A) COMMENTS ON PUBLISHED ARTICLES

Commentary on Treatment of HCV infection in chronic kidney disease

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To the Editor,

I read with interest the published article by Aoufi Rabih[1] in your journal recently. The prevalence of Hepatitis C virus (HCV) infection in hemodialysis (HD) patients varies markedly from country to country[2]. The main risk factors for HCV infection in this special group are blood transfusions, length of dialysis time and nosocomial routes of transmission including the use of contaminated equipment and patient-to-patient exposure[3-6]. Control of HCV infection in hemodialysis setting is possible[7,8]. Integration of surveillance system for early detection, treating all of treatable patients with alpha interferon, putting HCV-infected patients on the top list for renal transplantation, training the staffs in hemodialysis patients and using more the erythropoietin instead blood transfusion. The prevalence of HCV infection may be underestimated according to an antibody assay alone[9]. First of all, i would like to present a dilemma regarding liver biopsy in hemodialysis patients with HCV infection. Liver biopsy in hemodialysis patients is with higher risk of bleeding and other complication and it should do by Trans-jugular or in very specialized center. In treatment of HCV infection in hemodialysis patients, we are not sure regarding superiority of pegylated interferon (IFN) on conventional IFN[10,11] and in a meta-analysis The pooled sustained virologic response (SVR) for standard and pegylated IFN monotherapy in random effects model was 39.1% (95% confidence interval [CI], 32.1 to 46.1) and 39.3% (95% CI, 26.5 to 52.1), respectively[10]. The difference was not significant, but it is important to treat the patients before 40 years old and as soon as possible[10,12]. Individuals on dialysis with chronic hepatitis C who were treated with interferon or pegylated interferon plus ribavirin can have higher SVR rate than dialysis patients treated with interferon or pegylated interferon alone. Administration of ribavirin with close monitoring of CBC and serum ribavirin concentration can be safe[13].

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S.M. Alavian
Director of Baqiyatallah Research Center for Gastroenterology and Liver Disease. Tehran (Iran).
Correspondence: S.M. Alavian
Director of Baqiyatallah Research Center for Gastroenterology and Liver Disease. 14155/3651. Tehran. Iran.
alavian@thc.ir

Response to the comment made on Treatment of HCV infection in chronic kidney disease

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To the Editor,

Haemodialysis units' health policies and protocols are disparate meaning that prevalence of chronic hepatitis C infection is extremely variable in the haemodialysis population.[1-3] In some countries classic infection factors, such as material contaminated due to reuse or blood transfusions, have been

replaced by parenteral drug addiction or sexual transmission.[4,5] It is not surprising that prevention is the most adequate and cost-effective control measure for these patients.

Treating chronic HCV before kidney transplantation is currently not an essential criteria for including HCV-positive patients on the transplant waiting list. However, risk of post-transplant chronic hepatitis C and the difficulty to treat it at this stage of the chronic kidney disease have been reported.[6-11]

Pegylated interferon and interferon + ribavirin are better than conventional interferon, according to clinical trials. However, the differences are small. Combining ribavirin and pegylated interferon requires close follow-up during haemodialysis given the severity of the secondary effects. It has increased the sustained viral response, although it is still less in the population without chronic kidney disease.[12] This, along with the difficulty of treating patients with stages 4 and 5 chronic kidney disease in predialysis, highlights the importance of resolving the infection at early kidney disease stages.

Transjugular liver biopsy reduces the risks of bleeding associated with this procedure and the kidney patient, although there is little evidence in the literature.[13,14] This technique also allows the hepatic venous pressure gradient to be measured, providing diagnostic and prognostic data.

Studies that determine whether the association of protease inhibitors (telaprevir, boceprevir) with interferon and ribavirin is safe for kidney patients and may increase viral response rates.

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R. García Agudo[1], S. Aoufi Rabih[2]

[1] Nephrology Department. La Mancha-Centro Hospital Complex. Alcázar de San Juan, Ciudad Real, Spain.

[2] Digestive System Department. La Mancha-Centro Hospital Complex. Alcázar de San Juan, Ciudad Real, Spain.

Correspondence: R. García Agudo
Servicio de Nefrología. Complejo Hospitalario La Mancha-Centro. Enebro, 17.
13600, Alcázar de San Juan. Ciudad Real.
rgarciaagudo@hotmail.com
rganefrologia@hotmail.com

Cyclophosphamide-induced lupus flare?: the role of C4 and interferon-gamma in lupus flare

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To the Editor,

We read with great interest the contribution by Heras, et al.[1]. They reported a significant case that seemed not to respond to intravenous (IV) cyclophosphamide (CPM) induction treatment at 1 g but to respond to increased CPM dose to 1.5 g. Reading the case report, we wondered whether CPM certainly induced the lupus flare or other mechanisms were involved in the pathogenesis. They speculated