Letter to the editor in response to article: Reversal of Acquired Hepatocerebral Degeneration with Living Donor Liver Transplantation

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Cover letter

To the Editor,

Please find enclosed a letter to the editor in chief of Liver Transplantation. This is in response to the following article: Qavi AH. Hammad S. Rana AI. Salih M. Shah NH. Dar FS. et al. Reversal of Acquired Hepatocerebral Degeneration with Living Donor Liver Transplantation; Liver transplantation 2016;22:125-129. We have identified an error and wish to make a correction.

I give written assurance that this material has not, and is not being considered for publication elsewhere. As the corresponding author I speak on behalf of the co-authors and certify that all listed authors have participated meaningfully in the manuscript and they approve the final material. I also wish to declare a potential conflict of interest in that Dr Debbie Shawcross, co-author, has served as an advisory board member and has received lecture fees from Norgine.

Sincerely,

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Dr Debbie L. Shawcross, PhD

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To the Editor,

We have read with great interest the recently published single centre retrospective study by Qavi et al. (1). This study demonstrated marked clinical and complete radiological resolution of acquired hepatocerebral degeneration (AHD) in three patients following live donor liver transplantation. However, we wish to provide clarity with regards to the authors’ statement that there is no medical treatment that has been shown to reverse the progression of AHD.

Whilst we agree that liver transplantation provides an option to reverse ADH, this is clearly associated with significant morbidity, mortality, and resource implications. Furthermore, liver transplantation assessment is complex and may not be feasible in patients who are elderly, have complex morbidities or are too frail to undergo major surgery. A case series by Kok et al. (2) published in Hepatology in 2013 demonstrated significant clinical and radiological resolution of ADH in three patients following treatment with 600mg rifaximin, twice daily for four weeks. Comparable to the MRI brain findings by Qavi et al. (1), all patients had a reduction in the high T1 signal within the globus pallidus. Additionally, there were improvements in objective measures of disease severity, using neuropsychometry testing, in all patients post-rifaximin.

We thought it would be prudent to highlight the potential utility of rifaximin in AHD, particularly in the authors cohort of non-transplanted patients who were older (mean age 58.3 years vs 38.6 years) and with more complex comorbidities, including type two diabetes, hypertension and cerebrovascular disease. In such patients, medical management with rifaximin may provide an alternative to liver transplantation in managing AHD.

Abbreviations:
MRI: Magnetic resonance imaging.
AHD: Acquired hepatocerebral degeneration

References:
2. Kok B. Foxton M. Clough C. Shawcross D. Rifaximin is an efficacious treatment for parkinsonian phenotype of hepatic encephalopathy; Hepatology 2013; 58(4):1516-1517

Sincerely,
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