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Abstract: Background: The authors discuss the patho-physiological aspects of the rare 'aortoiliac-mesenteric steal syndrome', developing through a complex pelvic network of collaterals between the inferior mesenteric artery and the iliac-femoral trunks.

Results: The progressive insufficiency of this network complicated with a proper mesenteric steal syndrome. The particular histological textures of the mesenteric and of the lower limb vascular systems explain the differences between their 'remodeling', correlated to chronic ischemia, and promoted by the action of the same endogenous factors.

Conclusion: A complete angiographic study of the aorto-iliac-femoral trunks allowed to prefer the surgical technique of aortic re-implanting of the inferior mesenteric artery.

Suggested Reviewers: Teresa Pusiol
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Dear **Sir**,

We submit you our paper as correspondence / letter to the Editor, referring to a recently published article [Shiraev T, Neilson W. "Aortoiliac-mesenteric steal syndrome" treated with inferior mesenteric to common iliac artery transposition. *Ann Vasc Surg* 2019;58:377.e9-377.e11].

This paper has not been previously published or submitted elsewhere for publication and will not be sent to another journal until a decision is made concerning publication by *Annals of Vascular Surgery*.

The authors declare no conflict of interest, no sources of outside support to this research.

The text words number is # 468 and the references are limited to # 9.

You are completely free to change or correct the title or the text.

With many thanks for your collaborations and the best regards.

On the behalf of all the authors,

Sincerely yours,

A. Manenti, MD

A Pathogenetic Focus on the Aortoiliac - Mesenteric Steal Syndrome

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Key words: inferior mesenteric artery; mesenteric vascular insufficiency; arterial steal syndrome; aorto-iliac insufficiency; arterial re-implanting.

Dear Sir,

The interesting paper by Shiraev et al. concerning a case of ‘aortoiliac-mesenteric steal syndrome’ invites to deepen some of its patho-physiological issues.¹ The inferior mesenteric artery (IMA), when partially obstructed, can be largely collateralized by retrograde pelvic branches originating from the iliac-femoral trunks, overcoming the hemodynamic differences between these two vascular systems.² In fact, the splanchnic vessels are supplied by a systemic blood pressure which increases during the post-prandial phases; their medium-size branches are widely interconnected, while their

peripheral networks cannot easily expand, even in case of chronic ischemia. This agrees with their native architecture developed inside the digestive organs and with their venous return connected with the portal system.³ Moreover, this explains how a chronic mesenteric insufficiency is at first compensated by a process of ‘hypertrophy’ of the medium-size arterial digestive trunks and of their interconnections. On the contrary, the blood flow to the lower limbs is effort dependent, greatly varies according to muscle activity, is supported by second order aortic branches, and it has a lesser basic pressure of inflow. However, its extension can be largely increased by a neo-angiogenesis in its peripheral network, mainly developed inside the striated muscles.⁴ The differences between these two arterial systems explain the easy decompensation of their mutual collateralization, in course of their progressive insufficiency, leading to a proper ‘aortoiliac-mesenteric steal syndrome’.⁵ In the reported case, this particular adjustment most probably was caused by an accelerated IMA obstruction, in face of a preserved function of the pelvic collaterals and of a large network of new-generated collaterals inside the lower limbs.¹ From a pathogenetic point of view, we observe that these two distinct processes of arterial ‘hypertrophy’, typical of the mesenteric trunks, and of neo-angiogenesis, characteristic of the peripheral territories of the lower limbs, represent different aspects of the same basic mechanism of arterial ‘remodeling’, which compensates a chronic ischemia in two diverse histological contexts. However, we consider always determinant the role of the same humoral agents, released by hypoxic tissues, and acting in a paracrine fashion on their vasculature, in particular the vascular endothelial growth factor and the fibroblast growth factor, which stimulate the replication of vascular cells, typically the endothelial, and the recruitment of circulating blood elements, such as monocytes, medullary stem cells, or other endothelial progenitors.⁵⁻⁷ Clearly, their action on the mesenteric trunks is enhanced by the blood stress forces.^{4,6,7} The adopted surgical solution seems particularly appropriate, respecting the still patent network of pelvic collaterals, which could come at risk of thrombosis after a direct aorto-femoral revascularization, through the mechanism of ‘competition’, as observed in coronary artery by-pass surgery.⁸ Besides, we underline that, in case of mesenteric vascular insufficiency, a

complete angiography study of all the aortic collaterals and branches can indicate most appropriate surgical procedures, like vascular re-implanting, already experienced for the superior mesenteric artery.⁹

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