No central venous pressure protein losing enteropathy relation? Blame the albumin

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I read, with great interest, the paper ‘Haemodynamic characteristics before and after the onset of protein losing enteropathy (PLE) in patients after the Fontan operation’ by Ohuchi et al. [1]. Reviewing 354 Fontan survivors, the authors successfully stratify them for the risk of PLE using the central venous pressure (CVP) measured early postoperation. The significant difference in CVP between those 26 affected with PLE and 56 others considered ‘excellent Fontan survivors’ then disappears at the time of PLE diagnosis. The authors speculate that the failure to find a relationship between CVP and PLE at this point is due to the vasoactive drug regimens these patients inevitably endure.

Another explanation could be the drop in oncotic pressure due to hypoalbuminaemia, the primary consequence of PLE. As Bull notes, the maximum sustainable blood pressure in mmHg, given an albumin level of A (g/100 ml), is given by CVP = 5.2 × A [2]. Above this pressure, systemic or pulmonary oedema is generated. The authors report a mean drop of 2.6 mmHg in patients’ CVP at the time of PLE diagnosis from their immediate post-Fontan measurement. This would correspond to a mean drop of 0.5 g/100 ml in albumin levels.

A potentially useful consequence of this relation is in treatment planning for patients at diagnosis of PLE. Correcting CVP values for albumin could predict those who would benefit from interventions to lower pulmonary vascular resistance, either pharmacologically through the use of sildenafl or through use of a stent.

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REFERENCES

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