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LETTER TO THE EDITOR

Shear wave velocity might correlate with portal venous perfusion if correct portal venous perfusion techniques are used

Yoshito Tsushima[1] · **Ayako Taketomi-Takahashi**[1]

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Dear Editor:

We read the article by Esser and colleagues published in the January issue of your journal with interest. They correlated liver stiffness (on acoustic radiation force impulse: ARFI) with liver perfusion parameters (on maximum slope method perfusion CT) in 50 patients with liver cirrhosis [1]. Their finding of a significant linear positive correlation between tissue stiffness (shear wave velocity: SWV) and Child–Pugh staging was consistent with previous studies. However, portal venous perfusion (PVP) values in their study were, surprisingly, much lower than those in previous perfusion CT studies, and lacked correlation with SWV.

In the maximum-slope method, organ perfusion is calculated by dividing the peak gradient of the time density curve of the tracer (i.e., CT number increase) in the target organ by the peak increase of the input function [2–3]. For arterial liver perfusion (ALP), the input function would be for the hepatic artery (reasonably approximated by that of the aorta). ALP is reported to be approximately 10–30 ml/100 ml/min in normal liver [3–7], and may increase in liver cirrhosis [3, 8]. In Esser's study [1], the median value of ALP of liver cirrhosis was 13.2 ml/100 ml/min, consistent with previously reported values, and suggesting correct hepatic perfusion CT technique. For PVP, the input function would be for the portal vein. PVP is reported to be around 100 ml/100 ml/min in normal liver and decreases as liver disease progresses. Reports of PVP in cirrhosis range from 43 to 67 ml/100 ml/min [3–4, 9]. Using values from the aorta to derive the input function for PVP would result in an "indirect PVP" that would be approximately one-fifth of a "direct PVP" derived from a portal vein input function [2, 10]. Esser reported that median PVP was 8.4 ml/100 ml/min in cirrhosis [1], which is approximately one-fifth of previously reported values. This leads us to think that the values they obtained might have been those of "indirect (i.e., aortic input function)" PVP. If this is the case, total liver perfusion (TLP) calculated by adding ALP to an "indirect" PVP would obviously not reflect hepatic function (because the aorta does not supply and is unrelated to portal circulation) and would obviously lack correlation with SWV.

We hope the authors will recalculate PVP with a portal vein input function. We suspect that those values will show the expected significant negative correlation with SWV.

Compliance with ethical standards

Conflict of interest We do not have any COIs that should be disclosed.

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✉ Yoshito Tsushima
yoshitotsushima@gunma-u.ac.jp

[1] Department of Diagnostic Radiology and Nuclear Medicine, Gunma University Graduate School of Medicine, Maebashi, Japan

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