The potential association between the risk of post-surgical adhesion and the activated local angiotensin II type1 receptors, Need for Novel Treatment Strategies

Abstract

Background: Post-surgical adhesion bands (PSAB) are a common complication after abdominal or pelvic surgeries in different reasons like cancers treatment. Despite improvements in surgical techniques and the administration of drugs or the use of physical barriers, there has only been limited improvement in the frequency of postoperative adhesions. Complications of PSAB are included pain, infertility, intestinal obstruction, and increased mortality. The most important molecular mechanisms for the development of PSAB are inflammatory response, oxidative stress, and overexpression of pro-fibrotic molecules such as transforming growth factor β (TGF-β). However, questions remain about the pathogenesis of this problem, for example, the causes for individual differences, or why certain tissue sites are more prone to post-surgical adhesions. **Summary:** Addressing the pathological causes of PSAB, the potential role of local angiotensin II/ angiotensin II type1 receptors (AngII/AT1R), may help to prevent this problem. **Key word:** The objective of this paper was to explore the role of the angiotensin II/AT1R axis potential to induce PSAB and the therapeutic potential of angiotensin receptor blockers (ARBs) in the prevention and treatment of PSAB.

Keywords: Post-surgical adhesion band**,** Angiotensin II, Angiotensin type 1 receptor, Inflammation, Oxidative stress, Fibrotic molecules.

Abbreviation:
Angiotensin II (AngII); angiotensin II type 1 receptor (AT1R); angiotensin receptor blockers (ARBs), extra cellular matrix (ECM); hepatic stellate cells (HSCs); interleukin 6 (IL6); intercellular adhesion molecule-1 (ICAM-1); mitogen-activation protein kinase (MAPK); monocyte chemo-attractant protein 1 (MCP-1); matrix metalloproteinase (MMPs); Post-surgical adhesion band (PSAB); plasminogen activator inhibitor-1 (PAI-1); Platelet-derived growth factor (PDGF); alpha smooth muscle actin (α-SMA); tissue plasminogen activation (tPA); renin-angiotensin system (RAS); nuclear factor *kappa* B (NF-*K*B); transforming growth factor β (TGF-β); tissue inhibitor of metalloproteinase (TIMP); Tumor necrosis factor α (TNF-α).

Running title: AT1Rs activation and the Risk of PSAB