Outbreak of Trichinosis in Ontario Secondary to the Ingestion of Wild Boar Meat

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Trichinosis, an infection with a species of the tissue-invading nematode Trichinella, has been in significant decline in North America in recent years due to the implementation of public health legislation. As a consequence, health care providers and public health officials are less likely to recognize and be able to manage outbreaks which occur locally. We report the first outbreak of trichinosis in Ontario since 1977.1 It resulted from the consumption of cold smoked wild boar meat.

MATERIAL METHODS

Case definition
For the purposes of this study, an individual was considered to have trichinosis if he or she had recently consumed wild boar meat and had 1) positive serology for Trichinella or 2) an abnormal creatine phosphokinase plus three out of four of the following clinical findings: fever greater than 38°C, an eosinophil count equal to or greater than 0.5 x 10^9/L, periorbital edema or myalgia.

Data collection
All patients suspected to have trichinosis were seen at the Tropical Disease Unit, The Toronto Hospital or at a designated clinic in Orangeville, Ontario. Before undergoing a physical examination, patients were asked to complete a standardized questionnaire detailing dates of meat consumption and the time course and characteristics of their symptoms. Serum was collected and sent to the Ontario Ministry of Health Laboratory, Toronto, where the Trichinella complement fixation test (CF) was performed and to the National Reference Centre for Parasitology (Serology) for diagnosis by the ELISA method. An ELISA titre equal to or greater than 1:128 or any titre by CF was considered to be diagnostic of trichinosis.

RESULTS

Outbreak description and investigation
On January 8, 1993, three friends living in the same household in Toronto presented to their family doctor with fever, myalgia, periorbital edema and eosinophilia, findings which were recognized as being compatible with trichinosis. All three had eaten uncooked wild boar meat acquired at a food market in downtown Toronto. On January 13, 1993, these patients were seen in the Tropical Disease Unit, The Toronto Hospital and the suspicion of an outbreak was reported to the City of Toronto Public Health Unit. Within hours of this report, public health inspectors were in contact with the vendor of the boar meat at the market where the meat was sold. During this investigation one vendor was listed as selling wild boar meat from a farm (A) in Dufferin County. This farmer kept 15 free-range wild boar and supplemented their diet with uncooked meat scraps from a local slaughterhouse. Wild boar meat from this farm had been sold in Toronto for two years prior to this outbreak. In the fall of 1992, two wild boar were slaughtered and four hams were cold smoked for one week. Sixty to seventy half-pound
trays of ham, accounting for approximately 75% of the meat, were sold in Toronto. The other 25% was consumed by the farmer’s family, friends and local residents.

On January 14, 1993, the Wellington-Dufferin-Guelph Health Unit (WDGHU) contacted this farmer and the abattoirs where the meat was processed. The same day a sample of smoked sausage was obtained from farm A and submitted to the Central Public Health Laboratory, Ministry of Health. The remaining wild boar meat products on the farm were detained. The following day, personnel from Agriculture and Agri-Food Canada (AAFC) visited the farm and quarantined the wild boar herd and associated meat. The results of the slaughterhouse investigation showed that sausage from a wild boar was cold-smoked for 24 hours with hardwood sawdust without temperature monitoring. The farmer advised the Health Unit that no further cooking was undertaken and that his meat was sold to the Organic Farmer’s Market and several restaurants in Toronto. The City of Toronto’s Department of Public Health was immediately given the list of restaurants to which the meat was sold. It was not clear what instructions concerning further cooking were given to the farmer from the slaughterhouse or to the purchaser from the farmer.

On January 15, 1993, the Chief Medical Officer of Health of Ontario issued a Public Health Advisory to the media and local health units. It was published in two local newspapers on January 17th and January 18th. At the same time, the Medical Officer of Health for Wellington-Dufferin-Guelph County, where the contaminated meat originated, issued a Public Health Alert to all physicians in the area. All individuals known to have bought wild boar meat from the farmer were contacted and instructed to seek medical attention. However, since approximately 75% of the meat was sold in Toronto, most of the buyers could not be traced and case finding was dependent on responses to the medical advisory.

On January 18, 1993, a member of the public reported that he had purchased smoked wild boar meat from the same Toronto market but from a different farmer (B) also located in Dufferin County. On the day this report was received, farm B was investigated by two public health inspectors and an AAFC inspector who quarantined the farm. Once again, meat samples were sent to the Central Public Health Laboratory. Although initial testing of the meat samples in this laboratory was unrewarding, confirmatory samples from both farms sent to the AAFC laboratory in Saskatoon were found on January 27, 1993 to be positive in high concentrations for *T. spiralis* using the pepsin digestion test. Subsequently, wild boar herds from both farms were destroyed and sent for rendering.

One week after the outbreak was reported, the Medical Officer of the WDGHU issued a second trichinosis update to doctors in the Dufferin area along with a comprehensive trichinosis investigation questionnaire for any suspected cases. The questionnaire was produced in collaboration with physicians investigating Toronto cases referred to the Tropical Disease Unit, The Toronto Hospital. In addition, a local physician in Dufferin County organized a special clinic to assess suspected cases which came forward as a result of the media press releases. Eleven suspected cases were seen during the one-day clinic. In total, 12 cases were identified in the Dufferin area, mostly family members and acquaintances of the two farmers as well as farmer B and his wife.

**Clinical findings**

Between January and March 1993, 24 cases of trichinosis were identified in individuals who ingested wild boar meat between November 1992 and January 1993. Fifteen other individuals who had consumed the infected meat were identified but were found to be asymptomatic. Three of these persons had normal hematology and biochemistry and had no *Trichinella* antibodies detected; the remaining 12 individuals refused further investigations. Of the 24 cases of trichinosis, 21 were symptomatic. Approximately equal numbers of men and women were infected (46% and 54% respectively). Thirteen percent of patients were under the age of 30, 58% were between the ages of 30 and 60 and 29% were over the age of 60. The symptoms of the 21 patients are summarized in Table I. Most patients had classical symptoms of trichinosis with fever, myalgia and periorbital edema. Four patients had diarrhea within the first week following ingestion of the meat. Prolonged diarrhea was noted in 10 patients of whom only 7 had concomitant myalgia.

The onset of symptoms occurred between late November 1992 and early January 1993 with 76% occurring in the second half of December. The incubation period ranged from 8 to 31 days with a mean of 21 days. No significant association was found between the length of incubation period and the age and gender of the patients or the severity of illness. The duration of symptoms ranged from 3 days to more than 2 months. The symptoms of two patients resolved spontaneously and then recurred in a milder form. Two patients were hospitalized—one with severe diarrhea, and the other with myalgia and fever—before the diagnosis was determined. There were no documented cases with severe trichinosis in this outbreak.

Laboratory results were available for 23 of the 24 patients. Eosinophil counts ranged between 0.13 x 10^9/L and 10.82 x 10^9/L with a mean of 3.06 x 10^9/L (normal <0.5 x 10^9/L). Creatine phosphokinase ranged between 77 U/L and 1180 U/L with a mean of 428.6 U/L. The *Trichinella* ELISA test was positive in 7 out of 18 tested (39%) and complement fixation was positive in 7 out of 19 tested (37%). One or both tests were positive in 55% of patients tested (Table II).
Twenty-one of the patients were offered treatment with albendazole, 400 mg by mouth twice daily for 14 days. Fifteen patients initially accepted treatment; however two patients did not take the drug. Only five of the patients who completed therapy returned for follow-up. Of these, four patients noted that their symptoms worsened initially but resolved completely within the next 10 days. However, three of them experienced a mild recurrence of their symptoms (i.e., myalgia and periorbital edema) two weeks after completion of therapy. The fifth patient remained asymptomatic during and after treatment.

DISCUSSION

Human trichinosis, an infection with a world-wide distribution, is a zoonosis caused by one of five different species of Trichinella. In North America and Europe, T. spiralis is found in a variety of carnivorous animals of which bear, pigs, wild boar, and walrus are the most important sources of human infection.

Trichinosis is acquired by the ingestion of raw or inadequately cooked meat products containing encysted larvae of the parasite. Following gastric digestion of the cyst wall, larvae are released and mature into adults which penetrate the mucosa of the small intestine. After mating, male worms are dislodged and expelled; female worms produce and release large numbers of larvae in the mucosa for 4 to 16 weeks. Within 2 to 3 weeks of the infection, larvae migrate via the lymphatics and blood stream to striated muscle where they encyst. Before encystment is complete some larvae are destroyed by the host’s inflammatory response. Encysted larvae which survive remain viable and infectious for several years. Calcification of living and dead cysts usually begins within six months to one year.

Symptoms of trichinosis may begin when adult worms first penetrate the intestinal mucosa. Although this intestinal phase is usually asymptomatic, patients may develop anorexia, nausea, diarrhea and abdominal pain. These symptoms rarely last more than a week; however, several recent reports of trichinosis outbreaks in the Canadian Arctic describe prolonged diarrhea without significant muscle symptoms. It was interesting to note that 10 of our patients experienced prolonged diarrhea, suggesting the possibility that they may have been previously infected.

The triad of myalgia, fever and periorbital edema, classical symptoms of trichinosis, correspond to an allergic response to larval migration and encystment in striated muscle. As expected, over 70% of our patients experienced those symptoms. None of our patients experienced the neurological, cardiac or respiratory complications associated with severe disease. The mean incubation period in our series, 21 days, is comparable to that noted in previous reports from North America. Several of our patients remained symptomatic for several months. Some reports have described chronic myalgia for as long as 3 to 42 years following infection. However, a recent, prospective, controlled study found insufficient evidence to conclude that chronic trichinosis exists as a distinct entity. One of the limitations of a study such as ours is recall bias, in that much of our data were obtained from patients several weeks after they had ingested the infected boar meat.

Based on previous studies indicating that as few as 0.1% of infected individuals become symptomatic, we suspect that there were many more persons who were infected but asymptomatic or were symptomatic but not diagnosed as having the disease. If all 35 pounds of meat were consumed, there may have been as many as 140 cases of trichinosis from the meat sold at the Toronto Farmer’s Market, assuming that each person ate approximately 0.125 kg.

The laboratory diagnosis of trichinosis is often suspected by the presence of eosinophilia and/or an elevation of creatine phosphokinase (CPK). In our study, eosinophilia and elevations in muscle enzyme levels were noted in 86% (20/23) and 89% (17/19) of patients respectively. On the other hand, in only 41% (7/18) were Trichinella antibodies detected by the ELISA test which has been shown to have a sensitivity of 100% within two months of infection. The complement fixation test added little in that it was negative in the majority of cases and produced only one positive result (case 11) when the ELISA test was negative. These results confirm previous findings that Trichinella serology is often negative in the early stages of infection. When trichinosis is suspected and antibodies are not detected initially, serology should be repeated at a later date or a muscle biopsy should be performed.

### TABLE II

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* ≥ 1/128 was considered positive
† any titre was considered positive

NOTE: The triad of myalgia, fever and periorbital edema, classical symptoms of trichinosis, correspond to an allergic response to larval migration and encystment in striated muscle. As expected, over 70% of our patients experienced those symptoms. None of our patients experienced the neurological, cardiac or respiratory complications associated with severe disease. The mean incubation period in our series, 21 days, is comparable to that noted in previous reports from North America. Several of our patients remained symptomatic for several months. Some reports have described chronic myalgia for as long as 3 to 42 years following infection. However, a recent, prospective, controlled study found insufficient evidence to conclude that chronic trichinosis exists as a distinct entity. One of the limitations of a study such as ours is recall bias, in that much of our data were obtained from patients several weeks after they had ingested the infected boar meat.

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However, the muscle biopsy does not usually demonstrate encysted larvae until 2 to 3 weeks have elapsed after the onset of symptoms. The optimal method for examining a muscle biopsy is to compress a portion of the unpreserved specimen between two glass slides and examine for encysted larvae.

Therapy for trichinosis has not been satisfactory and is somewhat controversial. Treatment is aimed at stopping the production of larvae by adult worms in the gut, killing migrating and muscle-dwelling larvae, and alleviating the severe systemic symptoms caused by the inflammatory response of the host. Although the three available anthelmintic drugs, thiabendazole, mebendazole and albendazole, will kill adult worms, the benefits of such action are questionable since larval production is self-limited and diagnosis of infection is often delayed to the point where further worm-kill is not likely to be beneficial. Thiabendazole is poorly tolerated and mebendazole is not well absorbed from the intestine. This leaves albendazole, the newest benzimidazole derivative, which has good absorption and demonstrated activity against tissue-dwelling larvae. However, the down-side of larval kill is the potential for a worsening of symptoms due to a “dead-worm” reaction or Herxheimer-type of response which may accompany treatment. Although three of our patients showed temporary improvement with albendazole treatment, perhaps due to a reduction in larval migration, the benefit of such therapy could not be assessed without a comparison with untreated controls. To date, well-controlled studies have not been carried out to assess the clinical outcome of treatment using any of the available anthelmintics. Finally, in anecdotal reports, the anti-inflammatory benefits of corticosteroids have been shown to be beneficial in heavily infected patients with severe disease. Even this form of therapy has not been adequately studied.

The incidence and mortality rates of trichinosis have declined in Canada and the United States during the past 50 years, primarily because legislation has been enacted which ensures that garbage is cooked before it can be fed to hogs. Also, home freezers have been used more frequently to store pork and there is a greater tendency for pork to be thoroughly cooked before it is eaten. In the 1940s, the average number of cases reported per year in the United States was approximately 400, with 10 to 15 deaths annually. Between 1982 and 1986, only 57 cases per year were reported in the United States with only 3 deaths noted during that time.

This report describes the first outbreak of trichinosis in Ontario since 1977. Previous episodes in Ontario have been secondary to ingestion of uncooked domestic hog meat. The most recent cases in 1977 were secondary to the ingestion of Hackepeter (ground raw pork). Much larger outbreaks in Canada have occurred in the Arctic as a result of polar bear and walrus meat consumption.

Control of this outbreak was achieved through the collaborative efforts of Ministry of Health officials, public health units and the medical community. The most significant outcome of the investigation of this outbreak was the finding that meat processing plants and slaughterhouses in Ontario were not adhering to the guidelines developed for federally inspected plants by the Department of Agriculture to ensure that wild boar meat consumed by the public was not infected with Trichinella. As a result of this outbreak, provincially inspected plants will now follow the same protocol as the federally inspected plants. These measures and recommendations include:

1. Every wild boar submitted for slaughter must be held and tested for Trichinella spiralis by the Ontario Ministry of Agriculture and Food - Livestock Inspection Branch. All wild boar are considered to present an ongoing risk of trichinosis to humans.
2. Public health inspectors and Ontario Ministry of Agriculture and Food inspectors must ensure that all smoke houses, drying rooms and freezers used for the destruction of trichinae are equipped with accurate automatic recording thermometers, and that all products subjected to heating methods are checked with digital probe thermometers.
3. Smoke house operators and meat processors should be educated and knowledgeable about prescribed treatment and precise curing methods to effectively destroy any possible live trichinae in wild boar or pork meat. There must be no doubt or confusion as to whether or not the finished product is ready-to-eat and the consumer must be made aware with appropriate labelling if further preparation is required.

CONCLUSION

This incident represents a classic case of a trichinosis outbreak caused by the consumption of insufficiently smoked/cooked wild boar meat infected with Trichinella spiralis. The outbreak serves to remind physicians and public health workers about the disease and the need to educate the community, farms, smokehouse operators/processors and government inspectors of the measures necessary to prevent trichinosis and the risk involved in the consumption of wild boar meat. Strategies of health promotion, education, inspection and enforcement must be applied to prevent further outbreaks of this preventable and potentially serious disease.

REFERENCES


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DEUXIÈME CONFÉRENCE NATIONALE DE L’ASSOCIATION CANADIENNE DE SANTÉ PUBLIQUE SUR LA LUTTE CONTRE LES MALADIES TRANSMISSIBLES

Toronto, Ontario — du 9 au 11 avril 1997
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