Even with these sensitive tests VTEC infection was only demonstrated in some cases.

For the investigation of HUS every effort should be made to detect VTEC infection. However, in cases where VTEC cannot be isolated and no faecal verocytotoxin found alternative tests must be considered. One such test for implicating E coli O157 is to demonstrate a serum antibody response. Sera from 13 patients with HUS and from whom E coli O157 had been isolated and 8 healthy controls were examined for antibodies to E coli O157 antigens by sodium dodecyl sulphate-polyacrylamide gel electrophoresis with immunoblotting. Sera from the patients gave a strong IgM antibody reaction with E coli O157 lipopolysaccharide (LPS), while control sera did not react with O157 LPS.

Dr Kavi and Dr Rose (May 28, p 1224) raise concern about the validity of using serum antibodies to identify the aetiological infective agent in HUS. Antibody cross-reactions between the O-antigens of E coli O157, Yersinia enterocolitica (O:9), and Brucella abortus may be possible since the LPS of these bacteria share common sugar sequences. Indeed antigenic cross-reactions have been demonstrated with bacterial agglutination tests between strains of B abortus and Y enterocolitica (O:9), and between B abortus and E coli O157. On the other hand, E coli O157 and Y enterocolitica (O:9) did not cross-react. By immunoblotting we found antibodies in our HUS patients’ sera which reacted strongly with the LPS of B abortus but not with the LPS of Y enterocolitica (O:9), which agrees with Notenboom et al. Our studies and the published reports indicate that the major source of antibody cross-reaction is between the LPS of E coli O157 and B abortus, and probably not between E coli O157 and Y enterocolitica. The use of a serological test to implicate a particular infective agent in HUS is inconclusive and serological results should be considered carefully and interpreted in relation to the patient’s symptoms.

PRAZOSIN CONTRAINDICATED IN PATIENTS WITH NARCOLEPSY

SIR,—Mignot et al have demonstrated that central α2-adrenoceptors are involved in cataplexy in dogs. Prazosin hydrochloride, a highly selective α2-adrenergic receptor antagonist that easily crosses the blood–brain barrier, was found to be a potent cataplexy-inducing agent in narcoleptic dogs. We contacted several narcolepsy and HUS patients’ sera which reacted strongly with the LPS of B abortus but not with the LPS of Y enterocolitica (O:9), which agrees with Notenboom et al. Our studies and the published reports indicate that the major source of antibody cross-reaction is between the LPS of E coli O157 and B abortus, and probably not between E coli O157 and Y enterocolitica. The use of a serological test to implicate a particular infective agent in HUS is inconclusive and serological results should be considered carefully and interpreted in relation to the patient’s symptoms.

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