abnormal LFTs. After loss to follow-up, repeat LFTs remained abnormal and a liver biopsy (2009) showed established cirrhosis with active inflammation. Chronic HEV infection was documented with persisting viraemia in serum and stool over a 3 year period (Jan 2007—Jan 2010). HEV was also recovered from his cerebrospinal fluid. He remained antiretroviral naïve until January 2007, when he commenced on tenofovir/emtricitabine and lopinavir/ritonavir. Although transiently switched to efavirenz he settled on his current regimen of abacavir/lamivudine and lopinavir/ritonavir in June 2007. His HIV viral load became undetectable but his CD4 count remained low (100—170 /mm^3).

Results In July 2009 he was treated with Pegasys 135 microg/week. His LFTs normalised and his HEV viral load declined. At 6 months he achieved HEV clearance from his serum, but HEV was still detectable in stool. Ribavarin 1 g/day was added in for a further 3 months. HEV viral clearance was achieved from serum and stool. During treatment, neurological symptoms improved and, by the time viral clearance was achieved, they were abolished. He remains HEV PCR negative 3 months after completion of therapy.

Conclusion Chronic HEV infection can occur in the context of HIV and can be associated with neurological symptoms. HEV viral clearance can be achieved by combination therapy with Pegasys/ribavarin, with normalisation of LFT's and resolution of neurological symptoms. The mechanism of neurological damage in HEV infection is uncertain.

REFERENCE

1. **Dalton HR,** Bendall R, Keane F, *et al*. Persistent carriage of hepatitis E virus in patients with HIV Infection. *N Engl J Med* 2009;**361**:1025—7.

P62 BACTERIAL TRANSLOCATION IN HUMAN IMMUNODEFICIENCY VIRUS-INFECTED PATIENTS WITH HEPATITIS C VIRUS-RELATED CIRRHOSIS: IMPLICATION IN HAEMODYNAMIC ALTERATIONS AND MORTALITY

doi:10.1136/gut.2010.223362.88

[1]M Montes de Oca, [1]M Coello Mercedes, [1]S C M Jose, [2]R Claudio, [1]T Alberto, [1]V Antonio, [1]F G Clotilde, [1]C Sara, [1]G G J Antonio. *[1]Internal Medicine, Puerta del Mar Universitary Hospital, Spain; [2]Gastroenterology, Jerez General Hospital, Spain*

Introduction Hepatitis C virus (HCV)-related liver disease follows an accelerated course in patients with human immunodeficiency virus (HIV) co-infection. Liver fibrosis progression to cirrhosis is faster in HIV/HCV coinfected individuals than in HCV-monoinfected subjects. Immunosuppression (lower CD4 cell count), markers of severe liver disease (Child Pugh score) and the absence of HAART are independent predictors of liver-related mortality in patients with end-stage liver disease coinfected with HIV and HCV. However, the intrinsic mechanisms through which HIV infection have an influence in the worse prognosis of these patients remain unknown. Among the possible mechanisms involved in the evolution of both HIV and HCV infection in coinfected patients, we suggest that microbial translocation may contribute to the accelerated course of liver damage in HCV-HIV patients through the activation of immune system.

Aim Analysis of the influence of portal hypertension on intestinal permeability in HIV-infected patients with hepatitis C virus (HCV)-related cirrhosis, as well of the prognostic significance of consequent macrophage activation.

Method 20 HIV-monoinfected patients, seventy patients with HIV-HVC co-infection, twenty of them with compensated and fifty with decompensated cirrhosis, and twenty healthy controls were evaluated for intestinal permeability and bacterial translocation (lipopolysaccharide-binding protein (LBP)), macrophage activation (soluble CD14 (sCD14), soluble tumour necrosis factor receptor 55 Kd (sTNFRI), and interleukin 6 (IL-6)), and activation of the renin-angiotensin-aldosterone axis. Patients with decompensated cirrhosis were followed during a median period of 429 days.

Results LBP concentration was significantly increased in HIV monoinfected patients when compared with healthy controls. Patients with decompensated cirrhosis, but not those with compensated liver disease, show increased LBP levels when compared with HIV monoinfected patients. Patients with increased LBP concentration showed elevated sCD14, sTNFRI and IL-6 levels. 22 patients died, by a liver-related cause, during the follow-up and two more suffered liver transplantation. The mortality at 1 and 2 years was 36 and 56 %. Child-Pugh index, CD4 T cell count, plasma aldosterone and serum IL-6 concentrations independently predicted liver-related mortality.

Conclusion The increased intestinal permeability, observed in patients with monoinfection by HIV, is significantly higher in those with concomitant portal hypertension. Parameters indicative of macrophage activation, such as IL-6, in addition of immunodepression, liver function markers and haemodynamic derangement (plasma aldosterone concentration) influence the survival of HIV-HCV co-infected patients with decompensated cirrhosis.

P63 NEUROLOGICAL SEQUELAE OF ACUTE AND CHRONIC HEV GENOTYPE 3 INFECTION

doi:10.1136/gut.2010.223362.89

[1]H Dalton, [2]N Kamar, [1]H R Dalton, [1]R Bendall, [1]F Keane, [2]J M Peron, [2]P Cintas, [3]L Pruhomme, [2]J M Mansuy, [2]L Rostaing, [3]S Ijaz, [2]J Izopet. *[1]Peninsula College of Medicine and Dentistry, Royal Cornwall Hospital, UK; [2]Universite Paul Sabatier, France; [3]Centre for Infections, Health Protection Agency, UK*

Introduction HEV is an emerging infection in the developed world. It differs from HEV endemic in developing countries as it is caused by HEV genotype 3, is thought to be a porcine zoonosis, has a predilection for middle aged/elderly males, and can cause chronic infection in the immunosuppressed. Neurological symptoms associated with HEV infection have been described in both developed and developing countries. However, to date, no systematic study of the neurological sequelae of HEV has been undertaken.

Aim To describe the clinical and laboratory features of the neurological complications of HEV genotype 3 infection.

Method Retrospective review of 126 patients with locally acquired acute and chronic HEV genotype 3 infection from two University Hospitals: Truro (UK) and Toulouse (France).

Results Neurological complications were seen in 7/126 (5.5%) of patients with HEV. The neurological complications included inflammatory polyradiculopathy (n=3), Guillain-Barré Syndrome (n=1), bilateral brachial neuritis (n=1), encephalitis (n=1), ataxia/proximal myopathy (n=1). Three cases occurred during acute HEV infection in non-immunocompromised patients and four occurred in immunocompromised patients with chronic HEV infection (three transplant patients, and one HIV-positive patient). HEV RNA was detected in the cerebrospinal fluid (CSF) of all four patients with chronic HEV infection and neurological symptoms. In one of these cases unique HEV quasispecies were documented in the CSF. In patients with acute HEV who had neurological symptoms, HEV RNA was absent in the CSF (n=2). Follow-up was up to 4 years and neurological outcomes were: complete resolution (n=3), improvement with residual neurological deficit (n=3), no improvement (n=1).

Conclusion Neurological symptoms are an emerging extra-hepatic manifestation of HEV infection. The pathophysiology is not understood, but HEV may be neurotropic, as unique HEV quasispecies were isolated from the CSF of one of our cases.

Acknowledgements We thank the Royal College of Physicians (London) for supporting this study by the award of the Dame Sheila Sherlock Travelling Fellowship.