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Letter to the Editor

LETTER TO THE EDITOR

Response to: 'Chronic genotype 1 hepatitis E infection from immunosuppression for ileo-colonic Crohn's disease'

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To the Editor,

'Chronic genotype 1 hepatitis E infection from immunosuppression for ileo-colonic Crohn's disease'

We read with interest the case report by Robins and colleagues in which the authors describe what they claim to be the first case of genotype 1 (G1) hepatitis E virus, known to be of a Indian subcontinent phylotype consistent with reported travel, leading to chronic infection in a patient undergoing immunosuppression for inflammatory bowel disease [1].

The importance of this case should not be underestimated because the anthropotropic genotypes of HEV (G1and G2) have previously not been reported in association with chronic infection. Where studies have looked for chronic infections in G1-endemic areas cases have not been found [2]. This case has important implications in areas of the world where G1 HEV is common and patients may be iatrogenically immunosuppressed, such as India [3]. It also has important implications for travel advice for immunocompromised patients, their potential infectivity and to our fundamental understanding of pathogenic differences between HEV genotypes.

There are, however, several key points to the case, which render this unlikely to represent persistence and require further clarification. Crucially, the authors claim that this is a case of chronic hepatitis E but no virological data have been presented to support this conclusion. The current definition of chronic HEV infection is the persistence of HEV RNA in plasma for at least 3 months [4]. We would also expect the viraemia to be stable during this time, thus it would be important to provide quantitative viral load results for this case. In contrast, at the point of initiation of treatment the HEV RNA cycle threshold is reported to have fallen from 23 at presentation to a cycle threshold of 35. By any account, an apparent reduction in viraemia by $12 \times \log_2$ indicates an approximate 4000-fold fall in HEV RNA during the period before treatment and strongly suggests that the patient was spontaneously clearing the infection at this time. The patient was reportedly on high dose steroids which may well have slowed the immune response and thus prolonged viraemia. It is unfortunate that no data on viral shedding in stool both prior to and during treatment and on the development of the antibody were presented. It is well recognized that a proportion of HEV infections in the immunosuppressed host spontaneously resolve and it is probable that this was occurring at the time that treatment was initiated. It is for this reason that a period of surveillance is recommended before therapeutic intervention [4]. We believe that further details on viral load dynamics and serological markers are needed to better understand the implications of this case.

CONFLICT OF INTEREST

The authors declare no competing interests.

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