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Hepatitis A outbreak involving bread

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SUMMARY

An outbreak of hepatitis A involved more than 50 residents of a group of villages in the late spring and summer of 1989. The only food that was common to all the laboratory-confirmed cases was bread, purchased either unwrapped or as rolls, sandwiches or filled rolls, and supplied either directly from one shop or indirectly through its subsidiary outlets. It was concluded that this bread was the most likely vehicle of transmission of the hepatitis A virus and that the bread was contaminated by soiled hands which were inadequately washed because of painful skin lesions. Comprehensive control measures were successful in limiting further spread of the infection. This outbreak highlights the transmissibility of hepatitis A virus on food. The use of disposable gloves when handling food which is to be consumed without further cooking would prevent transmission of this or other infectious agents by this route.

INTRODUCTION

Hepatitis A is caused by a picornavirus and may be transmitted via food which is consumed without further cooking. There have been extensive outbreaks involving water [1], shellfish [2–4] and contaminated food [5, 6], some of which have been traced to infected food handlers [7]. We are not aware of a previous outbreak associated with bread. Here we describe an outbreak of hepatitis A involving more than 50 people where the evidence suggested that the vehicle of infection was contaminated bread.

THE OUTBREAK

The index case and her husband owned and ran a shop in a village in South Cambridgeshire with a population of 5000. The grocery section sold unwrapped and wrapped bread, sandwiches and filled rolls, in addition to vegetables, fruit and other comestibles. Bread and rolls were supplied by the shop to the canteen of a

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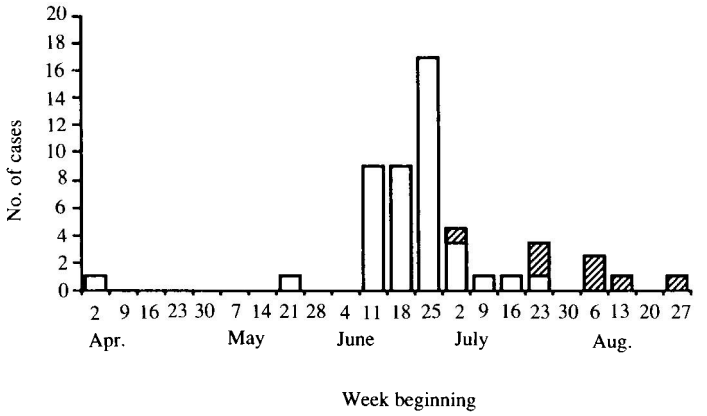

Fig. 1. Cases of hepatitis A April–September 1989 by date of onset of symptoms. □, primary cases; ▨, secondary cases.

food factory, and sandwiches and filled rolls were supplied to a number of public houses in the area. The owners also operated as outside caterers, providing sandwiches, rolls and filled rolls to a number of functions in the vicinity.

The index case became ill in early April 1989 and is presumed to have infected her husband, who, about 6 weeks later, developed a mild illness but continued to handle food. The husband ascribed his general malaise to pre-existing cardiovascular problems. After a further 3 weeks, cases of clinical hepatitis began to appear among the employees in a food factory (F1) and in the local population. Investigations by the Environmental Health Officers implicated the bread and bread rolls supplied to the factory canteen as the vehicle of transmission in a common source outbreak. At the same time bread supplied by the same shop and its various retail outlets was implicated in other cases in the local population. There were 68 notifications of cases of hepatitis in the primary outbreak including 43 laboratory-confirmed primary hepatitis A cases. There were seven laboratory-confirmed secondary cases (figure), who were household contacts of primary cases and became symptomatic approximately 4 weeks later.

Control measures

By 3 July, 20 cases of laboratory-confirmed hepatitis A had been notified, and extensive measures were taken to limit the spread of infection. All cases or contacts who were food handlers were advised of the risks of transmission of the virus. A public house closed temporarily. There were five symptomatic laboratory-confirmed cases in a local food factory (F1) which handled uncooked pork. Asymptomatic workers at this food factory, were tested for IgM and IgG anti-hepatitis A (anti-HAV) antibody to detect sub-clinical cases. Those asymptomatic workers who were not immune and who handled food were given prophylaxis with intramuscular normal human immunoglobulin (500 mg). None developed symptoms and all continued to work.

There was another food factory (F2) which employed people from the catchment area of the shop, and which processed cooked and uncooked meat. Although there were no clinical cases among these employees, those who lived within the catchment area of the shop were tested for IgM and IgG anti-HAV. Workers who

were IgG anti-HAV positive and IgM anti-HAV negative (indicating previous but not recent infection) were allowed to work in the cooked meat section of the factory; others were excluded.

Laboratory methods

Serum samples from suspected cases and all the workers in factories F1 and F2, were tested for IgM and IgG anti-HAV by IgM and IgG capture ELISA [8]. Saliva samples were collected, using a Salivette (Sarstedt, Leicester, UK), from asymptomatic controls in the village, 10 symptomatic laboratory-confirmed cases and 118 asymptomatic people who worked at factories F1 and F2. They were tested for IgM and IgG anti-HAV by class-specific RIA [9].

RESULTS

Serum samples from 50 of the suspected cases were IgM anti-HAV positive, indicating recent infection. No sub-clinical cases of hepatitis A were identified. A classical case control study was not possible because of poor compliance. However, a survey undertaken during the outbreak showed that under half of the local population had eaten bread or bread products from the suspected shop, whereas all primary cases had eaten bread or bread products originating from this shop. Systematic questioning revealed no other food common to the cases.

Tests for IgM and IgG anti-HAV in saliva yielded identical results to those found in 116 corresponding serum samples. None of the saliva samples from the 24 asymptomatic people from the same location contained IgM anti-HAV and only 4 (16·6%) had specific IgG indicating immunity to hepatitis A. Saliva samples from 15 of 57 (26·3%) workers in factory F1 and 17 of 61 (27·9%) asymptomatic workers in factory F2 contained IgG anti-HAV. None contained specific IgM and all were > 47 years old.

One patient whose first serum specimen was tested by ELISA 2 days after the onset of symptoms had no detectable IgM or IgG anti-HAV. A second serum taken from this patient on day 5 gave equivocal reactivity for specific IgM and IgG, and a third specimen taken on day 7 was unequivocally positive for IgM and IgG anti-HAV.

DISCUSSION

It is likely this epidemic occurred because the index case infected her husband, who subsequently transmitted the infection on to other cases via bread. He had several cuts and sores on his hands. He covered the lesions with adhesive dressings in line with health and hygiene recommendations. Because of the dressings, however, he was unable to wash his hands properly and they were visibly soiled. Presumably he contaminated the bread and sandwiches when he wrapped them up.

One patient did not produce a detectable IgM anti-HAV response, until 7 days after the onset of jaundice, which is a reminder that serum samples taken around the time of onset of symptoms may be negative. A serum sample taken at least 7 days after the onset of symptoms is required in order to exclude recent infection.

Sampling of saliva proved a simple and acceptable procedure which has been

shown to be as sensitive as sampling serum [9]. We obtained identical results in HAV antibody tests on serum and saliva from 116 patients. The availability of an RIA for testing saliva enabled us to test asymptomatic people from the location of the outbreak and to follow up asymptomatic factory workers. Tests were done on saliva alone on asymptomatic controls from the community and it is unlikely that we would have obtained the same degree of compliance had we sought serum samples.

We are unaware of any previous reports of outbreaks of hepatitis A transmitted by bread. This outbreak highlights the transmissibility of hepatitis A virus and the fact that the use of suitable disposable gloves while handling food which is to be consumed without further cooking would prevent transmission by this route.

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