Paracentesis-Induced Circulatory Dysfunction

Drew Brotherston[1], Alejandro Dau[2], Abbas Albaghli[2], Shane Arishenkoff[2]
[1] Division of General Internal Medicine, University of Calgary, [2]Division of General Internal Medicine, University of British Columbia

Definitions and Significance

PICD is a broad term referring to circulatory dysfunction following Large-Volume Paracentesis (LVP, >5L drained), as a result of RAAS activation. *It can persist up to six days post-paracentesis*.

-Formally defined as an increase in PRA by >50% of the pretreatment value to a level of >4ng/mL per hour on the sixth day after paracentesis [(8)]

-Occurs in up to 80% of LVP procedures in which plasma expanders are not used [(1-3)] *(see below)*

-Associated with post-procedural hemodynamic instability, rapid reaccumulation of ascites, renal failure, hyponatremia, and increased mortality [(8)]

Mechanism of Action

The pathophysiology of PICD is not well elucidated. Multiple mechanisms have been proposed:

1. Reduced intra-abdominal pressure post-paracentesis results in decreased right atrial/pulmonary pressures and systemic vasodilation. This leads to "over-compensatory" RAAS activation and ANP synthesis. [(4)]
2. Rapid re-accumulation of ascites resulting in decreased total circulating volume. This has been theorized but not proven in the literature [(4)]
3. "Shear stress" induced by increased cardiac output after paracentesis induces nitric oxide synthesis, resulting in systemic vasodilation [(5)]

Predictors associated with PICD

1. *Volume of fluid drained:* No significant hormonal or hemodynamic changes observed when <5L are evacuated[(7)]. In the context of LVP, there is a direct correlation between the volume drained and the incidence of PICD. [(1,6,] 13)
2. *Non-selective B-blockers (NSBB)*: Active use of NSBB has been reported to increase PICD, as well as baseline hypotension, renal failure, infection, and mortality in patients with refractory ascites [(9-10).] However, more recent studies have challenged this link [(11)]
3. *Baseline demographics:* Younger patients are at higher risk of PICD (hypothesized that older patients have blunted RAAS response and therefore protected from PICD).

*At baseline, the following populations are at increased risk of hepatorenal syndrome (and it can thus be hypothesized that their risk of PICD is thus higher also):

a) Recent/active GI bleed
b) Active infection (in particular, SBP)
c) Metabolic abnormalities: Hyponatremia, baseline renal failure, acute hepatitis

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