Review of Evidence for Zoonotic Listeriosis

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(Received for publication October 29, 1986)

ABSTRACT

Foodborne transmission, especially by milk and milk products and raw vegetables, appears to be the major means of zoonotic transmission of listeriosis. Four major foodborne outbreaks of listeriosis were reported in the United States and Canada 1979-1985. Implicated foods were cabbage, pasteurized milk, and Mexican-style soft cheese. Circumstances of the outbreaks and the implicated foods support the concept of zoonotic transmission of listeriosis.

Listeriosis traditionally has been considered a zoonosis for the following reasons. (Let us define “Zoonosis” as “an infection or an infectious disease transmissible under natural conditions from vertebrate animals to man” (7), and “animals” as nonhuman vertebrate animals.) It was first described by Murray in 1926 in a laboratory colony of rabbits (40), and was not described in a human patient until several years later (42). Since then Listeria monocytogenes has been isolated from many mammalian and avian species (25) and is now considered ubiquitous in nature (64). Early studies showed little difference in biological and biochemical properties of L. monocytogenes strains from human and animal sources (56). In domestic animals, listeriosis is recognized most frequently in cattle and sheep (8) and is associated with septicaemia, encephalitis and abortion—signs similar to those commonly seen in human cases.

EVIDENCE FOR FOODBORNE ZOONOTIC TRANSMISSION

For many years it has been assumed that humans usually were infected by ingestion and that listeriosis was primarily a foodborne disease (3,8,9,49,52,53). In the last decade, investigations of L. monocytogenes outbreaks in the United States and Canada have provided additional evidence for the foodborne transmission of listeriosis, and for its animal origin.

Transmission by milk

Early evidence for foodborne transmission includes anecdotal reports from Germany of mothers from rural areas who had histories of drinking raw milk (25). Potel is credited with the first description of foodborne listeriosis in humans with direct links to animals (48,52). He isolated L. monocytogenes from a cow with L. monocytogenes mastitis and from stillborn twins of a woman who had ingested raw milk from this animal before delivery. The same serotype of L. monocytogenes was recovered from the cow and the patient (48). In a large outbreak in Halle, Germany, 1949-1957, it was believed that many human cases were due to ingestion of raw milk (49,56).

Milk and cheese have been implicated in two recent outbreaks. Pasteurized milk was implicated epidemiologically in an outbreak of listeriosis in Massachusetts between June and August, 1983, during which 7 cases occurred in infants or fetuses and 42 in immunocompromised adults (19). Several case-control studies, one with cases and controls matched for neighborhood of residence and the other for underlying disease, strongly associated consumption of a specific brand of pasteurized whole or 2% milk, but not skim milk, with disease. Attempts to isolate L. monocytogenes from the pasteurized milk were unsuccessful. Listeriosis in dairy cows was reported to have occurred on some of the farms which supplied the implicated milk; multiple serotypes of L. monocytogenes, including 4b, the outbreak serotype, were recovered from 15 of 124 samples of raw milk.
(12%) and from 2 of 14 milk filters (14%) from some of these farms, but the isolates were of a different phage type than the epidemic isolate. Inspections at the implicated pasteurizing plant revealed no evidence of improper pasteurization. Postpasteurization contamination was considered unlikely because both whole and skim milk were produced with the same equipment on the same day. The authors speculated that a few organisms may have survived the pasteurization process by being protected in their intracellular location, and noted that in the implicated pasteurization plant clarification (removal of leukocytes by a centrifugal filtering process) was not practiced.

Another outbreak of listeriosis caused by \textit{L. monocytogenes} serotype 4b occurred in the Los Angeles area between January and August, 1985, in which 142 cases and 47 deaths were reported (M. Linnan, L. Mascola, D. Xiao et al., personal communication). Eighty-eight (62%) cases occurred in pregnant Hispanics. Case-control studies revealed that mothers with listeriosis were more likely to have consumed a certain brand of Mexican-style soft cheese—manufactured in Los Angeles—than age-matched controls. Hispanic women who had delivered at the same hospital (63). \textit{L. monocytogenes}, serotype 4b, of the outbreak phage type, was recovered from unopened packages of cheese. Audit of the cheese plant revealed no evidence of improper pasteurization. Postpasteurization contamination was considered unlikely because both whole and skim milk were produced with the same equipment on the same day. The authors speculated that a few organisms may have survived the pasteurization process by being protected in their intracellular location, and noted that in the implicated pasteurization plant clarification (removal of leukocytes by a centrifugal filtering process) was not practiced.

In a European study, when unpasteurized milk containing \textit{Listeria} in symptomatic and asymptomatic cattle was well-documented, as discussed below. Another plausible way for \textit{Listeria} to contaminate milk is to be shed within the milk itself. This is known to occur, and is a property which \textit{L. monocytogenes} shares with the brucellae and \textit{Mycobacterium bovis}, all facultative intracellular parasites (5). Shedding of \textit{L. monocytogenes} in milk as a consequence of \textit{Listeria} mastitis has been documented although the condition is rarely recognized (11,15,18,22,61,65). \textit{Listeria} also has been recovered from milk after \textit{Listeria}-associated abortion (13,19,44,46,49,60,65). In one study the organism was isolated from milk and uterine discharge up to 13 d after abortion (44). In another study 16% of dairy cows were found to shed \textit{Listeria} in their milk after abortion (49). In a study in the Netherlands, 72 (7.7%) of 938 quarter milk samples from cattle aborting from \textit{L. monocytogenes} were initially found to contain \textit{L. monocytogenes} (13). Following cold enrichment at 5°C, 40% of these samples were positive, indicating that \textit{L. monocytogenes} was present originally in small numbers in the milk (13). Isolation of \textit{Listeria} from milk of clinically normal cows has been reported often (18,22,31,32,36,55,58,61). In a study in Yugoslavia, \textit{L. monocytogenes} was isolated from milk of 3.2% of 845 clinically normal cows on 7 farms on which listeriosis had been previously diagnosed; prevalence per farm ranged from 0.85-5.34% (36). In another study, milk from 10 of 1004 cattle in 8 of 25 herds yielded \textit{Listeria} (55). Shedding was intermittent and could continue for at least 12 months and into succeeding lactation periods (31,61). Shedding of \textit{L. monocytogenes} in milk of sheep (26,27) and goats (38) has also been reported.

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The evidence that listeriosis may have been transmitted by pasteurized milk in the 1983 Massachusetts outbreak raised questions about the effectiveness of current pasteurization regulations (20) [71.7°C (161°F) for 15 s]. There are apparently conflicting studies on the effectiveness of pasteurization against \textit{L. monocytogenes}. Potel reported that \textit{L. monocytogenes} survived 80°C for 5 min (47). In another study it was found that if over 5 x 10^6 organisms/ml were present in milk before pasteurization (61.7°C for 30 min), the organisms could be recovered after pasteurization (6). Seeliger (57) believed that only temperatures above 85°C for 40 s were safe for \textit{Listeria}, while Donker-Voet (15) believed that 66.3°C for 15 s was satisfactory for naturally contaminated milk. Stajner (62) reported that at a concentration of 5 x 10^6/ml of milk, \textit{L. monocytogenes} did not survive pasteurization at 85°C for 15 s, but at temperatures below that a few survived. This underscores the fact that concentration of the organism is very important in these studies. Recent studies using the \textit{L. monocytogenes} isolate from the 1983 Massachusetts outbreak indicated that 1 x 10^6 \textit{L. monocytogenes} cells failed to survive pasteurization at
71.7°C for 15 s (10). In another study, milk from cows inoculated with \textit{L. monocytogenes} was pooled for 2 to 4 d then heated at 71.1 to 73.9°C for 16.4 s or at 76.4 to 77.8°C for 15.4 s in a high-temperature, short-time heat exchanger pasteurization unit. \textit{L. monocytogenes} was isolated from milk after heat treatment in 6 of 9 pasteurization trials done at 71.7 to 73.9°C and in none of 3 trials done at 76.4 to 77.8°C (16). Clearly, additional studies (several are now in progress) are warranted to resolve apparent discrepancies and to address the question of intraleukocytic protection from pasteurization, by use of milk from naturally infected animals for pasteurization tests.

Fecal contamination of vegetables

Two other recent foodborne outbreaks of listeriosis may have been associated with consumption of contaminated vegetables. Contaminated vegetables had been suggested as a possible source of human listeriosis even before these outbreaks (8).

In September and October 1979 an apparently common-source outbreak of listeriosis caused by \textit{L. monocytogenes} serotype 4b occurred in patients in 8 hospitals in the Boston area (30). The most likely risk factor seemed to be consumption of raw celery, tomatoes and lettuce. The investigators speculated that the raw vegetables may have been contaminated with \textit{Listeria}, which was able to survive ingestion because of gastric acid neutralization by antacids or cimetidine.

In 1981 a large outbreak occurred in the Maritime Provinces of Canada. Between March and September, 34 perinatal cases and 7 adult cases were identified (54). A case-control study identified consumption of coleslaw as a risk factor, and coleslaw, but no other food items, from the refrigerator of a patient yielded \textit{L. monocytogenes} serotype 4b, the outbreak serotype. \textit{L. monocytogenes} was also isolated from unopened packages of coleslaw purchased at two Halifax area supermarkets. Human and cabbage \textit{L. monocytogenes} 4b isolates were of the same phage type (4,52). By matching a list of livestock cases of listeriosis with a list of commercial cabbage growers, a farm was identified on which 2 sheep had died of listeriosis, one in 1979 and one in March, 1981 (52,54). Cabbage was grown in fields fertilized with both composted and raw sheep manure. The cabbage crop was kept in cold storage through the winter and early spring, when it was shipped to the implicated coleslaw company. No \textit{L. monocytogenes} isolates from cabbage or sheep were available for study. The authors speculated that contamination of cabbage occurred on the farm and that, under conditions of prolonged cold storage and the absence of cooking, adequate numbers of \textit{Listeria} were able to be ingested by persons at risk of developing clinical listeriosis.

Vegetables may become contaminated with animal feces. Fecal carriage of \textit{L. monocytogenes} by cattle, sheep and other animals is well-documented and has been reviewed in the literature (2,8,9,28); in a Dutch study, \textit{Listeria} was isolated from the feces of 24% of 219 cows aborting because of \textit{Listeria}, 14% of 85 cattle with \textit{Listeria} encephalitis, 7% of 622 healthy cattle from herds with listeriosis, and 2% of 120 cattle from herds without listeriosis (9,12). Contamination of vegetables can occur with the use of manure from carrier animals; this is especially important for vegetables such as lettuce and cabbage that are held under conditions of cold storage and are not cooked, conditions which favor survival and multiplication of \textit{L. monocytogenes} (54).

Transmission by contaminated meat or eggs not documented

Foodborne transmission of \textit{Listeria} by consumption of contaminated meat has been repeatedly considered but never documented (33,56), although \textit{Listeria} has been recovered from red meat (17) and poultry (14,17,21,37) for human consumption. Likewise, transmission of \textit{Listeria} by eggs or egg products has never been documented (23,33,56), although presence of necrotic lesions in oviducts of some hens with listeriosis suggested the possibility that the eggs might contain \textit{Listeria} (24). There was one report of fatal \textit{Listeria} meningitis of a man who worked in an egg products factory (34).

ARGUMENTS AGAINST ZOONOTIC TRANSMISSION

In many human cases of listeriosis there was either no indication of source of infection, or there existed evidence of human-to-human transmission (49,52). In very few cases was there clear evidence of transmission from animals or animal contact (9,35). The few cases for which there was good evidence of zoonotic transmission—e.g., veterinarians with cutaneous lesions—seemed atypical (35). In the United States the epidemiology of neonatal listeriosis remained unclear; there appeared to be no association between the disease in animals and neonatal listeriosis (1).

In addition, there was conflicting evidence of patterns of listeriosis in urban vs. rural human populations (9,49). There appeared to be no association between the seasonality of human and animal infections—winter and spring are often cited as seasons of greatest listeriosis incidence in livestock, but many countries reported summer and fall as seasons of high incidence in humans (49). Some authors found no correlation between serotypes in humans and animals (49).

CONCLUSIONS AND COMMENTS

Outbreaks of the last decade have offered additional evidence of foodborne transmission of \textit{Listeria} (25,49,52,53). The evidence, while not conclusive, clearly supports the thesis that zoonotic transmission of \textit{Listeria} is not an infrequent event. Although some human outbreaks were associated with clinical cases of listeriosis in livestock, the healthy carrier animal remains important as a source of the organism (8,15). At present there are
no good tools to identify these carrier animals; identification by bacterial isolation is impractical, and serological tests are not entirely satisfactory. Such new techniques as DNA probes offer promise for future identification of infected cows and affected farms. In the field of food safety, pasteurization studies are necessary, as is more attention to procedures to assure the safety of such newly-introduced dairy products as Mexican-style soft cheeses and other foods likely to be contaminated and kept in cold storage for prolonged periods without subsequent cooking.

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ACKNOWLEDGMENT

Financial assistance provided by the Department of Atomic Energy, Government of India is gratefully acknowledged.

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JOURNAL OF FOOD PROTECTION. VOL. 50, MAY 1987