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Truckin' pneumonia – an outbreak of Q fever in a truck repair plant probably due to aerosols from clothing contaminated by contact with newborn kittens

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SUMMARY

We describe an outbreak of Q fever affecting 16 of 32 employees at a truck repair plant. None of the cases were exposed to cattle, sheep or goats, the traditional reservoirs of Q fever. The cases did not work, live on, or visit farms or attend livestock auctions. One of the employees had a cat which gave birth to kittens 2 weeks prior to the first case of Q fever in the plant. The cat owner fed the kittens every day before coming to work as the cat would not let the kittens suckle. Serum from the cat had high antibody titres to phase I and phase II *Coxiella burnetii* antigens. The attack rate among the employees where the cat owner worked, 13 of 19 (68%), was higher than that of employees elsewhere, 3 of 13 (28%) [$P < 0{\cdot}01$]. The cat owner's wife and son also developed Q fever. None of the family members of the other employees with Q fever was so affected.

We conclude that this outbreak of Q fever probably resulted from exposure to the contaminated clothing of the cat owner.

INTRODUCTION

Q fever was first described in 1937 by Derrick following his investigation of an outbreak of a febrile illness that affected 20 of 800 employees of a Brisbane meatworks (Derrick, 1937). Subsequently it was shown that the rickettsial organism *Coxiella burnetii* was the aetiological agent of this infection and that it is acquired by inhalation of infectious particles (Turck, 1981). Infected cattle, sheep and goats are the usual reservoirs whereby this infection is spread to man (Babudieri, 1959). The placentas of these animals are heavily infected and at the time of parturition aerosols are created which when inhaled by man may result in a variety of illnesses including a self-limited febrile illness, an atypical pneumonia-like picture, hepatitis or rarely endocarditis (Babudieri, 1959; Welsh *et al.* 1958). Q fever occurs worldwide and has been reported in at least 51 countries on 5 continents (Leedom, 1980).

Q fever had not been reported in Nova Scotia prior to 1979, at which time we

began to study atypical pneumonia (Marrie *et al.* 1982). From 1979–87, 170 cases of Q fever were identified (Marrie, 1988). The epidemiology of Q fever in Nova Scotia seems unique in that exposure to infected parturient cats is a risk factor for acquisition of this illness (Marrie *et al.* 1988). In this report we describe an outbreak of Q fever in a truck repair plant. Our investigations led us to conclude that the outbreak was due to contamination of the clothing of one of the workers by newborn kittens and subsequent spread of this infection to his co-workers as a result of aerosols from his contaminated clothing.

MATERIALS AND METHODS

Case definition

Q fever was diagnosed on the basis of a fourfold rise in titre by the indirect immunofluorescence or complement fixation tests or on the basis of a stable titre of $\geqslant 256$ by the indirect immunofluorescence test.

Epidemiological investigation

The outbreak came to our attention on 21 October 1987 and one of us (DL) visited the plant on several occasions starting on the following day. A questionnaire was administered to all employees. The plant and surroundings were physically inspected. Chest radiographs performed at the local hospital were reviewed to find additional cases of pneumonia.

The plant

The plant consists of a modern two-story building and three adjacent one-story buildings located in the town of Truro, population 13000. Nineteen of the 32 employees work upstairs in the modern building. The upstairs portion consists of two parts, of which about one half is an office area and the other half the parts supply area. Downstairs is garage space for the repair of trucks and other heavy duty equipment, staff washrooms and the board room. One of the adjacent buildings is a truck repair area and two other buildings are used for storage of equipment. The heating system is a hot air system. There is no cooling tower and the water is from the mains supply to the town of Truro.

Serological tests

Antibodies to *Coxiella burnetii* phase I and phase II antigens were determined using an indirect immunofluorescence test (Marrie *et al.* 1984) and to phase II antigen only by a complement fixation test (Marrie *et al.* 1985). Antibodies to *Mycoplasma pneumoniae*, adenovirus, respiratory syncytial virus, influenza A, B viruses, parainfluenza 1, 2, 3 viruses and cytomegalovirus were tested for by a complement fixation technique (Marrie, 1982). Antibodies to *Legionella pneumophila* serogroup 1 were detected by an indirect immunofluorescence test using antigens obtained from the Centers for Disease Control, Atlanta, GA. (Marrie *et al.* 1984).

Antibodies to *Coxiella burnetii* phase I and phase II antigens were demonstrated in feline and rabbit serum samples using the IFA test and the appropriate antisera (Marrie *et al.* 1984).

Antigens used in the immunofluorescence test were whole cell antigens of strain Nine Mile obtained from Dr J. C. Williams, Fort Detrick, Maryland, USA.

Microbiological studies

Material obtained at bronchoscopy was cultured for bacteria and respiratory tract viruses by standard techniques. Urine samples from all affected employees were cultured for adenovirus and were examined by direct electron microscopy for the presence of this virus.

RESULTS

The outbreak was drawn to our attention by two patients who were admitted to our hospital with severe atypical pneumonia.

Case 1

On 12 October 1987 a 42-year-old secretary in a truck repair plant developed diarrhoea followed over the next 4 days by fever, nausea, arthralgia, dry cough and left shoulder and back pain. A chest radiograph showed left lower lobe changes and she was referred to our hospital where a clinical diagnosis of atypical pneumonia was made. Therapy was begun with erythromycin and rifampicin with rapid improvement and she was discharged on 27 October.

Blood, urine and stool cultures were negative for bacterial pathogens. Stool cultures were negative for adenovirus and enteric viruses. No viruses were seen when the stool was examined by electron microscopy.

Acute and 4-week convalescent serum samples were obtained. There was no evidence of recent infection by *Mycoplasma pneumoniae*, influenza A and B, parainfluenza viruses 1, 2 and 3, adenovirus, respiratory syncytial virus, cytomegalovirus, *Legionella pneumophila*. There was a 32-fold rise in antibody titre to *Coxiella burnetii* from 16 to 512 for phase II antigen.

Case 2

On the same day that case 1 was admitted, case 2, a 38-year-old male was transferred to our hospital. He had become ill on 10 October 1987 with headache fever, chills, night sweats and later increasing shortness of breath, fatigue and myalgia.

Over the next 3 days his illness progressed and he was transferred to our hospital on 20 October with clinical and radiographic signs of pneumonia. No bacterial pathogens were isolated but the titre to *Coxiella burnetii* phase II antigen rose from 128 to 1024 in the convalescent sample.

The outbreak – clinical features

Sixteen of the 32 employees met the case definition. Nine were ill enough to lose time from work and three were hospitalized. Five said they were not ill but they did seroconvert to *C. burnetii*. Two employees who did not meet the case definition were ill but did not seroconvert to *C. burnetii*. The epidemic curve for the 11 who were symptomatic with Q fever is given in Fig. 1. The onset in all cases occurred between 8 October and 15 October. The remaining five cases could not date the

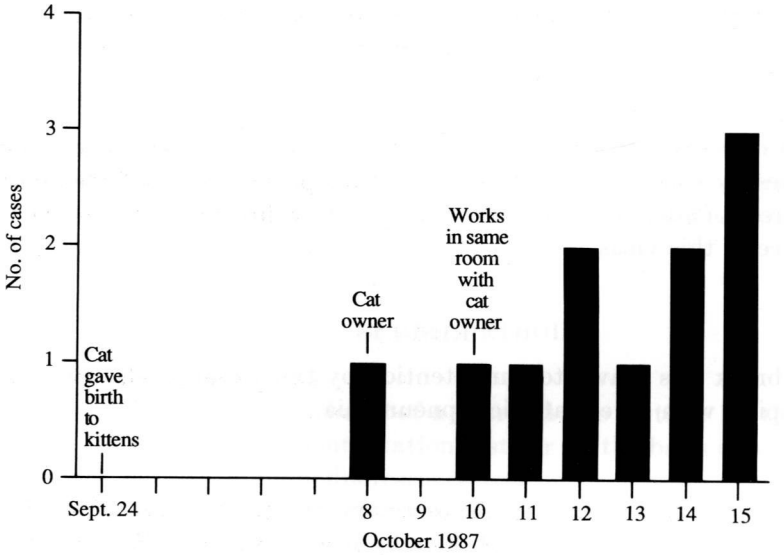

Fig. 1. Epidemic curve – outbreak of Q fever in a truck repair plant.

onset of their illness – indeed all five felt they were well although two had minor complaints (Table 1).

Thirteen of the 16 employees who did not have Q fever had no measurable antibody (< 1:8) to *C. burnetii* phase I and II antigens in three serum samples collected between 13 and 63 days following the onset of illness in the first case.

The most common symptom suffered by the cases was chills followed by headache and fever (Table 2). Fifty percent had cough, 44% had chest pain and 38% had diarrhoea. One person complained of haematuria, however all six who had a urinalysis carried out had microscopic haematuria. One of these had severe flank pain mimicking renal colic to such an extent that a renal calculus was diagnosed. Fifteen of the 16 cases had one or more of the symptoms listed in Table 1.

In 9 of the 16 of cases who had a chest radiograph performed an abnormality was noted. Subsegmental opacity which was present in 6, segmental opacity in 2 and 1 had plate-like atelectasis. Five of the 9 had involvement of more than one lobe.

Epidemiology

All employees were questioned regarding their activities in the month prior to the outbreak. None of those who seroconverted, worked on a farm, visited a farm, cleaned out a barn or visited a livestock auction. One case had slaughtered a cow within the past month. Cases and non-cases were compared for further exposure to animals (Table 3).

One of the patients owned a cat that gave birth to three kittens on 24 September 1987. The cat refused to let the kittens suck so the owner put on his work clothes each morning and fed the kittens using a dropper, and then he went off to work. The cat had antibody titre to *C. burnetii* phase I antigen of 256 and to phase II antigen of 1024. This cat's owner became ill on 8 October 1987 (Fig. 1). The worker with whom he was in daily contact in an enclosed area in the upstairs portion of the building area, became ill 2 days later on 10 October 1987. Thirteen

Table 1. *Selected clinical epidemiological and serological features of the 16 cases of Q fever*

Case	Age	Sex	Date of onset	Symptoms: Headache	Fever	Cough	Abdominal pain	Diarrhoea
1	42	M	Oct 8	+	+	+	0	+
2	33	M	Oct 10	+	+	0	0	+
3	38	M	Oct 11	+	+	+	0	+
4	37	M	Oct 12	+	+	0	+	0
5	45	M	Oct 12	+	+	0	+	0
6	44	M	Oct 13	+	+	0	0	0
7	41	F	Oct 14	+	+	+	+	+
8	29	M	Oct 14	+	+	+	0	0
9	42	M	Oct 15	+	+	0	0	0
10	28	M	Oct 15	+	+	+	0	0
11	31	M	Oct 15	+	+	+	0	+
12	40	M	not ill	0	0	0	0	0
13	59	M	not ill	+	0	+	0	0
14	53	M	not ill	+	0	0	0	0
15	55	M	not ill	0	0	0	0	0
16	58	M	not ill	0	0	0	0	+

Case	Pneumonia	*C. burnetii* phase II antibody titre – IFA*: Date of Acute phase serum	Acute phase titer	Convalescent phase titre	Date of convalescent serum sample	–CF** Acute/ convalescent sample
1	Yes	Oct 26	512	1024	Nov 13	32/256
2	Yes	Oct 26	256	1024	Dec 4	32/256
3	Yes	Oct 21	128	1024	Nov 13	32/1024
4	—	Oct 26	256	2048	Dec 4	64/1024
5	Yes	Oct 26	128	4096	Dec 4	16/64
6	Yes	Oct 26	512	128	Nov 13	16/128
7	Yes	Oct 21	16	512	Nov 13	8/512
8	—	Oct 26	32	256	Nov 13	8/64
9	Yes	Oct 26	32	128	Nov 20	< 8/64
10	—	Oct 26	64	512	Nov 13	64/256
11	Yes	Oct 26	512	1024	Nov 13	256/1024
12	—	Oct 26	256	512	Dec 4	64/64
13	—	Oct 26	< 8	4096	Dec 4	ND
14	—	Oct 26	< 8	256	Nov 13	< 8/64
15	—	Oct 26	512	512	Dec 4	8/8
16	—	Oct 26	128	128	Nov 13	< 8/32

* Indirect immunofluorescence test.
** Complement fixation test.

of the 19 (68%) upstairs employees developed Q fever compared with 3 of 13 (23%) ($P < 0{\cdot}01$ Fishers exact test) employees who worked downstairs. One of the downstairs employees who developed Q fever spent about 25% of his time upstairs. A cat owned by an employee who did not develop Q fever, gave birth to kittens in early September but had no antibody to either phase I or phase II *C. burnetii* antigens. The rabbit owned by another well employee gave birth on October I and had no antibodies to *C. burnetii*.

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Table 2. *Comparison of Q fever cases with employees (non-cases) who did not develop Q fever*

	Q fever	No Q fever
Males/females	15/1	15/1
Mean age (years)	42	47
No. who lost time from work	9	1
No. with any symptom	15	2
No. with this symptom		
Chills	14	0
Headache	13	1
Fever	12	1
Myalgia	11	0
Fatigue	9	1
Cough	8	1
Chest pain	7	0
Anorexia	7	1
Diarrhoea	6	1
Sore throat	5	0
Confusion	5	0
Abdominal pain	3	1

Table 3. *Exposure of employees with Q fever and employees who did not develop Q fever to various animals*

No. exposed to…	Q fever	No Q fever
Cats	3	5
Cat litter	1 (Sept 24)	1 (early Sept)
Dogs	3	6
Sheep	0	0
Cattle	0	1
Horses	0	0
Rabbits	0	1 (litter Oct 1)
Goats	0	0

Five family contacts of the 16 cases were ill during the period 7-15 October, amongst whom were the cat owner's wife and son; both seroconverted to *C. burnetii* phase I and phase II antigens. Blood samples from family members of the other employees who were ill were all negative for antibodies to *C. burnetii*. One family contact, a 10-month-old boy was not tested.

There were no activities in which all of the employees participated in the month prior to the onset of the outbreak. On 29 September, 20 of the 32 employees attended a golf tournament however there was no difference in the number of cases and non-cases attending this event. We interviewed 22 golfers who played on the same day and at the same course as the employees of the trucking company. Blood samples were obtained from 4 of these golfers who suffered a flu-like illness within the incubation period for Q fever, 3 had no antibody to *C. burnetii*, 1 had a low titre of 16 to phase II antigen.

None of the people interviewed who lived in the immediate vicinity of the plant were ill. Also those interviewed did not have cats that had given birth to kittens.

During the time that this outbreak was ongoing three individuals from the community in which the trucking plant was located, had radiographic evidence of pneumonia. We obtained acute and convalescent serum samples from these individuals and none seroconverted to *C. burnetii.*

DISCUSSION

We conclude that this outbreak of Q fever resulted from aerosols generated from the contaminated clothing of the owner of the infected cat. He contaminated his clothing when he fed the kittens each morning before he came to work. The differential attack rate between the upstairs employees where the cat owner spent most of his time and that among the downstairs employees lends support to our hypothesis. Furthermore, the first case of Q fever among the employees in the plant occurred in the person who worked in an enclosed area with the cat owner. The only cases of Q fever among family members of the employees were the wife and son of the owner of the infected cat. In a previous study of cat-associated Q fever we found that the incubation period for this illness ranged from 4-30 days with the onset in most cases about 2 weeks after exposure (Marrie *et al.* 1988). In the current outbreak the incubation period varied from 12-21 days.

There are several reports of Q fever following indirect exposure to infected animals. An outbreak in Switzerland involved over 350 persons who lived along the road where sheep travelled from mountain pastures (Dupuis, 1987). An outbreak of Q fever occurred among residents in urban Wales living near a road over which farm vehicles carrying straw, manure or dust contaminated with *C. burnetii* travelled (Salmon *et al.* 1982). *C. burnetii* can withstand very harsh environmental conditions (Christie, 1980) probably by virtue of its ability to form spores (McCaul & Williams, 1981). As such it is ideally suited for spread by the methods outlined above. It is also an extremely infectious pathogen and it has been suggested that a single microorganism may be sufficient to cause disease (Tigertt & Benenson, 1961).

The low attack rate among the downstairs employees is compelling evidence against the possibility that trucks with contaminated straw or dust were the source of the outbreak. The 10 unaffected downstairs employees were not protected by pre-existing antibody. All were seronegative and remained so during the 9-week period of follow up.

Oliphant *et al.* (1949) reported that two laundry workers developed Q fever after they handled laundry that had come from a laboratory that worked with C. *burnetii.* Marmion & Stoker (1956) described an outbreak of Q fever among actors, that they attributed to contact with the contaminated clothing of a shepherd who had a role in the play that they were performing. Johnson (1966) also invokes contaminated clothing as the possible means whereby a sheep shearer and an abbatoir worker infected their children. Edlinger (1987) reported the case of a French officer returned from French Guyana with his wife and two children to his home in Normandy. He became ill with Q fever, and 1 month later 5 of the 6 members of his family developed this infection.

In a previous study (Marrie *et al.* 1985) we found that 24% of 216 cats studied had antibodies to phase II *C. burnetii* antigen and 6% had antibodies to phase

I antigen. We have isolated *C. burnetii* from two cats epidemiologically associated with outbreaks of Q fever (Marrie *et al.* 1988; Marrie, T. J. and Williams, J. C., unpublished observations.) Cats experimentally infected with *C. burnetii* via the subcutaneous route became febrile and lethargic for 3 days whereas cats infected via the oral route did not become ill (Gillespie & Baker, 1952). Following laboratory-induced infection *C. burnetii* has been demonstrated in the blood of cats for up to 1 month and in the urine for 2 months (Gillespie & Baker, 1952). It is not known if the organism is excreted from a cat's uterus following natural infection.

The clinical features of this outbreak of Q fever are similar to those that we have observed in other outbreaks (Marrie *et al.* 1988) except for a higher incidence of diarrhoea than is usual in our experience (Marrie *et al.* 1988).

Both of the patients hospitalized at our hospital had elevated amylase levels. To our knowledge pancreatitis had not been previously reported as a manifestation of Q fever.

Several of our patients had microscopic haematuria a feature that has been described previously in cat-associated Q fever (Kosatsky, 1984). *C. burnetii* was isolated from the urine of three of the patients described by Derrick (1937).

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