Hepatitis G virus in intravenous immunoglobulin - Reply

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Hepatitis G Virus in Intravenous Immunoglobulin

Dear Sir:

Healey et al. conducted a comprehensive study of an acute hepatitis outbreak in patients receiving a particular batch of Gammagard intravenous (IV) immunoglobulin (Ig) and attributed the cause to hepatitis C virus (HCV) that was not completely inactivated. A routine follow-up of liver function tests in all IV Ig recipients is therefore recommended.

Two additional points are worthy of discussion. First, is there any cotransmission of other hepatitis viruses by this batch of contaminated IV Ig? Most imminently, the recently identified hepatitis G virus/GB virus-C virus (HGV or GBV-C) should be examined. HGV can be transmitted by transfusion; however, it has never been screened in blood donors. With a viremia rate of about 1% among healthy, immunocompromised, might lead to significant morbidity and C. J. HEALEY merits thorough investigation. If IV Ig-transmitted HGV is confirmed, a phylogenetic tree analysis of these viruses is also interesting.

In contrast to that of IV Ig-transmitted HCV, the analysis would very likely reveal heterogeneous origins of the HGV infection because of more HGV-carrying donors in the plasma pool. This information should be useful in understanding the risk of HGV infection in the contaminated IV Ig and in further defining the role of this new virus in causing human hepatitis.


Reply. We agree with Chen et al. that cotransmission of other hepatitis viruses should be considered. During the initial outbreak, coinfection with other hepatotropic viruses (hepatitis A and B viruses, Epstein–Barr virus, and cytomegalovirus) was excluded. More particularly, our collaborators, Jarvis et al., have examined batches of Gammagard IV Ig and serum from recipients of the HCV-contaminated batch of Gammagard for the presence of HGV. Six of six batches tested, including the contaminated batch (9E21AB11B) of Gammagard, were positive for HGV. However, HGV has not been detected in any U.K. recipients of this contaminated batch tested (14 of 14 serum samples from 3 to 6 months after exposure). These data suggest that if HGV transmission occurred in these patients, it was not associated with chronic infection or clinical episodes of hepatitis. However, the exact interaction between the immune response to repeated inoculation with HGV RNA and the subsequent HCV outbreak cannot be known. It is possible that such responses to HGV, which shares considerable homology to HCV, may contribute to the severity of acute HCV infection in immunodeficient patients found in both this and previous outbreaks of HCV.

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Abnormal Intestinal Motility in Irritable Bowel Syndrome

Dear Sir:

We read with interest the paper on jejunal sensorimotor dysfunction in irritable bowel syndrome (IBS) by Evans et al.1 They concluded that abnormal postprandial jejunal motor activity is related to jejunal mechanoreceptor hypersensitivity. In their study, which compared 11 patients with diarrhea-predominant IBS with 25 healthy controls, abnormal phase 2 and phase 3 activities were found in 75% and 64% of patients, respectively. Postprandial motor activity was abnormal in 36% after an 800-kcal meal and in 100% after a 400-kcal meal.

In a recent study, we analyzed 24-hour ambulatory jejunal manometry in 35 patients with diarrhea-predominant IBS and 50 healthy controls.2,3 Abnormal motility, defined as at least one motility variable outside the range of controls, was found in only 43% of patients, with an abnormal postprandial motility after a 600-kcal meal present in only 14%. Abnormalities in the fasting state were disturbed aboral migration of phase 3 (6%), prolonged irregular burst activity (3%), increased duration of clustered activity (23%), and increased contraction amplitude (23%) and increased proportion of aborally propagated contractions (5%) during phase 2. In the postprandial state, an increased percentage of propagated contractions was the only abnormal finding.

The results obtained by Evans et al.1 are obviously discrepant to several previous studies,4-6 including our own data. Besides different study conditions (patient selection, ambulatory vs. stationary recordings, and standardized food intake vs. self-selection of meals) and the heterogeneity of the IBS, we feel that another reason to explain these differences might be the size of the patient and control group.

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Reply.

We thank Schmidt et al. for their interest in our recent paper.1 They refer specifically to the 11 patients with diarrhea-predominant IBS in our sample of 24 patients with IBS and discuss the rates of small bowel motility abnormalities that we found in this subgroup.

The main thrust of our paper was to evaluate the relationships between enteric hypersensitivity, dysmotility, and psychosocial factors in IBS; thus, we did not present detailed data on the specific type of abnormalities observed in each of our subgroups. Apart from their own study,2 the studies to which Schmidt et al. refer3-5 cannot be compared directly with our recent study because of differences in experimental protocols and data analysis. In particular, only between-group comparisons of parameters were undertaken in these latter studies, and rates of abnormality were not determined. Moreover, postprandial recordings were not obtained in one of these studies,3 and, in the other two studies,4,5 postprandial motility was not analyzed quantitatively. The test meals also differed between our study and that of Schmidt et al.2 In regard to the study of Schmidt et al.,2 it is likely that the small bowel motor parameters that were analyzed differed from those evaluated in our study. It is not stated which parameters were analyzed in their study and whether, for example, motility indices or the presence of phase 3–like activity during the fed pattern were determined. We observed alterations in these latter parameters postprandially in our patient group. We acknowledge that larger control groups may result in wider “normal ranges”; however, in determining rates of abnormality based on one or more parameters outside a control range, the results are clearly also dependent on the number and type of parameters evaluated. This in turn may be dependent on the type of computer-assisted analysis used.

Overall, because of the variations in study methodology and data analysis, we cannot agree that the results obtained in our recent study are “obviously discrepant” to those of earlier studies. Finally, and perhaps most importantly, in addition to the problems of patient heterogeneity in IBS, as outlined by Schmidt et al.,1 it is clear that until the methodology and analysis of human small bowel motility recordings can be standardized between centers, differences will continue to be apparent.

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