Milk-borne campylobacter infection

D A ROBINSON, DENNIS M JONES

Abstract
The common factor in 13 recent outbreaks of Campylobacter jejuni enteritis was the consumption of unpasteurised or incompletely pasteurised milk. C jejuni is a common commensal in the alimentary tract of milking cows, but it is not clear how the milk becomes contaminated with the organism. Pasteurisation will readily eliminate the organism from milk. In England and Wales 3% of milk retailed is still unpasteurised, and in the light of these findings it is suggested that only pasteurised milk should be sold to the public.

Introduction
Unpasteurised milk is the major vehicle of transmission that has been identified in human outbreaks of Campylobacter jejuni infection. This review is intended to summarise the known facts on this aspect of campylobacter infection and to give substance to a suggestion that increased pressure should be applied to reduce the general distribution of unpasteurised milk.

Milk-associated outbreaks
Since 1978 in Britain milk has been implicated in at least 13 documented outbreaks of campylobacter infection of substantial size. These are summarised below.

MINEHEAD, SOMERSET. MARCH 1978*
About 100 cases occurred when excessive snowfall prevented pasteurisation of milk from a healthy herd. No bacteriological confirmation was obtained of infection of the herd or contamination of the milk, but the epidemiological evidence relating the cases to the consumption of unpasteurised milk was strongly suggestive.

ARNSIDE, LANCASHIRE. OCTOBER 1978*
The 64 people of all ages affected over a period of six days had drunk unpasteurised milk from the same farm. There were no cases among drinkers of pasteurised milk. All the human strains tested were serologically identical. A campylobacter of the same serotype and biotype was isolated from two cows in the suspected herd but not from milk filters examined a week after the onset of the first case. Campylobacters of other serotypes were also isolated from other cows in the herd. About 10% of the herd was found to be excreting campylobacters.

BRADFORD, YORKSHIRE. NOVEMBER 1978*
The 16 people affected had drunk unpasteurised milk from the same farm. There had been some mild scouring in the suspected herd. All the human strains tested were of the same serotype. A campylobacter was isolated from a milk filter from the farm.
ABERDEEN. JANUARY 1979

In this outbreak 616 people had consumed milk from the same dairy, which habitually pasteurised all its output. A power failure had resulted in the milk on two days being distributed unpasteurised. There was no illness in the herd. Campylobacter was isolated from a milk filter but not from bottled milk. All six human strains tested were of the same serotype.

LUTON, BEDFORDSHIRE. MARCH 1979 (A T Willis, personal communication)

In an extensive outbreak affecting about 3500 individuals (mainly children under 8 years old) a statistical association was shown between drinking free school milk and the development of symptoms. Pasteurisation of milk from the large dairy in question had failed in the week before the outbreak. There was no other linking factor. At least two different serotypes were isolated from patients, and C. jejuni of the predominant serotype was also isolated from two workers in the dairy.

LINCOLN. APRIL 1979

An unknown number of patients, probably over 75, were affected during a very cold spell of weather that hampered pasteurisation. The outbreak included cases in a community that normally received pasteurised milk but which had recently started taking churned unpasteurised milk. C. jejuni was isolated from 33 symptomatic patients. No campylobacters were found in milk filters tested more than two weeks after the event.

GILLING WEST, NORTH YORKSHIRE. MARCH 1979 (M Barnham, personal communication)

Inhabitants of a village, cut off by severe weather from their usual supply of pasteurised milk, turned to unpasteurised supplies from a local farm. At least 13 people became ill. Campylobacter was isolated from the faeces of nine of 11 patients tested. Milk filters and milk were examined unsuccessfully two and a half weeks after the incident.

LONG SUTTON, HAMPSHIRE. JUNE 1979

An outbreak of four confirmed infections but many symptomatic cases occurred in a school of 440 boys. The school was supplied with unpasteurised milk from its own attached farm. Campylobacter was isolated from a milk filter.

MAIDSTONE. AUGUST 1979

Fourteen residents in an institution became ill over a two-week period. C. jejuni was isolated from the faeces of all eight patients tested. Many common factors were considered but grossly faecally contaminated milk (sent back on one occasion because it was "dirty") was considered the most likely source. Three of three strains examined were found to be of identical serotype. This was the same serotype as that found in the Asnside outbreak.

CHELMSFORD, ESSEX. JANUARY 1980

At least 75 of 300 resident students in an agricultural college were affected over a period of three weeks. Questionnaires showed a statistical correlation between the development of symptoms and the quantity of milk regularly consumed. The milk was unpasteurised and supplied in churns from a neighbouring model farm where there had been a greater than average increase in milk cell counts in November and December 1979. A similar but uninvestigated outbreak of gastroenteritis had occurred during November 1979. A serological survey found that 77% of the sample of 46 students had complement-fixing antibodies to C. jejuni. This compares with the 5% found in a collection of normal antenatal sera from a mixed rural and urban population in East Anglia. Two different serotypes were found among the eight strains examined: 60% of the sample were found to have bactericidal antibodies to one strain and 9% to the other. Only 30% of the sample had had symptoms recently, and the serological findings suggest a high level of infection in this community, which habitually consumed unpasteurised milk.

KENT. MARCH 1980

About 30 girls in a boarding school presented over a period of about 10 days with abdominal pains and diarrhoea. The school received unpasteurised milk in churns from a neighbouring farm and was the only community in that part of the county to take such milk. No cases had occurred in the population outside the school. Three cows in the herd had had mastitis in the previous month, one of them on two occasions. Campylobacter was not found in milk or milk filters examined two weeks after the start of the episode.

WHITTEHAVEN, CUMBRIA. MARCH 1980

A sudden increase in campylobacter gastroenteritis was noticed in a housing estate, where at least 40 people had symptoms over a two-week period. House-to-house questionnaires and a serological survey showed evidence of a significant excess of infection among the 60% of the population who purchased unpasteurised milk compared with those who drank pasteurised milk. No other associations were found. Seventeen (12%) of the 143 residents of the estate investigated were found to have complement-fixing antibody to C. jejuni. Only one of these regularly consumed pasteurised milk. The conclusions drawn from these results were that, although unpasteurised milk from one of the local producer/retailers was probably responsible for the immediate outbreak, the regular consumption of unpasteurised milk from any source was a risk factor for campylobacter infection. Two different serotypes were detected among six human isolates studied.

BLACKBURN, LANCASTASHIRE. APRIL 1980

A boy of 2 years and his mother presented with campylobacter enteritis. She was a child-minder, and all her five charges were found to be infected. None of the family contacts of the children were infected, and the unpasteurised milk supplied to the child-minder’s household was suspected. Milk filters were tested ten days after the event and found to be negative. Support for the incrimination of milk was supplied by cases occurring in three other unrelated families supplied by the same farm. All isolates were of the same serotype. No secondary spread occurred within any of the children’s families.

Campylobacter infection in cows

The demonstration of milk as a vehicle of human infection with campylobacter has raised several questions concerning the role of the cow in the epidemiology of this infection.

It has been recognised for a long time that cows may be infected with C. jejuni and C. coli and that there may or may not be associated symptoms. An important determining factor in the occurrence of symptoms appears to be the age of the animal (D J Taylor, personal communication). Nothing, however, was known of the long-term significance or behaviour of campylobacter infection in herds, and a study was carried out in 1979-80 to provide some of the answers.

Two milking herds, one of 28 head and the other of 40 head, were studied for 12 and six months respectively. Rectal swabs were taken fortnightly from all members of both herds and weekly from animals found to be positive. Milk filters were examined three times a week.

The principal findings were that campylobacters can produce symptomless and persistent infection or colonisation in milking herds without any detectable contamination of the milk, that the prevalence of faecal excretion may vary considerably with the season; and that the infection may be more easily established in young animals, possibly persisting into adulthood. In addition it was found that several different campylobacter serotypes could be present within a herd with little evidence of cross-infection between animals.

Mechanism of contamination of milk

The route by which contamination of milk occurs is not known. From some outbreaks investigated the period of contamination may
be as short as one milking, which may account for the difficulties experienced in isolating the organism from milk or milk filters after outbreaks have occurred. The possible mechanisms include direct faecal contamination during or after milking, which implies a low infecting dose or the ability of the organism to multiply in milk. An alternative is suggested by the demonstration of artificially induced campylobacter metritis in milking cows. A naturally occurring metritis has not yet been shown (R W A Park, personal communication). Work is in progress to examine both of these hypotheses.

Survival and multiplication in milk

Studies on the survival and multiplication of C jejuni in milk have shown that:
1. Multiplication does not occur under either laboratory or normal household conditions.4 (S Waterman, personal communication).
2. The organism can survive for at least 24 hours at room temperature and for at least three weeks at 4°C.14
3. C jejuni does not survive pasteurisation temperatures (J M Watkinson, personal communication).

Discussion

The great majority of human isolations of campylobacter are from cases commonly described as “sporadic” or “isolated.” Only rarely is the source of these infections convincingly shown. In England and Wales unpasteurised milk comprises 3% of the total quantity of milk retailed, and 70% of this is sold in the northern counties of England. Because of the geographical distribution of unpasteurised milk sales in the country, it is unlikely that a large proportion of sporadic cases of campylobacter enteritis infection are milk associated. In outbreaks of campylobacter infection, however, the consumption of unpasteurised milk by the individuals affected is a regular finding. In several instances the outbreak has occurred in the only group of raw milk drinkers within a large population drinking pasteurised milk. Other outbreaks have been attributed to uncooked or poorly cooked chicken,6 to contaminated water,15 and to food-borne or person-to-person spread among nursery school children.16 17

The importance of milk as a vehicle lies in its wide and rapid distribution. As in the Luton outbreak many individuals can be infected within a short period from a single source.

Bovine tuberculosis is now uncommon, and there were only 36 cases of brucellosis, which is now rarely associated with milk, recorded in 1979. At present, the main accepted risk from unpasteurised milk is salmonellosis. In the past five years there have been 27 reported outbreaks of milk-borne salmonellosis in England and Wales, accounting between them for 413 cases. This may be compared with 12 milk-borne outbreaks of campylobacter infection detected in the past two-and-a-half years, which have caused at least 3000 cases.

If the campylobacter risk from milk was confined to outbreaks its importance might be considered to be limited, but the serological surveys in Chelmsford and Whitehaven suggest that communities which regularly take raw milk are exposed to a higher than normal level of infection with campylobacter, irrespective of whether detectable outbreaks occur or whether the cases are merely “sporadic.” The methods used by most of the dairy farms concerned in the reported outbreaks were blameless. C jejuni can be found in many, if not all, dairy herds, but as it causes no symptoms in cows and as the mechanism of contamination of milk is so far not known, it is difficult to see what preventive action could be taken by farmers or their veterinary advisers.

Pasteurisation, if correctly carried out, will effectively eliminate campylobacter from milk, and it seems reasonable to suggest that campylobacter infection should be put forward as a further reason to press for more widespread, if not universal, pasteurisation of milk for distribution to the general public.

References


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