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Letter to the editor

Budd-Chiari syndrome hemodynamic: Clinical setting more complicated than computational model

I read with interest the study, recently published in *Medical Engineering & Physics*, reporting that, using a computational model (CM) of Budd-Chiari syndrome (BCS), in consequence of hepatic veins (HV) thrombosis there would be a reduced total portal flow and increased hepatic arterial flow [1]. Overall, the CM findings are in line with the belief that regenerative nodules, a common finding in BCS, and probably hepatocellular carcinoma could be triggered by increased hepatic arterial flow [2].

However, in the real life clinical setting, using common diagnostic tools just like Doppler ultrasound (US), in BCS there are generally not significant changes of hepatic arterial flow, differently from what usually happens in cases of portal vein thrombosis, when it is usual to find a significantly increased arterial flow.

In fact, the interesting model proposed does not consider two main issues.

The former is that, despite the geographical difference between BCS in the West and the East [3,4], BCS is generally the consequence of a chronic thrombotic disease of HV. In fact, irrespective to the geographical areas, apart from acute/fulminant cases, complications of BCS likely appear after months or years HV or Inferior Vena Cava (IVC) obstruction has instituted [5]. Moreover, most patients at presentation have extensive fibrosis or cirrhosis in liver biopsies and, finally, subcutaneous and/or abdominal venous collaterals are often evident at diagnosis [6,7].

The latter is that, due the generally chronic course of BCS, it is clinically frequent the observation of intrahepatic venous collaterals bypassing the obstruction, aiming at compensating liver congestion due to HV thrombosis, so rendering the model proposed too simple for clinical application [1].

Consequently, first of all a CM should take in consideration chronic development of HV thrombosis and, consequently, gradual development of intrahepatic collaterals, to improve the likelihood of the model to the real-life BCS.

In conclusion, I believe that the CM of BCS represents an important starting point but needing substantial improvements to adequate it to clinical settings.

Conflict of interest

I declare I have nothing to disclose.

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Trial registration number

Not applicable.

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I declare I contributed to this paper.

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Andrea Mancuso*
Medicina Interna 1, ARNAS Civico - Di Cristina - Benfratelli, Piazzale Leotta 4, Palermo, Italy
*Corresponding author: Prof Andrea Mancuso, MD, Medicina Interna 1, ARNAS Civico, Piazzale Leotta 4, 90100, Palermo, Italy.
E-mail address: mancandrea@libero.it

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Abbreviations: CM, computational model; BCS, Budd-Chiari syndrome; HV, hepatic veins; US, ultrasound; IVC, Inferior Vena Cava.

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