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Five Things to Know About Intradialytic Hypertension

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1. Intradialytic hypertension is a paradoxical rise in blood pressure (BP) that occurs specifically during the hemodialysis (HD) procedure.

There are no accepted criteria that define intradialytic hypertension, but several definitions exist:

- An increase in systolic blood pressure (SBP) >10 mm Hg during HD (most common definition).[1]
- Rise in the mean arterial pressure >15 mmHg during or immediately post-HD.[2]
- Any BP rise during HD.[3]

2. Persistent intradialytic hypertension is common and is associated with poor prognostic factors and increased mortality.

Persistent intradialytic hypertension (>10 mm Hg rise in SBP) is reported to occur in 15% to 30% of patients treated with HD,[2,4] and is associated with twofold to fourfold increased risks of hospitalization, all-cause mortality, and cardiovascular mortality.[5,6] Whether these relationships are causal is not certain, as intradialytic hypertension tends to occur in patients with markers of more severe illness, including older age, lower serum albumin, lower kT/V, lower body mass index, and greater use of antihypertensive medications.[7] Persistent intradialytic hypertension is likely a marker of cardiovascular risk and vascular stiffness.[3]

3. Patients with intradialytic hypertension often have high ambulatory BP between dialysis sessions, but this is not universal.

Intradialytic BPs are highly correlated with ambulatory BP between HD sessions, suggesting that patients with intradialytic hypertension are also likely to have uncontrolled ambulatory hypertension.[8]

4. The causes of intradialytic hypertension are complex and not yet fully elucidated.

Intradialytic hypertension has been observed with equal frequency in patients with normal, low- and high-volume status by bioimpedance,[4] suggesting volume overload is not the only factor. Factors that increase total peripheral resistance appear to be important, especially in patients with underlying vascular stiffness (Figure 1). Contributing factors thus include volume overload,[1] sympathetic overactivity,[9] renin angiotensin aldosterone system,[10] endothelial dysfunction and relative increase in endothelin 1 to nitric oxide,[11] administration of erythropoiesis stimulating agents (ESA) during dialysis,[12] and sodium loading during dialysis.[13]

5. Several strategies have been proposed but strong evidence is lacking:
   A. *Optimize the treatment of essential hypertension.* This includes carefully reassessing the target weight,[15] the use of appropriate antihypertensives such as beta blockers and angiotensin receptor blockers (see below) and ensuring medication adherence. Extended hours[16] or frequent HD[17] may also be considered when circumstances permit.
   B. *Consider using a beta blocker with additional alpha blockade activity, such as carvedilol*. In a prospective 12-week pilot study of carvedilol titrated to 50 mg twice daily in 25 patients with intradialytic hypertension, carvedilol was associated with modest improvements in endothelial function, improved BP between dialysis sessions, and reduced frequency of intradialytic hypertension.[18]

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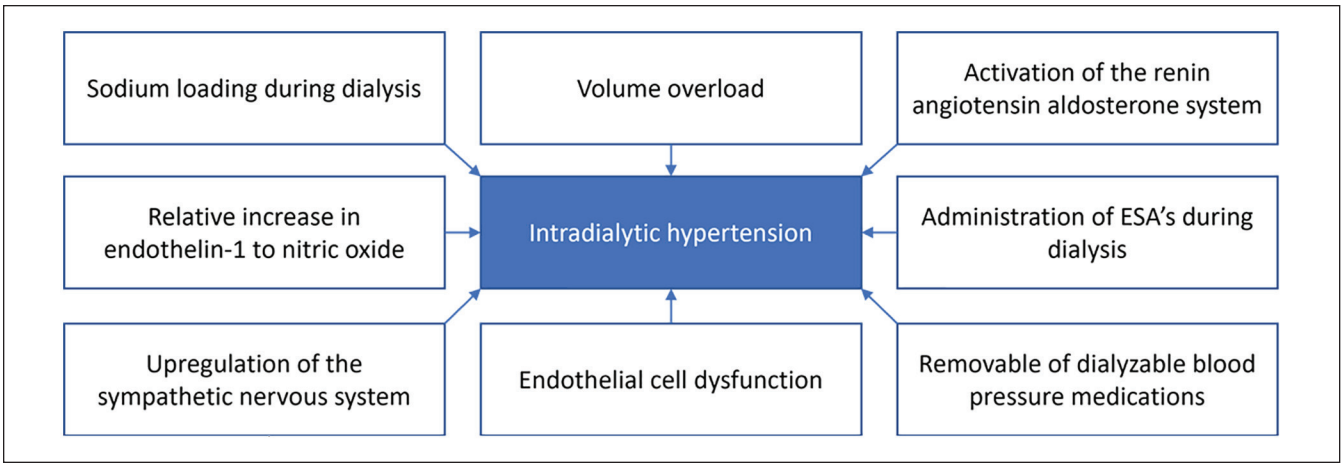

Figure 1. Risk factors and proposed mechanisms for intradialytic hypertension.[14]
Note. BP = blood pressure; ESA = erythropoiesis stimulating agents.

C. *Consider giving a short acting antihypertensive agent prior to the hemodialysis session.* Certain antihypertensives such as captopril and hydralazine have rapid onset and offset and may be used to prevent intradialytic hypertension while not causing hypotension in between dialysis sessions.[19]
D. *Choose antihypertensives that are less dialyzable.* Angiotensin converting enzymes, except for fosinopril, are highly dialyzable, whereas all angiotensin receptor blockers are non-dialyzable. Commonly used beta blockers such as metoprolol and atenolol are highly dialyzable[20] while bisoprolol is moderately dialyzable and carvedilol is the least dialyzable.[20] Nebivolol, another non-dialyzable beta-blocker, and irbesartan were effective in reducing intradialytic hypertension in one randomized trial.[21]
E. *Consider lowering dialysate sodium.* In a small pre-post study (n = 50), reducing the dialysate sodium from 140 mmol/L to 136 mmol/L led to significantly lower third hour and post-HD SBP.[22]
F. *Avoid high calcium dialysate.* As there is an association between lower calcium dialysate and lower post-HD systolic BP,[23] a low-calcium dialysate is sometimes recommended to treat intradialytic hypertension. Strong evidence is lacking, however, and this approach may result in a negative calcium balance and increased risk of cardiac arrythmias.[24]
G. *Consider raising the dialysate temperature.* Lowering the dialysate temperature below core temperature increase intradialytic BP,[25] while lower BPs are observed with higher dialysate temperatures.[26]
H. *Consider switching administration of ESA to the subcutaneous route.* Intravenous ESA is associated with rises in mean arterial pressures 30 minutes after administration, and this effect can last up to 3 hours. Subcutaneous administration does not appear to have this effect.[27]

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