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Lecture title: Q fever

Query fever, Balkan influenza, abattoir fever

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Summary:

Q fever is a common zoonosis (infection that could transmit from animals to humans), caused by Coxiella burnetii. Natural reservoirs include several domestic and wild animalsIt can cause reproduction problems in livestock and severe respiratory (lung) and liver disease in humans

Etiology

It is caused by a rickettsia, *Coxiella burnetii*, an obligate intracellular bacterium. The causative organism has a global distribution and it is possible for many species, including ticks, fleas and lice, as well as many vertebrates, to carry the disease.

The main significant zoonotic reservoir is considered to be bovines and also sheep. Once infected, the organism colonises and produces infective foci in the mammary glands and the placenta of pregnant animals. During birth large quantities of the organism can be found in the amniotic fluid and on the placenta. The organism is capable of forming an environmentally resistant spore form capable of forming the inoculum for delayed outbreaks.

The presence of the organism in milk results from the colonisation of the mammary system, and host animals can carry the disease for prolonged periods, with shedding occurring sporadically or constantly during lactation.
The organism is resistant to heat but ideal pasteurisation conditions will remove it from milk; however, there is a risk from unpasteurised or incompletely pasteurised milk or milk products. It has been postulated that urine or faeces from infected animals may also be a carrier medium for the organism.

Seventeen cases of Q fever were reported in England during 2005, and six in Northern Ireland. Most cases were in male agricultural workers who were probably exposed to the pathogen in the course of their work.

Transmission

Transmission to humans usually follows exposure to infected material . The people at highest risk are veterinary surgeons and stock people who assist at births, although the organism is highly resistant to **desiccation** and therefore can infect individuals working with hides, fleece or bones of infected animals. Transmission is by **direct contact** with contaminated materials, especially the afterbirth or material contaminated with amniotic fluid. There is some evidence that inhalation of dust from infected straw or bedding and even soil may also cause infection. Further down the food-processing chain, transport drivers and abattoir workers may also be at risk. **Drinking milk** or consuming contaminated milk products is also a possible route of infection, and transmission via ticks, lice or fleas has been demonstrated.

Disease in humans

Most exposed individuals display no signs of clinical disease.
Infection rates and recording of clinical cases correspond to lambing and calving cycles, allowing for the time lag associated with the organism's incubation period.
After infection there is an incubation period of between 2 and 4 weeks followed by an acute onset with high fever, associated chills, profuse sweating and severe headache. Unlike other rickettsial diseases, in humans there is no skin rash. The patient may also present with anorexia, sickness and lethargy. The fever may last anything from 9–14 days and can recur at intervals, with a total duration of up to 3 months. A dry cough may be present, with pain in the chest cavity similar to pleuritic pain. 'Cracking' in the chest may also be heard during respiration. Lesions in the lungs may be apparent on radiographic examination. Liver enlargement or tenderness with associated hepatitis-type symptoms can be seen.

Untreated cases can resolve within 5–14 days, although symptoms may not regress for more than 7–8 weeks and relapses may occur. The untreated fatality rate is estimated at 1% of cases. Following severe infection there may be a need for

prolonged convalescence. Elderly patients are particularly badly affected by this disease and may require prolonged supportive measures.

A chronic form also exists that causes a prolonged endocarditis leading to valvular damage, especially of the aortic valve. Recent figures show that damage is more common in patients with pre-existing valve damage.

Symptoms can appear long after the disease has run its clinical course and may require replacement of damaged valves. The fatality associated with this form is estimated to be as high as 60% of cases unless corrective surgery is undertaken. Chronic hepatitis also develops in a small number of cases.

Diagnosis

Diagnosis follows serological testing, because the organism is slow growing and almost impossible to culture from clinical specimens. There are several techniques, of which the most reliable are indirect immunofluorescence, complement fixation, enzyme-linked immunosorbent assay (ELISA) and microagglutination.

Prevention

As with many other zoonoses, prevention strategies revolve around good personal and environmental hygiene. Bedding contaminated by postpartum Zoonoses of agricultural animals **.**
material and the material itself should be carefully handled, with collection and subsequent burying or incineration.
Disinfection of housing.
Protective clothing, including respirators, overalls and gloves, must be worn wherever feasible. In the USA a vaccine for cattle has been developed; it is not licensed for use in the UK.
Carrier animals have been subject to eradication by slaughter policy.
All milk and milk products should be pasteurized.