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Escherichia coli O157:H7 in Malta

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Epidemiology

E coli O157:H7 was first recognized as a human pathogen in 1982, when it was associated with two separate outbreaks of haemorrhagic colitis in the USA[1]. Both outbreaks were linked to consumption of hamburgers. Since then, E coli O157:H7 has emerged as a leading food-borne pathogen, particularly in North America[2] where its household name ('hamburger disease') underlines the important association of ground beef with the condition.

Of particular public health concern are the serious complications of infection, namely haemorrhagic colitis and the haemolytic uraemic syndrome (HUS); the high frequency of secondary person-to-person transmission; the very low (possibly less than 100 organisms) infecting dose [3,4] and the established reservoir of the pathogen in cattle. In one review, 1% of healthy cattle were intestinally colonised with E coli O157:H7 [5].

Ground beef is the most common vehicle for E coli O157:H7 transmission in investigated outbreaks in the USA [6]. Infection through contaminated beef can occur if it is insufficiently cooked (internal temperature less than 68 °C, or visibly uncooked with oozing blood inside), or if such meat contaminates ready-to-eat food. Beef may be contaminated during slaughter, and the process of grinding beef may transfer pathogens from the surface to the deep layers of meat. Since multiple carcases may be involved in one ground beef production, a small proportion of infected animals in the 'pool' can cause widespread contamination.

Other less common vehicles of infection described include vegetables, unpasteurized cow's milk, unpasteurized apple cider, yoghurt, mayonnaise and unchlorinated drinking water. Swimming in contaminated lake water and contact with raw cow manure have also been implicated [7].

Person-to-person transmission has been an important mode of spread in day care centres [4,8]. It was responsible for an outbreak in a nursing home in Canada[9], and was the mode of transmission to staff in an outbreak in institutions for the mentally subnormal [10]. A nurse caring for a child with HUS was infected by direct contact with the patient [11].

Risk factors for infection include extremes of age [5], antimicrobial therapy administered early [8,9] and previous gastrectomy [8]. Occupational exposure to cattle, to minced beef and to microbiological specimens and cultures in laboratories are also known hazards [12,13,14,15].

Clinical features

E coli O157:H7 infection causes a wide spectrum of disease ranging from asymptomatic carriage to severe disease leading to death. Typical disease includes a non-bloody diarrhoea, haemorrhagic colitis, HUS and thrombotic thrombocytopaenic purpura [16]. While the incubation period is commonly 3-4 days, it can range from one to eight days [17,18,19].

Infections caused by E coli O157:H7 have been reported in many countries, and in many of them incidence appears to be increasing [20]. The recent large outbreak of EHEC in Sakai City, Japan generated wide press interest: between 12 July and 16 August 1996, 6561 persons were affected, mostly schoolchildren (96%). Ninety-two developed HUS and one died. Meanwhile, by 13 August, more than 9,200 individuals with EHEC were reported throughout the country, with nine associated deaths [21].

Microbiology

E coli O157:H7 belongs to the enterohaemorrhagic (EHEC) subgroup. It produces two distinct lysogenic, bacteriophage-encoded toxins that inhibit protein-synthesis and are active against cultures of Vero and HeLa cells. These Shiga-like toxins have been called verotoxin 1 (VT1) and verotoxin 2 (VT2). Isolates of human origin most commonly produce either VT2 or both VT1 and VT2. Over 62 different phage types have been recognised. Other less common EHEC serotypes causing disease in man include O26:H11, O111:H8 and O104:H21.

The detection of E coli O157:H7 in stools by using an appropriate selective and differential medium is a relatively straightforward and inexpensive procedure. Moreover, the labour involved in the review of plates is minimal. The toxin/s can also be detected directly from the stools by enzyme-linked immunosorbent assay techniques. Other laboratory diagnostic strategies are also available.

Irrespective of the method used, early collection of faecal specimens for detecting E coli O157:H7 is imperative. A positive culture is to be expected in 100% specimens collected within two days of onset of symptoms: this proportion drops to 33% if the sample is taken seven days or more from onset [22].

Antibiotic therapy of E coli O157:H7 infection is not usually recommended since it is generally ineffective and may even result in a worsening of the condition due to further release of toxin from killed intracolonic bacteria[23]. Subinhibitory concentrations of cotrimoxazole

have been shown to increase the production of Shiga-like toxin in vitro [24]. The prophylactic use of antibiotics is contraindicated; indeed this has been identified as a risk factor for the development of the condition [10,12]. Orally administered toxin-binding resins and antitoxin given intravenously [25] seem to hold more promise.

The local situation

Plans to introduce laboratory diagnostic facilities for E coli O157:H7 at St Luke's Hospital were made in 1993, and the service was eventually introduced in September 1994. Thanks to the introduction of this new diagnostic service, SLH Bacteriology Laboratory joined the European VTEC Club in 1994. The Club promotes communication amongst various groups throughout Europe studying EHEC infection.

It is currently the policy to screen routinely all stool specimens submitted for culture. All isolates of E coli O157:H7 are referred to the Public Health Laboratory in Valletta for phage typing and toxigenicity tests.

So far, two patients have been diagnosed with E coli O157:H7 infection, while a third clinically suggestive case could not be confirmed bacteriologically, possibly due to late submission of the stool specimen. The first two cases are the subject of an article in this issue [26].

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