Tissue Remodeling in Vascular Wall in Kawasaki Disease-Related Vasculitis Model Mice

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Kawasaki disease is the most common acute systemic vasculitis of unknown etiology in children [1] and can cause inflammation of the coronary arteries leading to aneurysms. Tenascin-C, an extracellular matrix protein, and c-Jun N-terminal kinase (JNK), an intracellular signaling protein, are known to be associated with inflammation and tissue remodeling [2, 3]. The purpose of this study was to demonstrate tenascin-C and JNK might be involved in tissue remodeling in a *Candida albicans*-induced murine model of aneurysm.

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1. More than 80 % of the mice showed the macroscopic features of aneurysms in the aorta and/or iliac and coronary arteries.
2. Marked inflammatory cell infiltration was observed in vascular wall and perivascular connective tissue, accompanied by fragmentation of elastic fibers.
3. Expression of tenascin-C was highly observed in vascular wall, accompanied by active degradation of elastic fibers.
4. Pharmacologic inhibition of JNK attenuated the aneurysm formation in the mice model.

In conclusion, these findings suggest that both tenascin-C and JNK are involved in abnormal tissue remodeling and inflammation in the *Candida albicans*-induced Kawasaki disease murine model of aneurysm and that JNK inhibition may represent a novel therapeutic target for preventing a Kawasaki disease-related aneurysm.

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