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INCREASED GALECTIN-3 LEVELS ARE ASSOCIATED WITH ABDOMINAL AORTIC ANEURYSM PROGRESSION AND INHIBITION OF GALECTIN-3 DECREASE ELASTASE-INDUCED AAA DEVELOPMENT

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The evolution of abdominal aortic aneurysm (AAA) is unpredictable. Moreover, no specific treatment exists for AAA, except surgery to prevent aortic rupture. Galectin-3 has been previously associated with cardiovascular disease (CVD), but its potential role in AAA has not been addressed. Galectin-3 levels were increased in plasma of AAA patients (n=225) compared to controls (n=100). Moreover, galectin-3 concentrations were associated with need for surgical repair, independently of potential confounding factors. Galectin-3 mRNA and protein expression were increased in human AAA samples compared to healthy aortas. Experimental AAA in mice was induced by aortic elastase perfusion. Mice were treated intravenously with the galectin-3 inhibitor modified citrus pectin (MCP, 10 mg/kg, every other day) or saline. Similar to humans, galectin-3 serum and aortic mRNA levels were also increased in elastase-induced AAA mice compared to control mice. Mice treated with MCP showed decreased aortic dilation, as well as elastin degradation, vascular smooth muscle cell (VSMC) loss and macrophage content at day 14 post-elastase perfusion compared with control mice. The underlying mechanism(s) of the protective effect of MCP was associated with a decrease in galectin-3 and cytokine (mainly CCL5) mRNA and protein expression. Interestingly, galectin-3 induced CCL5 expression by a mechanism involving STAT3 activation in VSMC. Accordingly, MCP treatment decreased STAT3 phosphorylation in elastase-induced AAA. In conclusion, increased galectin-3 levels are associated with AAA progression, while galectin-3 inhibition decreased experimental AAA development. Our data suggest the potential role of galectin-3 as a therapeutic target in AAA

Recent Publications

1. Dale M A, Ruhlman M K and Baxter B T (2015) Inflammatory cell phenotypes in AAAs: their role and potential as targets for therapy. Arterioscler Thromb Vasc Biol 35:1746-55.
2. Daniels L B, Clopton P, Laughlin G A, Maisel A S and Barrett Connor E (2014) Galectin-3 is independently associated with cardiovascular mortality in community-dwelling older adults without known cardiovascular disease: the Rancho Bernardo study. Am Heart J 167:674-82.
3. Filipe M D, Meijers W C, Rogier van der Velde A and de Boer R A (2015) Galectin-3 and heart failure: prognosis, prediction & clinical utility. Clin Chim Acta 443:48-56.
4. Jagodzinski A, Havulinna AS, Appelbaum S, Zeller T, Jousilahti P, Skytte Johanssen S, Hughes M F, Blankenberg S and Salomaa V (2015) Predictive value of galectin-3 for incident cardiovascular disease and heart failure in the population-based FINRISK 1997 cohort. Int J Cardiol 192:33-9.
5. Maiolino G, Rossitto G, Pedon L, Cesari M, Frigo A C, Azzolini M, Plebani M, and Rossi G P (2015) Galectin-3 predicts long-term cardiovascular death in high-risk patients with coronary artery disease. Arterioscler Thromb Vasc Biol 35:725-3.

Biography

Mónica M Torres Fonseca is a Vascular Surgeon who, along with her clinical work, devotes a large part of her time to the field of research, which she is passionate about. She and her group's work are mainly directed to the study of AAA for years. This is the object of study in her doctoral thesis, based on the diagnosis, prognosis and treatment of AAA through clinical and experimental studies. She is concerned about the situation of patients with AAA without surgical indication, who cannot be offered effective medical treatment at this time. Therefore, part of their study aims to determine possible particles that can reduce or delay the progression of the aneurysm, which would mean a substantial change in the prognosis of these patients.

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