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Fenoldopam as a Possible Medication for Non-Occlusive Mesenteric Ischemia (NOMI) and Contrast-Induced Nephropathy?

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To the editor,

Non-occlusive mesenteric ischemia (NOMI) is a type of mesenteric ischemia in which patency of duct is maintained; but there is decreased perfusion. It is present in about 20%-30% of all acute mesenteric ischemia¹. The basis behind NOMI's pathophysiology shows that by preserving blood supply to viscera, perfusion in mesenteric vessels is compromised; thus, in theory, a drug that can vasodilate these vessels can play a crucial role in providing sufficient perfusion to distressed vessels². Prominent animal studies have shown that Fenoldopam acts as a vasodilator and increases blood flow in mesenteric arteries³⁻⁴.

Fenoldopam is a dopamine (DA1) receptor agonist, primarily used as an antihypertensive drug that acts on smooth vessels, causes vasodilation, and plays a pivotal role in increasing splanchnic and renal perfusion³. Fenoldopam alone did not show any significant isolated vasodilation, especially in the elderly (>60 years), but while working with prazosin and other vasodilators, it showed significant mesenteric vasodilatory effect⁵.

Contrast-induced computed tomography (CT) scan is a gold standard to diagnose NOMI and other abdominal vessels related problems⁶. Contrast CT may lead to contrast-induced nephropathy, which generally occurs in preexisting renal function anomalies⁷. Fenoldopam can be given to patients who are at risk of contrast-induced nephropathy, like those susceptible to hypersensitivity reactions due to contrast medium used or kidneys with low glomerular filtration rate (due to decrease renal perfusion)⁷. As Fenoldopam increases renal

perfusion, it helps prophylactically in contrast-induced nephropathy⁷. Therefore, when a patient is planned to undergo contrast CT for diagnosis of NOMI with risk of contrast-induced nephropathy, Fenoldopam can be given to act against NOMI and contrast-induced nephropathy, where a decrease in renal perfusion might be seen after administration of the contrast dye.

Considering the clinical urgency of NOMI and promising animal trials of Fenoldopam, it is astonishing that no clinical trials to test the therapeutic effect of Fenoldopam in NOMI and to prevent contrast-induced nephropathy, simultaneously. However, a clinical trial of Stone et al⁸ showed that Fenoldopam did not help prevent contrast-induced nephropathy in patients with chronic renal insufficiency. Patients in this trial were already having renal insufficiency. So more human trials with patient population having danger of contrast-induced nephropathy but they still have a functioning kindney assorted in different age group and different regions should take place to test the effect of Fenoldopam in diagnosing NOMI, simultaneously preventing contrast-induced nephropathy.

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