a tube agglutination method, and those found to be E coli 0157 were grown in a modified brain heart infusion broth and tested for verotoxin production. Samples were also examined for free heart faecal verotoxin; if this was found in the absence of E coli 0157 then other toxigenic serogroups were sought by culture of the faecal sample on ordinary MacConkey agar (Oxoid CM7b) and by checking all E coli isolates for verotoxin production as above. All samples were also examined for the presence of other recognised enteric pathogens. All methods have been detailed in previous reports.

We found VTEC in 31 (78%) of 40 paucisemic patients with haemorrhagic colitis, but in only two (0.9%) of 229 control patients (p<0.001). With the exception of one verotoxin producing E coli 0128, all VTEC isolated were sorbitol negative E coli 0157. Other recognised enteric pathogens were not isolated from any patients with haemorrhagic colitis; they were recovered from 52 (23%) of 229 control patients.

Thus our findings differ sharply from those of Larson and Welch. Our findings are, however, similar to those of workers who studied sporadic cases of haemorrhagic colitis in England and Wales and Canada. We therefore disagree with the view expressed by Larson and Welch, and indeed regard VTEC, particularly serogroup 0157, as the major aetiological agent in sporadic cases of haemorrhagic colitis.

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Dr Larson comments:

If acute diarrhoea in adult patients caused by Salmonella, Shigella, Campylobacter and Clostridium species are regarded as separate clinical entities, haemorrhagic colitis not due to these organisms is the single most common clinical presentation of acute diarrhoea to our infectious disease unit. It was patients with this clinical presentation whom we subsequently studied for evidence of infection with verotoxin producing Escherichia coli and failed to find it. If these cases are, in fact, caused by infection with E coli 0157, this organism would be the single most prevalent gastrointestinal pathogen in the community. But I continue to be sceptical about this conclusion.

Dr Chapman, Wright, and Norman do not describe the patient population which was the source of the faecal samples they tested. Thus it is not possible to estimate a prevalence for E coli 0157 infection from their data (nor from data in their references 6 and 7) to compare it with our own. The percentage of cases of haemorrhagic colitis they found to be due to E coli 0157 is very high, however, significantly higher even than that found by Smith et al., or Pai et al. This suggests that they do not sporadic but epidemic haemorrhagic colitis.

Reference

Staphylococcus lugdunensis endocarditis

We were interested to read the description of a new type of staphylococcal endocarditis by Smyth et al. No species identification was done for the staphylococcal strain which has most of the characteristics of Staphylococcus lugdunensis, a new coagulase negative staphylococcal species. This species produces smooth, glossy colonies, initially cream-coloured, but becoming pale yellow to golden after five days. Using the API Staph gallery (API-Système, Montalieu-Vercieu, France), S lugdunensis is incorrectly recognised as S hominis biotype 3. Such strains are identified as S lugdunensis if they have an ornithine decarboxylase and a fibrinogen affinity factor. They also have a thermostable DNAase activity, like S aureus but not like S epidermidis.

S lugdunensis, however, is probably more responsible for infections such as infective endocarditis. Three cases of infective endocarditis due to S lugdunensis occurred in France in 1977, 1982, and 1983. The three strains, primarily recognised as coagulase negative staphylococci close to S hominis but with atypical characters, were correctly identified in 1982. Unlike the usual hospital S epidermidis isolates, the strains were susceptible to all the antibiotics tested (benzylopenicil, meticillin, aminoglycosides, chloramphenicol, tetracycline, macrolides, fusidic acid, vancomycin). If S lugdunensis seems, like S epidermidis and S saprophyticus, to be a coagulase negative staphylococcus isolated from human infections, its correct identification is a necessity and can be done easily. Coagulase and fibrinogen affinity factor (clumping factor) must both be detected, and the positivity of the second test suggests only that the isolate is S lugdunensis; its definite identification is achieved by the other biochemical tests, including ornithine decarboxylase detection.

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References

Dr Smyth et al comment:

We agree that the organism we described as causing endocarditis can probably be identified as S lugdunensis, a new species first described by Etienne et al in 1988. The description was not available to us at the time we studied the strain from this patient, but we were cognisant that ornithine decarboxylase positive staphylococci resembling