CDR REVIEW

Volume 7
Review Number 5
2 May 1997
ISSN 1350-9349

Communicable Disease Review

Adverse events after school leavers received combined tetanus and low dose diphtheria vaccine

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Summary

Since October 1994, children in the United Kingdom have been offered tetanus vaccine combined with a low dose of diphtheria vaccine (Td) at the age of 15 to 18 years. It is recommended that schoolchildren who have already received a booster of tetanus vaccine at the time of an injury should be given low dose diphtheria vaccine alone. When this vaccine is not available, however, it is recommended that Td vaccine should be given to all children. This study was performed to compare the frequency of adverse events after Td vaccine in 15 year old children with and without a history of an additional tetanus booster in the preceding 10 years.

Two hundred and sixty-five children were followed up – 52 pupils (20%) with a history of an additional tetanus booster, 157 (59%) with no such history, and 56 (21%) whose history was unclear. Mild local reactions were common and occurred more commonly in children with a history of an additional tetanus booster. Twenty-three pupils (44%) who had received an additional tetanus booster had swelling over 2cm diameter at the injection site, compared with only 39 (25%) of those with no such history ($p < 0.013$). Systemic symptoms were equally unusual in both groups. Only three children experienced symptoms attributed to vaccine that were severe enough for them to miss school or attend a doctor; and none of these had received an additional tetanus booster. We conclude that, in the absence of a supply of low dose diphtheria vaccine, offering Td vaccine to children with a history of additional tetanus booster is an acceptable policy.

Key words: adolescent health services – adverse drug reaction reporting systems – immunisation, secondary – vaccination

Introduction

An increased incidence of diphtheria in the former Soviet Union and concern about low levels of diphtheria antitoxin in the adult population of the United Kingdom[1,2] led, in October 1994, to the tetanus booster vaccine offered at the age of 15 to 18 years being replaced with a preparation containing tetanus toxoid and a low dose of diphtheria toxoid suitable for use in adults and adolescents (Td)[1] – 4IU of diphtheria toxoid in a 0.5ml dose compared with 30IU in the vaccine intended for primary immunisation .

Until 1994, schoolchildren who had received a booster dose of tetanus vaccine at the time of an injury would not receive another dose at 15 years of age because of an increased risk of adverse events[3]. Since 1994, the Department of Health has advised that such children should be offered low dose diphtheria vaccine. As this vaccine was in short supply[4], Td vaccine was offered to all children in some areas, whether or not they had previously received a tetanus booster. This study was performed to compare the frequency of adverse events after Td vaccine in children with and without a history of an additional tetanus booster within 10 years.

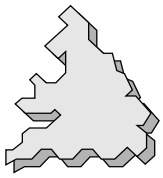

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Table Proportion of pupils who reported adverse events after Td vaccination

Symptom	Day	Additional tetanus booster received within 10 years					
		Yes (n = 52)		No (n = 157)		Not sure (n = 56)	
		number	(%)	number	(%)	number	(%)
Pain preventing arm movement	day 1	9	(17)	16	(10)	5	(9)
Swelling ≥ 2cm	day 2	23	(44) †	39	(25) †	11	(20)
Redness ≥ 2cm at the injection site	day 2	12	(23)	23	(15)	9	(16)
Headache	day 1	9	(17)	31	(20)	11	(20)
Temp ≥ 37ºC *	evening 1	15/52	(29)	35/152	(23)	12/55	(21)
	evening 2	13/52	(25)	49/153	(32)	20/55	(36)
Time off school	days 1–7	5	(10)	12	(8)	6	(11)
Consultation with doctor	days 1–7	1	(2)	3	(2)	1	(2)

† χ^2 test for difference between groups p = 0.013.

* denominators are number of pupils who had recorded the temperature at the correct time.

Methods

Adolescents aged about 15 years who were scheduled to receive Td vaccine at six schools (year 11) or at one general practitioner surgery were recruited by study nurses in North Hertfordshire, who sought their written consent to being followed up after vaccination and wrote to their parents for details of the children's vaccination histories. Td vaccine was given by deep subcutaneous injection into the anterolateral aspect of the upper arm by a doctor or practice nurse according to national recommendations[5]. The pupils were then asked to record their oral temperature, the size of local reactions, and any symptoms in a diary for 10 days after vaccination. The study nurse telephoned the pupils on the second and seventh days after vaccination to complete a further check list for other symptoms. The diaries and check lists were returned to the PHLS Communicable Disease Surveillance Centre (CDSC) in reply paid envelopes. Data were entered on a computerised database and the proportions of pupils with or without histories of tetanus boosters with symptoms were compared using a Chi squared test.

Results

Two hundred and sixty-five (82%) of the 324 children vaccinated were followed up. A history of a tetanus booster in the preceding 10 years was obtained from the parents of 52 pupils (20%), 157 (59%) had no such history, and for 56 (21%) the history was unclear. As the local accident and emergency department did not routinely write to general practitioners about tetanus boosters we were unable to verify their vaccination records.

Mild local reactions were common and occurred more commonly in the group that had received an additional tetanus booster within 10 years (table). Among pupils with a history of additional tetanus boosters, reports of local reactions did not differ with the interval since the last tetanus vaccination: the proportions of pupils with swelling of ≥2cm diameter on the second evening who had received the additional tetanus booster six or more years, three to five years, or less than three years previously were 40% (4 / 10), 53% (10 / 19), and 39% (9 / 23) respectively. The proportions of pupils who reported mild systemic symptoms did not differ between groups (table).

Time off school and visits to the doctors were uncommon (table) and mostly unrelated to the vaccination. Three children who missed days off school and one child who consulted a doctor had symptoms that were attributed to vaccination; none of these children had received an additional tetanus booster.

Discussion

This study indicates that children with a history of an extra tetanus booster are more likely to suffer local reactions after Td vaccine than children with no such history, but are at no higher risk of systemic symptoms. Most of the local reactions described were mild and required neither time off school nor a visit to the doctor. Local reactions to Td occurred less frequently than in a previous study of adsorbed tetanus vaccine at this age[3]. This difference may be explained by the different preparations of vaccine used or because fewer children in the current study had received extra tetanus boosters.

We conclude that, in the absence of a supply of low dose diphtheria vaccine, offering Td vaccine to children with a history of additional tetanus booster is an acceptable policy. The risk of local reactions should be explained, however, and low dose diphtheria vaccine should be obtained for children who have had moderate or severe local reactions to a previous dose of tetanus vaccine. With better access to records of routine vaccination in accident and emergency departments – for example, through increasing use of parent held records – unnecessary tetanus boosters should be given less frequently. When a child of 13 years of age or older requires a tetanus booster at the time of an injury Td vaccine should be used in preference to tetanus vaccine alone.

Acknowledgements

The authors wish to acknowledge the cooperation of local general practitioners, school nurses, and clinical medical officers. We would also like to thank the study nurses and Liz Turner, the school nurse manager, for their help with the design and conduct of the study and Michael Stevenson for administrative support.

Conflict of interest: None

Funding: Department of Health

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Management of school leavers given a diphtheria and tetanus vaccine intended for children instead of the intended low dose preparation

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Summary

In November 1995, 102 school leavers in two North Staffordshire schools were given high dose diphtheria and tetanus vaccine (intended for primary immunisation of children) rather than a preparation with a low dose of diphtheria vaccine intended for adults and adolescents.

We describe the management of the incident and the action taken to minimise the risk of such an error being made again.

Pupils who had received the high dose vaccine and a control group were surveyed with a self-administered questionnaire. Thirteen children out of 67 given the higher dose diphtheria vaccine consulted their general practitioner and the same number had time off school, compared with none of 25 from a control school. This excess morbidity was probably attributable to the higher dose of diphtheria vaccine.

Key words: adolescent health services – adverse drug reaction reporting systems – follow up studies – immunisation, secondary – risk management – vaccination

Introduction

In October 1994 the chief medical officer recommended that school leavers should be offered a booster dose of diphtheria vaccine as well as tetanus vaccine[1]. The later dose was added in response to the increased incidence of diphtheria in Eastern Europe and the need to ensure that young adults were adequately protected against the disease. A study of immunity levels to diphtheria showed that about 38% of blood donors in the United Kingdom were susceptible to diphtheria and immunity decreased with advancing age[2]. Diphtheria vaccine is therefore now offered to children at 2, 3, and 4 months of age and before starting school, and in a lower dose to school leavers. The lower dose is given because of the possibility of a reaction in an individual who is already immune.

On 20 November 1995, diphtheria and tetanus vaccine intended for primary immunisation of children (DT) was given to school leavers (aged 15 to 16 years) at a North Staffordshire secondary school (school A) instead of diphtheria and tetanus vaccine for adults and adolescents (Td), which has a lower dose of diphtheria vaccine (4IU in a 0.5ml dose compared with 30IU in DT).

The school's nursing sister told the immunisation coordinator (consultant in communicable disease control) for North Staffordshire and an incident team was convened the same day.

Investigation

The aims of the incident team were to ensure that no further DT vaccine was given to school leavers; to contact on the same day the parents and general practitioners of the children who had been given DT and advise them of the error and its implications; to investigate whether a similar error had occurred at other schools and take appropriate action; to put procedures in place to minimise the risk of such an incident recurring; and to inform the PHLS Communicable Disease Surveillance Centre (CDSC) and Department of Health so that action could be taken to prevent similar incidents in other areas.

It was confirmed that 34 school leavers at school A had been given DT vaccine on 20 November, and that 68 children at another school in the area (school B) had received DT vaccine between 13 and 15 November.

A self administered questionnaire was devised to include questions about time off school, consultations with general practitioners, and adverse effects of the vaccine. Questionnaires were sent to the parents of all pupils at schools A and B who had been given DT vaccine and to the parents of pupils at another school (school C) who had been given the correct vaccine (Td) on the same day as pupils at school A. The questionnaires were posted on 30 November. For pupils at school B the questionnaire accompanied the letter advising parents of the error.

No attempt was made to check with general practitioners about consultations reported, but general practitioners, in their letter, had been asked to report any severe reactions.

Questionnaires were sent with a prepaid return envelope. No reminders were sent. The responses were analysed using Epi Info version 6.

Results

A total of 151 questionnaires were distributed to parents of pupils at the three schools; 34, 68, and 49 at schools A, B, and C, respectively. Ninety-two questionnaires were returned (74% (25) in school A, 62% (42) in school B, and 53% (25) in school C), an overall response rate of 61%. Most parents responded within two weeks.

Mild local reactions were common and occurred with higher frequency in the pupils who had received DT vaccine (table). No children from school C reported a fever compared with seven (28%) from school A and 10 (24%) from school B.

About a fifth of pupils from schools A and B visited their doctor after the immunisation session as a result of a perceived side effect of the vaccine compared with none in school C (the questionnaire specified no time period by which side effects should be reported and no attempt was made to validate them). A third of children from school A, a fifth from school B, and no children from school C took days off school because of the perceived side effects of the vaccine (table).

Fifteen parents telephoned for advice, three wrote comments on the questionnaires, and two requested interviews to discuss their concerns. Two written complaints were received. One article was published in a local newspaper.

The following steps were taken locally to minimise the risk of such an incident occurring again:

- school nurses were briefed about the importance of checking all vaccines;
- the community pharmacist began labelling DT vaccine boxes "for children under 10 years only";
- the vaccine ordering system was reviewed to ensure adequate checks were made at each step where errors could occur;
- general practitioners were warned in the local communicable disease newsletter.

Discussion

The incidence of adverse effects was higher in children given DT vaccine than in those given Td. The excess existed both for the children who were told about the error on the same day as they were immunised and for those told nearly two weeks later. No serious effects were noted – for example, admission to hospital – but it appears that a considerable excess of morbidity (as indicated by consultations with general practitioners and time off school) was caused, consistent with investigations after similar incidents[3]. This excess was probably due to the higher dose of diphtheria vaccine.

It could be argued that some of the morbidity reported could have arisen because of concerns about the safety of the vaccine after recipients and their parents were told that an error had occurred. If this was the only explanation, considerably fewer reports of adverse effects would have been expected from school B, whose pupils were not informed of the event until later. Anxiety may have contributed to some of the reports but there appears to have been a real excess in morbidity.

Table Proportion of children at schools A, B, and C who reported adverse events

	School A (DT given) n = 25 number (%)	School B (DT given) n = 42 number (%)	School C (Td given) n = 25 number (%)
Redness at injection site*	19 (76)	26 (62)	6 (23)
Swelling at injection site*	16 (64)	25 (59)	4 (15)
Fever*	7 (28)	10 (24)	–
Consultation with GP*	5 (20)	8 (19)	–
Time off school*	6 (32)	7 (17)	–

* Pupils at schools A and C and B and C differed significantly ($p < 0.05$ by χ^2 or Fisher's exact test as appropriate) for all five parameters. No significant differences were found between pupils of schools A and B.

This incident, and others in which vaccine errors have occurred[3-5], emphasise the importance of assessing the potential for error in all steps of the storage, ordering, distribution, and administration of vaccines. A final check, however, must always be made by the person who gives the vaccine and responsibility for this must be clear. The 1996 edition of *Immunisation against Infectious Disease*[6] offers clear guidance on the importance of doctors providing immunisation having received training and being proficient in the appropriate techniques. Section 6 of the guidance highlights the conditions that should be fulfilled when a doctor delegates the responsibility for immunisation to a nurse. In North Staffordshire annual 'immunisation days' are held each year to update those who administer vaccines and regular training sessions are held for smaller groups.

Although the Td vaccine was clearly labelled "to be used in adults and adolescents", only in the fine print of the DT vaccine was it indicated that the vaccine should not be used in those over 10 years of age. Clearer labelling might help reduce the risk of further incidents.

Acknowledgements

The authors wish to acknowledge the cooperation of the school nurses and clinical medical officers in this investigation. We thank Dr Simon Lenton for his help with the questionnaire and Dr Mary Ramsay for her comments on the manuscript.

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Outbreak of trichinosis in France associated with eating horse meat

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Summary

The investigation of a trichinosis outbreak in Auvergne, France identified 23 cases in 12 households living in two cities – Clermont-Ferrand and Montluçon – between 15 February and 7 March 1991. One patient required intensive care, 15 had major symptoms, and seven had minor or no symptoms. Two case control studies demonstrated a significant ($p < 0.01$) association between eating horse meat and acute trichinosis. Veterinary services found that three supermarkets where the patients had bought horse meat during the suspected period had been supplied by a single wholesaler. The analysis of the wholesaler's records revealed that the implicated horse meat had been imported from a slaughterhouse in the United States. This outbreak occurred despite a requirement in France for all meat from horses slaughtered in France and in countries exporting meat to France to be examined systematically for trichinella.

Key words: communicable disease control – disease outbreak – food poisoning – parasitic diseases – trichinella

Introduction

Human trichinosis is a parasitic infection caused by larvae of *Trichinella* species, which are roundworms that live in living tissue[1]. Many infections are subclinical. Symptomatic cases suffer diarrhoea and abdominal pain in the first week after infection followed by facial oedema, muscular pain, fever, and eosinophilia, and subsequent widespread vasculitis and muscle damage in the most severe cases[1]. Trichinella larvae form cysts in the striated muscles of mammals such as bear, seal, wild boar, and pig, and may be ingested when rare or undercooked meat is eaten. The larvae mature in the small intestine, where the adult worms in turn produce larvae that cross the intestinal mucosa, migrate through the blood to striated muscles, and form cysts.

The first outbreak of trichinosis reported in France occurred in 1881 and was ascribed to eating pork[2]. Only one recent family outbreak has been associated with eating pork[3]. Other cases reported in France have resulted from eating wild boar[4-8]. The incidence and prevalence of infection in wild boar is not available.

Since 1975, imported horse meat has been associated with several outbreaks in France[9-12] and Italy[11,13-15]. We report the fourth outbreak of trichinosis in France related to imported horse meat. Brief reports of aspects of this outbreak have been published in French[16-18].

Methods

Several members of a family living in the district of Clermont-Ferrand, in Auvergne, developed muscular pain, oedema, and eosinophilia. Their general practitioner diagnosed trichinosis on 25 February 1991. At the same time a patient unrelated to the former family was admitted to the infectious diseases ward of Clermont-Ferrand's university hospital with similar clinical features. All had recently eaten horse meat. The suspected diagnosis of trichinosis raised the possibility that the outbreak could be widespread in the community. The district health and veterinary services were immediately alerted and active case finding was carried out.

Case finding

The district health services wrote to all local clinicians informing them that cases of trichinosis had recently been diagnosed in Auvergne, describing the symptoms and signs, and encouraging them to look for eosinophilia in such patients, arrange serological tests, and notify the district health service of all suspected cases.

All the medical laboratories of the province were asked for information about unexplained cases whose blood films showed eosinophilia. Local media informed the public about the possible risk of trichinosis associated with eating rare or undercooked horse meat. Members of the public were encouraged to consult their family doctor if they had a flu-like illness with muscular pain or facial oedema.

The national health service ordered all district health services to collect details from hospitals and private laboratories about all suspected cases with unexplained eosinophilia and raised levels of muscle enzymes in blood.

Case definition

A probable case was defined as a patient with recent symptoms and signs of trichinosis (fever, facial oedema, or myalgia) or eosinophilia ($>1 \times 10^9$ eosinophils per litre) associated with a positive indirect immunofluorescence assay (IF) for trichinella antibodies and who was diagnosed between 15 February and 15 March 1991. A confirmed case was defined as a person with either a trichinella positive muscle biopsy or in whom serial IF assays demonstrated seroconversion.

Epidemiological studies

Two case control studies began on 3 April 1991. Asymptomatic relations who lived with the patients were selected as controls in one study, and cases nominated asymptomatic neighbourhood contacts in the other. The following data were collected by telephone interview: age, sex, symptoms and signs, prescribed treatment, meat

consumption between 15 January and 15 March 1991, and usual method of cooking meat. No blood specimens were taken from controls. Data were analysed using Epi Info version 6[19].

The district veterinary services carried out inquiries into the distribution network of horse meat, focusing on the supermarkets that supplied the affected families.

Local dietary habits, particularly relating to the types of meat eaten and the ways they were cooked, were surveyed by interviewing a sample of 480 consecutive customers in a supermarket in Clermont-Ferrand.

Parasitology

Serological tests were performed on blood from all patients suspected to have trichinosis. Three different laboratories performed indirect immunofluorescence assay (IF): university hospital Pitié-Salpétrière Paris (M Danis), Institut Pasteur Lyon (A F Petavy), and the university hospital of Clermont-Ferrand (M Cambon). Positive results were defined as an antibody titre > 1/200 in the first laboratory and > 1/80 in the second and third laboratories.

Two muscle biopsies from patients suspected to have trichinosis were examined for trichinella.

Samples of meat seized at the time of the investigation, and one sample of horse meat bought during the suspected risk period by an affected family and stored in their freezer, were examined microscopically by the district veterinary services.

Results

Alert and interventions

When the first suspected case of trichinosis was confirmed by seroconversion (IF: 1/800) on 20 March 1991, the infectious diseases ward informed the district public health service. On 21 March, the French Ministry of Agriculture and Forestry banned further importation of frozen horse meat and recommended that all horse meat already imported should be examined systematically for trichinella cysts[17].

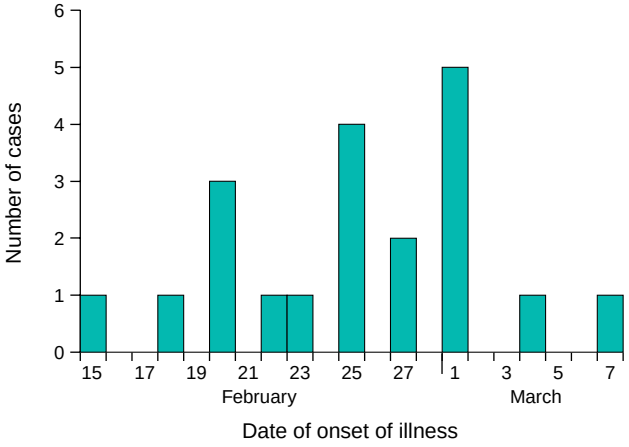
Clinical and pathological findings

Active case finding identified 23 cases (15 men and 8 women) in 12 households in two cities of Auvergne – Clermont-Ferrand and Montluçon. No cases from other areas of France were notified during the investigation. The median age of the affected individuals was 38 years (range: 4.5-68 years).

Twenty symptomatic cases reported having become ill between 15 February and 7 March 1991 and the date of onset of illness was unavailable for one patient. The epidemic curve peaked during the last week of February (figure). The median incubation period, based on information from ten symptomatic patients who reported eating horse meat on a specific date, was 13 days (range: 8-23 days).

Clinical severity varied. The index case required intensive care for shock, respiratory distress, myocarditis, and thrombocytopenia[18]. Fifteen further cases had severe features (fever higher than 39°C, myalgia, or oedema of the face and the hands) and two were admitted to hospital. Among the 15 with such features, eight patients had gastrointestinal symptoms (diarrhoea and abdominal pain) and four had headache. Seven additional cases had minor or no symptoms, ten had rash, and none had neurological

Figure Outbreak of trichinosis in Auvergne, France associated with imported horse meat by dates of onset



symptoms. The median duration of the disease in the 14 patients for whom data were available was 18 days (range: 2-90 days).

A white blood cell count greater than 10 x 10^9/l was found in 11 patients among 22 tested and eosinophilia (eosinophils >1 x 10^9/l was found in 21 cases (range: 0.5-15.5). Muscle enzymes (creatine phosphokinase and/or lactate dehydrogenase) were increased in 10 of the 14 serum specimens tested. Liver enzymes were moderately increased in six patients tested.

Trichinella larvae were detected in muscle biopsies from two patients and a rise in titres between consecutive specimens was observed in four patients. Single tests on serum from 14 patients showed titres of trichinella antibodies that exceeded the laboratory's threshold for positivity. One further patient had stable positive titres on serial testing.

All the patients were treated with flubendazole or albendazole for at least 10 days. The most severe case, the index case, recovered slowly after receiving three 21 day courses of treatment over six months.

Epidemiological findings

All the patients reported having eaten horse meat in the month before becoming ill. Horse meat had been eaten within 24-48 hours of purchase and had been eaten rare or half cooked by 17 patients. Only one affected family had stored horse meat (a 300 g piece) in their freezer .

Both case control studies showed a significant association between eating horse meat and acute trichinosis ($p < 0.01$; table). Patients were more likely to have eaten horse meat rare or undercooked than controls ($p < 0.01$, odds ratio 19.8, confidence interval 2-274), and were more likely than controls to have bought the implicated horse meat in supermarkets of one particular chain ($p < 0.01$).

The patients had bought horse meat in three different supermarkets of the same chain, two supermarkets (A and B) in Clermont-Ferrand and one (C) in Montluçon. Some patients ate horse meat every week or had bought it several times in February. Only a few patients could recall the dates when they bought the implicated pieces; before 14 February in A and on 6 February in C. No relevant information was available from the patients who shopped

Table Meats eaten by cases of trichinosis and family and neighbourhood control groups in Auvergne, France March 1991

Meat consumption	Cases (n = 20) exposed	Family controls (n=10) exposed	Odds ratios (confidence interval)*	p value	Neighbourhood controls (n=35) exposed	Odds ratios (confidence interval)	p value
Beef	19	8	4.7 (0.2 - 294)	NS	34	0.6 (01 - 46)	NS
Pork	20	10	–	NS	32	–	NS
Wild boar	1	–	–	NS	–	–	NS
Mutton	10	2	4.0 (0.6 - 46)	NS	17	1.1 (0.3 - 3.7)	NS
Fowl	20	10	–	NS	31	–	NS
Horse	20	5	–	<0.01	4	–	<0.01

* Odds ratios and confidence intervals (exact method) if calculable. NS = not significant.

at B. The district veterinary services found that during the suspected period, mainly the first two weeks of February, A and C received deliveries from the same wholesaler in Maine-et-Loire, in the western part of France, on 11 and 4 March respectively.

This wholesaler imported horse carcasses from several slaughterhouses in the United States, Canada, and Argentina. The horse carcasses were cut up, boned, vacuum-packed, and distributed throughout the country after a routine microscopical examination of their diaphragms for trichinella. The meat was finally packaged in the supermarkets just before sale. Both of the implicated deliveries were of hindquarters, and weighed 89 and 63 kilograms (kg) for A and C, respectively. Examination of the wholesaler's records (dates of importation of the carcasses and dates of delivery of pieces of horse meat to supermarkets A and C) suggested that the implicated meat was imported from a specific slaughterhouse in the United States.

The same wholesaler made a further delivery (weighing 73kg) to A on 27 February. The single piece (weight 300g) that had been stored in a freezer came from this delivery and had been bought on 2 March in A.

The survey of local dietary habits undertaken in one supermarket revealed that 36% (171/480) of customers ate horse meat, 59% (101/171) of whom ate it rare or undercooked.

Parasitology findings

By the time the district veterinary services were alerted, all horse meat from the implicated deliveries had already been sold. Examinations of samples of horse meat from subsequent deliveries seized at the time of the investigation were all negative. Examination of the single piece of frozen horse meat stored by one of the affected families revealed one larva of *Trichinella* sp[17].

Discussion

This outbreak of trichinosis associated with imported horse meat occurred in France despite a recommendation in 1985 that all meat from horses slaughtered in France, and in countries exporting horse meat to France, should be examined for *Trichinella* spp.

Only 23 cases were identified in this outbreak, in a single province of France, although meat from the implicated carcasses had been distributed throughout the country.

The detection of only one larva of *Trichinella* sp in a 300g piece of the implicated horse meat after an active examination suggests that it must have had a low parasitic load. There may have been other cases with mild or no symptoms who could have been misdiagnosed or may not have presented to health services.

The method used to cook meat in Auvergne may also have protected residents from infection. Our investigation of local dietary habits revealed that only 59% of those who ate horse meat ate it rare or undercooked, compared with 98% in Paris[10]. The investigation also showed that 53% of them bought horse meat in butchers' shops and would therefore have avoided these infected batches.

Most of the case finding was retrospective since it officially started on 20 March, when local clinicians and the population were informed. The main period when contaminated horse meat was suspected to have been bought was the first two weeks of February. Systemic symptoms usually appear between eight and 15 days after eating infected meat. Most of the cases would therefore have occurred at the end of February or at the beginning of March and some may have been missed by the case finding exercise.

Cases were scattered in a large area around the implicated supermarkets. In previous trichinosis outbreaks reported in France, horse meat was delivered to butchers' shops rather than to supermarkets and cases were confined to a small area around the shops. Without the index case, who was very ill with myocarditis and acute respiratory failure requiring mechanical ventilation, the present outbreak might not have been recognised[18].

An outbreak of flu-like illness occurred at the beginning of March. General practitioners could have misdiagnosed cases of trichinosis as flu, because they do not routinely order serological tests.

Three previous outbreaks of trichinosis related to eating imported horse meat have been reported in France. An outbreak near Paris in 1976 consisted of 125 patients who were affected by horse meat from Poland[9]. The second and third outbreaks occurred in 1985, and were even larger, consisting of 431 patients in Melun and the 14th district of Paris, and 642 patients in the 12th district of Paris, the city's suburbs, and other towns scattered in France. One carcass from a slaughterhouse in the United States and one carcass from West Germany were implicated[10].

Although control measures were instituted after the outbreaks in 1985 and the outbreak reported here, two further outbreaks have occurred in France. In 1993, 538 cases were associated with horse meat from Canada[20]. In 1994, seven cases were associated with horse meat from Mexico[12]. Following a recommendation of the European Community, the French government decided, on 18 September 1995, that the examination for *Trichinella* spp should be improved by taking a larger sample of muscle tissue (10g from the tongue or the cheek) or by treating a 5g sample of muscle tissue with proteolytic enzymes before examination.

How a horse, which is herbivorous, can be infected by trichinella larvae is not well understood. Horses can be experimentally infected with *Trichinella spiralis*[11,20]. *T. spiralis* muscle larvae were detected in four naturally infected horses from a slaughterhouse in Mexico[20]. Natural infection could occur by accidental ingestion of contaminated rodents or meat ground up with hay or compounded feed.

Consumers need to know that horse meat must be cooked to at least 65°C or frozen before consumption (at least 10 days at -18°C) to prevent infection[20].

Clinicians should remember the clinical features of human trichinosis (facial oedema, muscular pain, and fever) and that people who eat horse meat remain at risk of it. Suspected cases should be screened for eosinophilia and elevation of muscular enzymes. Seroconversion or a muscle biopsy is needed to confirm the diagnosis. One negative serological test for trichinella does not exclude acute trichinosis (the index case in this outbreak, for example) and a second serological test is needed to confirm the diagnosis. Several weeks may elapse between infection and seroconversion. A single low serological titre of trichella antibodies in a person should be interpreted with caution. False positive results may occur due to cross reaction with other nematodes (ascaris, toxocara) and laboratories have defined thresholds to improve the specificity of serological tests. Results of serological tests need to be interpreted in the light of epidemiological and clinical data.

Epidemiologists, clinicians, parasitologists, and veterinarians all have a role to play in preventing and investigating human outbreaks of trichinosis.

Acknowledgements

We wish to thank Dr Patrick Wall from PHLS Communicable Disease Surveillance Centre for his helpful comments on drafts of the manuscript.

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An outbreak of *Salmonella enteritidis* phage type 4 infection in a rural community in Northern Ireland

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Summary
An outbreak of gastroenteritis arose in people who attended a charity barbecue at a hotel in a rural area of Northern Ireland in July 1995. About 120 people attended the barbecue, 98 of whom were identified. Fifty-one of them and seven members of hotel staff met the case definition. An epidemiological investigation showed that illness was significantly associated with eating foods containing mayonnaise that had been prepared using raw shell eggs and stored at too high a temperature. *Salmonella enteritidis* phage type 4 was cultured from 17 out of 24 faecal specimens received from people who attended the barbecue and in 17 out of 34 faecal specimens from staff, including all seven staff cases. The primary source of infection was not identified despite thorough investigation. This paper highlights the value of administering questionnaires by telephone when investigating community outbreaks of infection in rural areas, the important role of general practitioners in the identification of community outbreaks, and the need to periodically reiterate public health messages, in particular for food handlers and caterers.

Key words: communicable disease control – disease outbreak – eggs – intestinal diseases – salmonella food poisoning

Introduction
Three cases of gastroenteritis in members of a rural farming community in Northern Ireland were notified to the duty public health doctor by a general practitioner on Sunday 23 July 1995. One of the patients had been admitted to hospital and was positive for *S. enteritidis*; faecal specimens from the other two patients were still being examined. All three patients had eaten at a church barbecue in a nearby hotel on 18 July 1995. Other general practitioners in the surrounding area were contacted. Several had noted a recent increase in the number of patients complaining of gastrointestinal symptoms and ten other patients with gastroenteritis who had also eaten at the barbecue were identified. An outbreak of gastroenteritis was confirmed and an outbreak control team was convened with representatives from public health medicine, environmental health, Northern Ireland Public Health Laboratory (NIPHL), and the Department of Agriculture, Northern Ireland (DANI). A case was defined as a person who had attended the barbecue and developed vomiting and / or diarrhoea within the next seven days.

Investigation
Epidemiology
The hotel manager was asked to provide a menu, a guest list, and information about recent illness among staff. No guest list was available because tickets for the barbecue had been sold 'door to door' by a number of ticket sellers. Case finding was undertaken by telephoning all local general practitioners, known cases, ticket sellers, the parish priest who had organised the barbecue, and the owners and manager of the hotel. Local media and local churches were asked to publicise the outbreak and ask people who had attended the barbecue to contact the department of public health medicine.

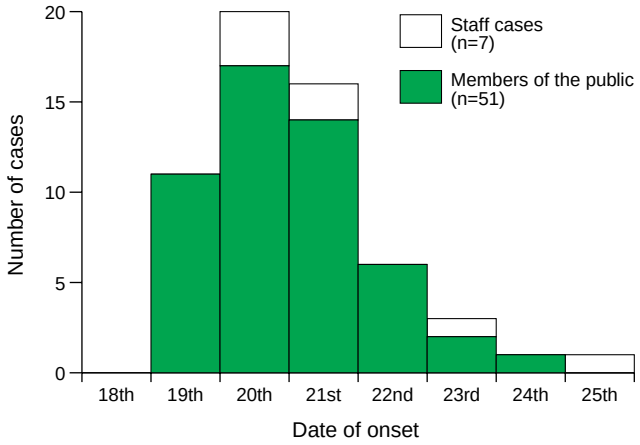
A questionnaire was prepared about symptoms and foods eaten. The questionnaire was administered by telephone to hotel staff and to people known to have attended the barbecue. All cases and hotel staff were asked to submit faecal specimens. People who attended the barbecue but did not become ill were not asked for specimens.

Public health departments in other Health and Social Services Boards in Northern Ireland were told about the outbreak and asked to provide information about the follow up of patients with *S. enteritidis* infection notified in the two weeks after 18 July 1995.

Chi squared tests with appropriate corrections were performed on the data collected using Epi Info, version 6.

Environmental
The food preparation and storage practices at the hotel were inspected in detail. Specific questions were asked about the menu, and the preparation and storage of food served at the barbecue. A preliminary review of the menu revealed that raw shell eggs had been used to make fresh

Figure Onset of gastroenteritis following barbecue on 18 July



mayonnaise. A request was made for any remaining foods and any eggs from the same batch as those used to make the mayonnaise for the barbecue to be submitted for culture. The hotel's egg supplier was identified and visited to obtain samples of eggs for culture and details of the farms that provided the eggs. These farms were visited by veterinary service staff at DANI. Cloacal swabs were obtained from hens and samples of dust and litter from laying houses.

Microbiology

Faecal specimens from cases who had attended the barbecue and from members of the hotel staff with and without symptoms were examined using standard bacteriological methods at NIPHL. The specimens were tested for the presence of the common bacterial faecal pathogens (salmonella, shigella, campylobacter, and *Escherichia coli* O157). Suspected colonies of salmonella were confirmed biochemically and serologically, and all strains identified as *Salmonella enteritidis* were referred to the PHLS Laboratory of Enteric Pathogens in Colindale for confirmation of their identity and phage type (PT).

A small portion of potato salad and a pork chop left over from the barbecue were examined for the presence of salmonellas at the Food Hygiene Laboratory, NIPHL, using standard methods. Eggs and environmental samples from laying houses were tested at the salmonella unit of the Veterinary Sciences Laboratories, DANI.

Control measures

The hotel staff were told to stop making fresh mayonnaise with raw shell eggs. The chief medical officer of Northern Ireland issued a press release advising about food precautions to be taken in hot weather and reiterating advice about the use of raw shell eggs. All cases were informed about the need for standard enteric hygiene precautions[1]. Cases who were also food handlers were excluded from work until they had supplied one stool specimen that was negative for *S. enteritidis.*

Results

Epidemiology

Ninety-eight people who attended the barbecue were identified, over 80% of the estimated total of 120. Ninety completed the questionnaire administered by telephone, a response rate of 92%. The other people who attended could not be identified. Fifty-one (57%) of those interviewed fulfilled the case definition. All cases were

Table Association between eating foods at the barbecue and becoming ill

	Eaten			Not eaten					
Food item	Ill	Not ill	Attack rate (%)	Ill	Not ill	Attack rate (%)	Relative risk	(95% confidence interval)	p value
Meat and fish									
rump steak	52	38	58	7	9	44	1.32	(0.74 - 2.37)	NS
beefburger	33	25	57	26	22	54	1.05	(0.75 - 1.48)	NS
pork chop	23	19	55	36	28	56	0.97	(0.69 - 1.38)	NS
pork sausage	31	23	57	28	24	54	1.07	(0.76 - 1.05)	NS
kipper	5	5	50	54	42	56	0.89	(0.47 - 1.69)	NS
Vegetables									
baked potato	18	20	47	41	27	60	0.79	(0.53 - 1.16)	NS
potato salad*	25	10	71	34	37	48	1.49	(1.08 - 2.06)	0.0217
rice salad*	26	9	74	33	38	46	1.60	(1.16 - 2.19)	0.007
mixed salad	34	23	60	25	24	51	1.17	(0.83 - 1.66)	NS
egg mayonnaise*	21	8	72	38	39	49	1.47	(1.07 - 2.02)	0.0331
garlic mayonnaise*	6	3	67	53	44	55	1.22	(0.74 - 2.00)	NS
coleslaw*	44	20	69	15	27	36	1.92	(1.24 - 2.98)	0.0008
Bread	12	11	52	47	36	57	0.92	(0.06 - 1.42)	NS
Orange juice	2	2	50	57	45	56	0.89	(0.33 - 2.42)	NS
Any food containing mayonnaise	55	4	93	29	18	62	1.57†	(1.33 - 1.85)	0.00007

* Foods containing mayonnaise. † Mantel-Haenzel. NS = not significant.

included in the analysis. The predominant symptom was diarrhoea. Cases became ill between 19 and 24 July 1995, and the modal date of onset was 20 July (figure). No secondary cases were identified. Twenty-three members of staff worked on the day of the barbecue; 17 ate at the barbecue and seven of them fulfilled the case definition. Three staff became unwell on 20 July and the other staff cases arose on 21, 23, and 25 July 1995, with a peak of cases on 20 July 1995.

All 34 members of staff ate hotel food while at work. They could not say for certain what they had eaten on the days immediately before and after the barbecue. None reported any illness in the days leading up to the barbecue.

Consumption of several foods was significantly associated with the development of gastroenteritis (table) – potato salad, rice salad, egg mayonnaise, and coleslaw. All of these foods contained freshly prepared mayonnaise. Further analysis of the data revealed that eating any foods containing mayonnaise was significantly associated with subsequent gastroenteritis (table). Eating foods that did not contain mayonnaise was not associated with gastroenteritis (table).

Environmental

The kitchens and food preparation and storage areas were satisfactory when inspected. Investigation of food preparation for the barbecue revealed that the mayonnaise had been prepared with raw shell eggs on the day of the barbecue by a member of staff who was in good health, did not eat at the barbecue, and whose faecal specimens were negative when subsequently tested for *S. enteritidis*. The mayonnaise was stored at ambient temperatures of about 18° to 20°C for four hours before being used as a dressing for coleslaw and potato salad, and in egg mayonnaise. Three members of staff who mixed the mayonnaise with the salads and who ate at the barbecue remained asymptomatic. Their stools were found to be positive for *S. enteritidis* PT 4 on subsequent testing.

No salmonellas were cultured from the pork chop or potato salad, which had been kept frozen after the barbecue. No eggs from the batch used to make the mayonnaise were available for testing.

The egg supplier was unable to provide eggs from the same batch as had been used at the hotel at the time of the barbecue. Two farms were identified that had definitely provided the fresh eggs. No salmonellas were cultured from samples of eggs obtained from them. *S.colorado* and *S. livingstone* were isolated from environmental samples taken from one laying house in one of the farms; *S. enteritidis* was not, however, isolated from any farm.

Microbiology

S. enteritidis PT 4 was isolated from 17 of the 24 faecal specimens obtained from guests who had attended the barbecue . Faecal specimens were also obtained from the 34 members of hotel staff. *S. enteritidis* PT 4 was also isolated from 17 members of staff – seven who fulfilled the case definition, five who ate at the barbecue but did not become unwell, two who neither worked nor ate at the barbecue, two who became ill after the barbecue but had not eaten at the barbecue, and one from a member of staff who worked at the barbecue but ate none of the food. *S. enteritidis* PT 5 was isolated from a faecal specimen from a member of staff who neither worked nor ate at the barbecue. This was considered to be a coincidental finding.

During the outbreak investigation, a patient with *S. enteritidis* PT 4 infection was notified to a public health department in another health board area. Follow up revealed that this person had eaten two lightly fried eggs, bacon, sausages, fried bread, fruit juice, and tea for breakfast at the hotel on 19 July 1995. Diarrhoea and abdominal pain began the next day.

Discussion

This large outbreak of food poisoning associated with *S. enteritidis* PT 4 was identified due to the vigilance of a general practitioner who made the link between one of his patients with salmonella infection and other patients with gastroenteritis who had attended the barbecue. Local general practitioners helped to identify further cases and the outbreak was confirmed.

This outbreak highlights the difficulties of controlling and investigating such an incident in a rural area. The outbreak occurred in a scattered farming community, many members of which worked in the fields during the day and returned home late in the evening. This and the absence of a guest list made it difficult to identify and to contact cases. Thorough case finding by two of the authors (LD, MMcC), however, ensured that a large proportion of people at the barbecue were identified. Case finding was aided by local knowledge of several members of the community and early help from the local media and churches.

The telephone questionnaire was very useful in the investigation of this outbreak. It was often difficult to obtain accurate addresses for people who had attended the barbecue, but telephone numbers were often readily available. The scattered rural homes would have made face to face interviews difficult to hold and slowed down the investigation and control of the outbreak. Interviewing by telephone achieved a 92% response rate among those known to have attended the barbecue.

Epidemiological analysis of data collected during the investigation showed that eating food containing mayonnaise made with raw shell eggs was significantly associated with subsequent development of gastroenteritis. It is therefore likely that this mayonnaise was the vehicle of infection. A person who ate lightly cooked eggs at the hotel on the morning after the barbecue subsequently developed symptoms and tested positive for *S. enteritidis* PT 4. Other epidemiological studies have also linked *S. enteritidis* PT4 infection with eating raw or undercooked shell eggs[2-7]. The source of the salmonella could not be demonstrated microbiologically because by the time the outbreak was identified none of the eggs of the same batch was available for examination, as was reported in a similar outbreak[4].

We considered the possibility that the mayonnaise had been contaminated by an infected food handler, because faecal specimens from over half of the staff were positive for *S. enteritidis*. No member of staff reported illness in the week before the barbecue, however, and most of those whose stools were positive ate at the barbecue. Recall by staff of other food eaten at the hotel in the days before and after the barbecue was poor. The mayonnaise was prepared by a member of staff who was

well and whose stool was negative for *S. enteritidis*. The three members of staff who mixed the mayonnaise with salads had stools positive for *S. enteritidis*, but they also ate foods containing mayonnaise at the barbecue. There is little evidence to suggest that asymptomatic food handlers contribute to outbreaks of salmonella infection[1]. It seems likely that the staff were part of the outbreak but not its cause.

However the mayonnaise became contaminated, faulty food handling practices probably contributed to the high attack rate in this outbreak. The mayonnaise was left to stand at ambient temperatures of 18° to 20°C for up to four hours before serving, providing ideal conditions for salmonellas to multiply. Storage of food at inappropriate ambient temperatures has been a contributory risk factor in other salmonella outbreaks[5].

Advice on avoiding the use of raw shell egg in uncooked dishes had not been followed, as has been the case in other outbreaks of *S. enteritidis* infection[6-8]. This outbreak of *S. enteritidis* could have been prevented if government advice issued in 1988[9] – reiterated by the Advisory Committee on the Microbiological Safety of Food[10] – that people should avoid eating raw eggs or uncooked foods made from them, had been followed. This outbreak emphasises the need to reiterate important public health messages from time to time, in particular to food handlers and caterers. It is also clear that due consideration must be given to finding the most effective method of communicating messages about risk if similar outbreaks are to be prevented. General practitioners also need to remain vigilant and consider the possibility of food poisoning in patients who present with gastrointestinal symptoms.

Acknowledgements

The authors would like to acknowledge the help of Ms Jennifer Murphy and Mr John Mc Kee, Environmental Health, and Mr. Michael Sherrey, DANI, in the investigation of this outbreak.

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