Editorial

Intradialytic Hypotension: Is Reducing Extracorporeal Blood Flow Rate an Appropriate Intervention?

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Intradialytic hypotension (IDH) definitions vary widely, and none is universally accepted. Generally, IDH is defined as a condition in which there is a decrease in systolic blood pressure (SBP) > 20 mmHg or mean arterial pressure (MAP) > 10 mmHg, accompanied by symptoms of end-organ ischemia and requiring medical intervention.[1,2]

Despite numerous identifications of risk factors for IDH, the main causative factors are excessive or rapid ultrafiltration, rapid changes in plasma osmolality, excessively high post-hemodialysis (HD) target weight, reduced cardiac reserve, and autonomic neuropathy.[1] The pathophysiology that unites them is a reduction in effective blood volume, resulting from a rate of fluid loss that exceeds the plasma refill rate. This leads to decreased plasma tonicity. In the absence of an adequate compensatory response by adequate activation of the neurohumoral system, a fall in arteriolar tone and BP is expected. BP serves as a measure of arterial tone, reflecting the internal pressure exerted on the vessel wall by circulating volume, plasma tonicity (Hematocrit, total proteins, and sodium), vascular smooth muscle tone (sodium content of vascular smooth muscle and the balance between local vasoconstrictors and vasodilators), and sympathetic nerve activity.[3]

Extracorporeal Blood Flow Rate Changes and Cardio-Vascular Response

The body's physiological response to decreased extracellular fluid depletion is to increase venous return (DeJager-Krogh effect), autonomic responses (activating the sympathetic nervous system and subsequent non-osmotic release of renin and vasopressin), and cardiovascular responses. Cardiovascular responses include peripheral vascular resistance, contractility, and changes in heart rate.[1,3] The immediate response is an increase in HR. However, HR will decrease to baseline values if additional compensatory mechanisms are triggered. In cases of extreme refractory hypovolemia, HR may decrease as a result of the Bezold-Jarisch reflex.[1]

The most common interventions for IDH are fluid withdrawal, volume replacement, and extracorporeal blood flow rate **(**EBFR) reduction. Whether EBFR reduction is appropriate in IDH is debatable, as knowledge of the effects of EBFR reduction on BP and available data is very limited and conflicting. Reducing EBFR essentially reduces the efficacy of HD (Kt/V).[3]

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The following are research findings investigating the relationship between EBFR changes and cardiovascular responses.

- Alfurayh et al studied the effect of increasing EBFR on left ventricular (LV) function in 10 stable HD patients. EBFR was randomly selected as 250, 350, or 450 ml/min. The results showed that increasing dialysis EBFR to 450 cc/min had no detrimental effects on the left ventricle. Cardiac output (CO) or left ventricular ejection fraction, as well as pulse rate (PR) or BP, did not change significantly.[4]

- Trivedi et al investigated 34 HD patients, changing the BFR from 200 ml/min to 400 ml/min and vice versa, but with constant fluid UFR. Systolic BP, diastolic BP, and mean BP were significantly higher in patients during BFR 400 mL/min in comparison to BFR 200 mL/min. These findings suggest that BP is maintained higher at higher BFR during HD than at lower BFR.[5]

- Flythe et al examined 218 HD patients in a prospective observational study to assess the relationship between EBFR changes and SBP variability. The results indicated an association between greater fluid removal and greater SBP variability. However, no relationship was found between EBFR changes and SBP variability.[6]

- Post-hoc analysis from the HEMO Study found a greater risk of IDH among the higher Kt/V group in comparison to the standard Kt/V (target single-pool Kt/V of 1.65 vs. 1.25) with an OR of 1.12 (95%CI 1.01–1.25). Higher dialysis dose may be associated with an increased risk of IDH.[7]

- Philip et al. conducted a randomized, crossover study involving 22 hemodynamically stable HD patients, showing no consistent BP change trend due to EBFR reduction. No patients experienced intradialytic hypotension. The study does not support using EBFR reduction to increase systemic BP during HD.[8]

- Hafez et al investigated 40 hemodynamically stable HD patients to assess the effect of changes in EBFR 200, 300, and 400 ml/min on BP, PR, and COP. SBP was significantly higher at EBFR 200 ml/min in comparison to 300 ml/min, but there was no difference in SBP between EBFR 300 ml/min and EBFR 400 ml/min. At EBFR 200, 300, and 400 ml/min, DBP, MAP, PR, and COP did not show significant changes.[9]

According to the above data, it can be concluded that the treatment of IDH by reducing EBFR is not based on convincing evidence. This contrasts with findings from the HEMO study, which raises the possibility that the rate of intradialysis plasma osmolality decline may play an important role in mediating hemodynamic instability. This is because patients in the higher Kt/V group had significantly longer session durations, higher blood flows, and higher dialysate flows compared to the lower Kt/V group.[7] Further research is still needed to evaluate the impact of EBFR changes on BP during IDH.

Declarations

Competing interest

The author declares no conflict of interest.

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