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An outbreak of Q fever in Staffordshire

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INTRODUCTION

Q fever was first recognised in 1935 in Brisbane (Australia) and was referred to as 'abattoir fever' (Derrick, 1937). In 1937 Burnet & Freeman showed that the infection was due to *Rickettsia*. During World War II the disease caused widespread sickness among members of the allied forces operating in the Mediterranean area and subsequently the disease has been found to be world-wide in distribution.

Incidence of confirmed cases occurring in England and Wales has been reported by the Public Health Laboratory Service as follows (Vernon, 1967):

1962 (53), 1963 (31), 1964 (30), 1965 (33), 1966 (32).

In this country it has been recognized for some time that the disease is associated mainly with sheep and cattle. Sheep farmers were shown to be at special risk in the lambing season when the infection is acquired from the infected placenta (Marmion & Stoker, 1956). Dairy farm workers are likewise at risk during calving, but this has no seasonal incidence, as calving takes place all the year round. Dust from the byre has been shown to be the source of infection on several occasions and infected dust from straw was held to be responsible for an outbreak in East Kent among the students and staff of a college (Harvey, Forbes & Marmion, 1951).

Case-to-case infection is uncommon but has been described in a small outbreak among R.A.F. personnel when four attendants in the sick bay and one visitor to the sick bay were all infected from a patient who had acquired his original infection on a farm in Cornwall (Holland *et al.* 1960).

In another outbreak involving R.A.F. personnel, the men affected had helped to clean out sheds into which sheep had strayed and probably lambed. The infection was presumed to have been acquired from the infected dust (Holland *et al.* 1960).

Raw milk has been suspected as the vehicle of infection on more than one occasion. Marmion & Stoker (1950) stated that those more likely to be affected from this source were town dwellers who drink non-pastuerized milk on a visit to the country. Marmion & Harvey (1956) reported 23 sporadic cases in Kent between 1948 and 1954 which they concluded had probably been infected by milk.

Evans (1956) recorded that approximately 5 % of milk samples from individual herds in South Wales were infected with *R. burneti*.

Huebner & Bell (1951) recorded that 10 % of dairy cows in Los Angeles County area were infected with *R. burneti* and that butter made from unpasteurized milk, from a known infected herd, was itself infected.

Unpublished work of one of us (W. L. H.) has shown 18 % of herds from which raw milk is retailed within the county of Staffordshire to be infected with *R. burneti*.

THE OUTBREAK

The Detention Centre in which the outbreak occurred is situated 4½ miles from the nearest town centre near open farm land and is about 700 ft. above sea level. The main building is of stone with outhouses, workshops and farm buildings. The standard of hygiene in the Centre and on the farm is of a very high order and rodents are rarely seen. The Centre has accommodation for 100 boys and in March and April 1967 the daily population varied from 95 to 99. The average length of stay is 10 weeks and the age range of detainees is from 18 to 21 years. There is a staff of twenty officers and some clerical assistants. All entrants are given a number on the day of admission, provided with clean clothing and bedding and then medically examined. Their first night is spent in separate cubicles and then they are transferred to the dormitories. The new entrants are purposely distributed throughout the establishment for administrative reasons.

Detainees are employed in the Centre in one of three ways: (i) domestic work within the Centre, (ii) agricultural work in the fields or tasks connected with animal husbandry, (iii) in manufacturing concrete blocks, fence supports, etc.

The outbreak was explosive in nature and occurred between 15 and 29 April 1967.

Boys began reporting sick from 15 April onwards and the clinical picture they presented was remarkably uniform. In practically every case there was high temperature ranging from 101 to 105° F associated with shivering, sweating and backache. Sore throat, headache, chest pains and neck stiffness were also common. Most had an irritating cough. X-ray examination revealed consolidation of the lungs in five cases. Three cases had transient, diffuse rashes on the trunk, of a fine punctate erythematous nature.

The duration of pyrexia varied from 2 to 7 days. No organized trial of antibiotic therapy was carried out but approximately half the patients were treated with tetracycline and the remainder had none. The general impression was that both groups progressed equally well. All patients were isolated, kept in bed and treated with aspirin only, except for those who also had tetracycline. Recovery was quick in most instances owing probably to the patients being fit young men with good physique.

One patient who was definitely more ill than the others was admitted to the local isolation hospital. He had a temperature of 105° F. on admission. He was shivering and this was followed by profuse sweating. He complained of headache, backache and pain in the loins. He had a slight cough but there were no obvious physical

signs in the chest. Crepitations became audible in the right lung 48 hr. after admission. The liver was not enlarged but the spleen was palpable. There was no enlargement of lymph glands and no rash was present. The blood picture was uninformative except for a raised E.S.R. X-ray of the chest showed pneumonic consolidation at the right base. He was treated with tetracycline and the temperature fell to normal within 72 hr. It was 6 weeks before the lungs were radiologically clear. A total of 24 boys and five officers suffered a clinical illness but a full investigation was not possible in all of them. Some of the boys were discharged before second specimens of blood for serology had been obtained. Follow up of these patients proved difficult. It was, however, possible to show serological as well as clinical evidence of Q fever in nineteen boys and five officers.

LABORATORY INVESTIGATION

The advice of the virologist from the local Public Health Laboratory was sought as soon as it became clear that an epidemic of unknown cause was occurring.

Blood specimens and throat swabs were taken from nine of the inmates who were ill on 21 April 1967. The sera were separated and stored at $-20°$ C. The swabs were broken off in transport media containing Hanks's basal salts solution, 0·2 % bovine albumen and antibiotics at the bedside and transported immediately to the laboratory in a flask of melting ice. On receipt, equal volumes of the transport media were inoculated into tissue culture tubes containing monolayers of monkey kidney, human embryo kidney, HeLa and W.I. 38 fibroblast cells. Tubes were incubated at 33° C. and examined over a period of 21–28 days for cytopathic changes and also for haemadsorbing properties using guinea-pig blood cells at 4° C. and at room temperature. No viral agents were detected.

Table 1. *Tests on paired serum specimens from patients*

Case no.	Register no. of inmate	Q fever C.F. antibody titre 21.iv.67	3.v.67	Biological test results Guinea-pig serology
1	637	< 1/5	1/80	Negative
2	647	< 1/5	1/160	Positive for *R. burneti*
3	672	< 1/5	1/80	Negative
4	673	< 1/5	1/160	Negative
5	674	< 1/5	1/320	Positive for *R. burneti*
6	676	< 1/5	1/80	Negative
7	628	< 1/5	< 1/5	Negative
8	677	< 1/5	1/160	Negative

When it seemed that it was unlikely that an agent would be isolated it was decided that an attempt should be made to diagnose the disease by serological methods. Convalescent sera were obtained from seven of the nine patients bled on the earlier occasion. The remaining two had completed their stay at the Detention Centre and had been released. Acute and convalescent sera from the patient admitted to the isolation hospital was also available. Complement-fixation tests were carried out in 80 hole plastic agglutination trays by the method of Bradstreet

& Taylor (1962) using 3 M.H.D. complement, and antigens supplied by the Standards Laboratory. Antigens employed were Influenza A, B and C, Sendai, Psittacosis/L.G.V., Adenovirus, respiratory synctial virus, Q fever (*R. burneti*) and *Mycoplasma pneumoniae*. None of the pairs of sera showed rises in antibody titre except to Q fever. Fourfold or greater rises in complement-fixing antibody were shown in seven of the eight patients as shown in Table 1.

When these results were known it was decided to inoculate guinea-pigs with 1 ml. amounts of the acute phase sera which had been stored for 2 weeks at $-20°$ C. The specimens were inoculated intramuscularly into the thigh and the animals were bled and killed after a period of 4 weeks. Presence of a high level of complement-fixing antibody in the guinea-pig serum was taken as an indication that the inoculum had contained live *R. burneti* organisms. This was demonstrated in two patients (Table 1).

Convalescent sera from 11 out of 14 other inmates who were clinically ill at the same time as those which were fully investigated showed complement-fixing antibody at a titre 1/20 or more. At the same time eight out of nine sera from inmates who had had no symptoms had antibody titres of less than 1/5. Only one symptomless inmate had a raised antibody titre of 1/60 and he denied having experienced anything which could have been attributed to infection.

When it was established that the epidemic was due to Q fever, the source of infection was looked for and the obvious possibility was the farm and milk produced from the dairy herd.

The possibility of environmental contact with infected animals was fully investigated but it was found that, although the centre had a herd of cows and a number of pigs, none of the sick inmates had been engaged in duties which brought them in contact with any of the animals in the 3 weeks before the outbreak. The one outstanding feature was that milk from the farm was consumed raw on one or two mornings weekly when cornflakes were served at breakfast. All other milk used in the kitchens was subjected to some form of heat during the cooking process and cold milk drinks were never issued. Officers at the Centre did not drink the raw milk or eat meals prepared in the kitchen but there was evidence that they had drunk cups of tea containing the untreated milk and it must be assumed that the organism was able to survive the temperature of the hot tea. Officers' families purchased heat-treated milk from local retailers and excess milk from the farm at the Centre was always sold to a large dairy, where it was duly pasteurized. One officer, who was clinically and serologically a definite case, denied ever consuming milk in any form. Huebner *et al.* (1949) showed that naturally infected milk failed to yield *R. burneti* after high-temperature–short-time heat treatment at $160{\cdot}5°$ F. $\pm 0{\cdot}3°$ F. for 15 sec. but that the organism could survive 35 min. at a temperature of $143°$ F $\pm 0{\cdot}5°$ F.

The farmer and his wife and the two regular estate hands never drank the raw milk but did have constant contact with the farm animals. None of them became infected clinically or serologically with the disease.

Milk being the probable source of infection, it was arranged with the veterinary surgeon to have blood samples from each milking cow examined for Q fever

antibody and milk from each cow injected into guinea pigs. The results in Table 2 show that, of the 20 cows examined, two were actively excreting *R. burneti* in their milk and three others had Q fever antibody titres of 1/10 or more in their serum. The opportunity was also taken to examine the milk for the presence of *Brucella abortus*. These results are also shown in the table. Although brucella organisms were not isolated from any of the milk specimens, the Ring Test for brucella antibody was positive in the milk from five animals.

Table 2. *Investigations carried out on milking cows*

Cow no.	Cow serum Q fever antibody titre	Tests on guinea-pigs after injection of milk			Direct milk tests		Months since parturition
		Q fever CFT	*Br. abortus* agglut.	*Br. abortus* culture	Brucella ring test	Brucella culture	
1	–	–	–	–	–	–	3
2	–	–	–	–	–	–	0
3	1/10	–	–	–	–	–	2
4	–	–	–	–	+	–	0
5	–	–	–	–	+	–	3
6	–	–	–	–	–	–	2
7	–	–	–	–	–	–	8
8	–	NS	NS	NS	NS	NS	·
9	–	–	–	–	–	–	2
10	–	+	–	–	–	–	3
11	–	–	–	–	+ +	–	1
12	1/10	–	–	–	–	–	10
13	–	–	–	–	+	–	9
14	–	–	–	–	–	–	11
15	–	–	–	–	–	–	9
16	–	–	–	–	–	–	3
17	1/10	–	–	–	–	–	9
18	1/20	+	–	–	+ +	–	13
19	–	NS	NS	NS	NS	NS	·
20	–	NS	NS	NS	NS	NS	·

NS = no specimen of milk available.

As an emergency measure, early in the outbreak, arrangements were made for all milk to be boiled. When it was established that the milk from the herd contained live Rickettsia organisms, the authorities arranged that all the milk produced on the farm should be sold for pasteurization and a heat-treated supply obtained for the needs of the establishment.

CONCLUSIONS AND OBSERVATIONS

This outbreak is of particular interest because it occurred in an institution where the activities of the inmates were under strict control.

The explosive nature of the outbreak and the scatter of the cases throughout the institution was very much against the infection being from a human source.

The fact that most of those affected were known to have had no direct contact with the dairy farm excluded the possibility of a dust-borne infection.

Spread by parasitic insects could be completely excluded, as there was no infestation among the inmates. A high standard of hygiene is maintained in the institution and enforced personal cleanliness of the inmates makes such an occurrence very unlikely.

Food-borne infection appeared to be the only likely explanation of the outbreak. The only food substance possible in this particular case was raw milk and this prompted us to investigate the cattle in the institution farm which supplied the raw milk. The evidence obtained from this investigation points strongly to milk as the vehicle of infection and we believe this to be the first milk-borne outbreak of Q fever to be reported in Britain.

Clinically the outstanding features were the short duration of symptoms even when no antibiotic was given, and the low incidence of pulmonary complications.

Q fever is yet another of the health hazards associated with raw milk. Heat treatment of all milk supplies is the only health policy compatible with safety.

It is a curious anomaly in this country that retailers are allowed to charge more for raw milk than for pasteurized or sterilized milk. The consumer may indeed be paying for something extra but it is often not quite what he expects.

All milk in Britain to-day comes from Tuberculin Tested herds, but there is no guarantee that untreated milk is free from typhoid and other salmonella organims, brucella, Q fever, streptococci, staphylococci or faecal bacteria. Most milk sold in urban areas is heat-treated but raw milk is still widely available in rural and semi-urban districts. Q fever must clearly be considered when outbreaks of obscure pyrexia occur in such areas.

Evans, Powell & Burrell (1959) reported a fatal case of endocarditis associated with Q fever. Recent published work of Grist, Ross & Sommerville (1967) and Kristinsson & Bentall (1967) has implicated *R. burneti* as a cause of subacute endocarditis. If a patient is, therefore, known to have had Q fever and later in life develops an unexplained low grade febrile illness, the possibility of rickettsial endocarditis should be borne in mind.

SUMMARY

This article describes an outbreak of Q fever involving 24 men (five prison officers and nineteen detainees) in one of H.M. Detention Centres. The evidence collected points strongly to the consumption of raw milk as the route of infection. This appears to be the first milk-borne outbreak of Q fever to be reported in Britain.

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