsible rickettsia and
its subsequent identification is possible;
however, these procedures are expensive
and time-consuming, and they are seldom
done. Preferably, a rise in complement-
fixing antibody titer between blood speci-
mens taken during the acute phase and con-
valescence should be demonstrated. The
Weil-Felix test may serve as a diagnostic
aid but results must be interpreted with
caution since high concentrations of ag-
glutinins in the serum may develop during
nonrickettsial infections. The rickettsias
that cause rickettsialpox and Rocky Moun-
tain spotted fever are so closely related
antigenically that the two diseases can not
be distinguished by complement-fixation
tests. Differentiation must be based on
clinical and epidemiologic findings. In the
South, murine typhus and spotted fever
may be confused; hence, the diagnosis of
these diseases should be confirmed sero-
logically. Since the symptoms of Q fever
in man are similar to those of severe influ-
enza or primary atypical pneumonia, diag-
nosis can be confirmed only by a rise in
antibody titer during convalescence. Diag-
nosis of these infections may be compli-
cated if patients are treated early with
broad-spectrum antibiotics. These drugs
cause a prompt remission of symptoms and,
also, they may delay or even impede de-
velopment of antibody.

In many western states, Colorado tick
fever, which is caused by a virus also trans-
mitted by D. andersoni, may be confused
with Rocky Mountain spotted fever. Severe
neurologic involvement has been associated
with Colorado tick fever in young children.
In 1955, 1956, and 1957, more than
100 isolations of virus were made annually at
the Rocky Mountain Laboratory from
blood specimens of patients who had been
bitten by ticks. A diphasic fever, leuko-
penia and absence of rash characterize this
disease and distinguish it from spotted
fever.

In summary, knowledge of the pertinent
features of the natural cycles of infection
of these rickettsial agents and of exposure
Epidemiologic Aspects of Leptospirosis

By James H. Steele, D.V.M., M.P.H.
U. S. Department of Health, Education and Welfare, Public Health Service, Bureau of State Services, Communicable Disease Center

More than 70 years have passed since Weil first described several cases of leptospirosis in humans although he had no knowledge of the etiologic agent. In 1905, Stimson,1 in New Orleans, first observed leptospires in kidney sections from a patient believed to have died of yellow fever. A few years later (1915) pathogenic leptospires were isolated first from patients with Weil’s disease in Japan by Inada et al.2 and independently in Germany by Uhlenhuth and Fromme3 in the same year. About the same time the Japanese workers4 found that the rat (Rattus norvegicus) is a carrier of Leptospira icterohaemorrhagiae. Since this early work, extensive investigations have shown that leptospirosis occurs in humans and animals in all parts of the world and is not a single disease but a group of diseases caused by a variety of leptospiral serotypes. Possible explanations for the wide distribution of these diseases may be (1) that they are insidious infections which have appeared gradually and become established almost unnoticed,5 or (2) that they have been present for many years and remained largely undetected because of the variable masking symptoms and the problems associated with laboratory diagnosis. Although considerable attention has been directed toward studies of leptospiral infections by European and Asian investigators since the early 1930’s much of our knowledge regarding the epidemiology, public health importance and distribution of these diseases in the United States has been gained only during the past decade.

The epidemiology of the leptospiroses has been shown to follow a characteristic pattern based, in part, upon the fact that distribution of Q fever: Human, animal and arthropod infection. Am. J. Hyg., 57:125, 1953.

REFERENCES

AMERICAN ACADEMY OF PEDIATRICS – PROCEEDINGS

ADDRESS: Atlanta, Georgia.

Downloaded from http://pediatrics.aappublications.org/ by guest on October 23, 2017
RICKETTSIAL DISEASES OF ANIMALS TRANSMISSIBLE TO CHILDREN
Lauri Luoto

Pediatrics 1958;22;384

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://pediatrics.aappublications.org/content/22/2/384