

## Circulating platelet derived microparticles are not increased in patients with cirrhosis

To the Editor:

In their *Snapshot*, Tapper, Robson, and Malik suggest that circulating platelet-derived microparticles are increased in cirrhosis as a function of systemic inflammation [1]. Ogasawara and colleagues observed higher levels of circulating platelet microparticles in 22 patients with cirrhosis as compared to 17 healthy controls [2]. However, the results of two other independent studies assessing circulating platelet microparticle levels in patients with cirrhosis do not concur. Sayed and colleagues included 60 patients with hepatitis C related cirrhosis and 20 healthy controls [3]. Platelet microparticle levels, expressed as a percentage of the total platelet count, were not different between patients with cirrhosis and controls [3]. When calculating their circulating concentration, one can conclude that the platelet microparticle level was even lower in patients with cirrhosis than in controls ( $14.2 \times 10^9/L$  vs.  $36.5 \times 10^9/L$ , respectively). Our group also did not observe any difference in circulating levels of platelet microparticles between 26 patients with cirrhosis and 30 healthy controls [4]. In addition, there was no correlation between circulating levels of platelet derived microparticles (CD41<sup>+</sup>) and inflammatory markers, either in the pilot cohort (C-reactive protein: Spearman correlation coefficient = 0.412,  $p = 0.071$ ,  $n = 20$ ; leukocytes: Spearman correlation coefficient = 0.272,  $p = 0.179$ ,  $n = 26$ ) or in the additional cohort (C-reactive protein: Spearman correlation coefficient = 0.211,  $p = 0.129$ ,  $n = 53$ ; leukocytes: Spearman correlation coefficient = 0.068,  $p = 0.597$ ,  $n = 63$ ). Therefore, the currently available data demonstrate that platelet derived microparticles are not systematically increased in patients with cirrhosis and likely do not contribute to the procoagulant imbalance associated with cirrhosis.

Certain subpopulations of microparticles also express tissue factor, the primary initiator of coagulation, at their surface. These microparticles are highly procoagulant [5]. Platelets are not a major source of tissue factor positive microparticles in healthy individuals and patients [6]. It remains to be determined whether these circulating tissue factor positive microparticles are increased in patients with cirrhosis and contribute to the procoagulant imbalance in this setting.

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### Conflict of interest

The authors declared that they do not have anything to disclose regarding funding or conflict of interest with respect to this manuscript.

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