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Letter to the Editor

The role of lysyl oxidase-like 1 and fibulin-5 in the development of atherosclerosis and pelvic organ prolapse

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Dear Editor:

I would like to congratulate Zhou et al.^[1] on their study of the correlation between expression of lysyl oxidase-like 1 (LOX-1) and fibulin-5 (F5) in the cardinal ligament tissue and pelvic organ prolapse (POP). In their elegant work, they evaluated the levels of LOX-1 and F5 in connective tissue of the cardinal ligament in order to demonstrate signs of elastinopathy in women with POP.

They stress the concept that several environmental risk factors could cause qualitative and quantitative changes in the connective tissue promoting POP. The above authors conclude that the specific mechanism of LOXL1 and F5 involved in the development of POP is unclear.

Starting from the evidence (confirmed by the epidemiology) of a strong correlation between POP and some age-correlated diseases, I believe that the physiopathological mechanism of POP might be atherosclerosis.

The present study could represent the first demonstration of this mechanism: in many common cardiovascular diseases (such as atherosclerosis, hypertension, and heart failure), F5 has been demonstrated to be important as the predominant binding protein of extracellular superoxide dismutase, whose mechanism of action is known to play a significant role in the development of endothelial dysfunction and atherosclerosis^[2]. LOX-1 is known to be involved not only in the cross-linking of collagen and elastin in the extracellular space, but also in some vascular diseases^[3].

The presence of increased levels of these markers of atherosclerosis not only strengthens the hypothesis of a correlation between POP and connective changes induced by atherosclerosis, but may also contribute to identifying some markers predicting the development of pelvic floor disorders.

For this reason, and in order to confirm this pathway from atherosclerosis to POP, this study could provide an interesting starting point for further studies.

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References

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