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Possible involvement of IL-23/IL-17 axis in pathogenesis of Kawasaki disease like vasculitis

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Introduction: Although mice of vasculitis induced with Candida albicans water-soluble fraction (CAWS) is widely used as an established model of Kawasaki disease (KD), the pathogenic mechanism of vasculitis has yet to be determined. Some studies have recently reported that IL-23/IL-17 axis is closely related to inflammation and autoimmune response. β-glucan and mannnan of a major component of CAWS enhance the production of IL-23 by dendritic cells through dectins which are receptors for β-glucan and mannnan. Therefore, to test the hypothesis that IL-17-producing cells (IL-23 receptor+) activated by IL-23 from dendritic cells initiate KD vasculitis, we investigated the distribution of IL-23 receptor and IL-17 in predilection sites of CAWS-induced vasculitis.
Methods: CAWS was intraperitoneally injected to mice for five consecutive days. At 1, 7, 14 days after CAWS injection, mice were sacrificed. We studied the expression of IL-23 receptor and IL-17 in the aortic root including coronary bifurcation, which is one of the most vulnerable sites in the experimental vasculitis, with immunohistochemistry.
Results: IL-23 receptor+ and IL-17+ cells presented in the aortic valves, the proximal region of it and adventitia of the aortic root in both CAWS-administrated mice and control. Furthermore, these cells apparently increased by administration of CAWS.
Conclusions: These findings suggest that IL-23/IL-17 axis activated by β-glucan and mannan is involved in the pathogenesis of CAWS-induced vasculitis, and can also explain a part of the mechanism to define the site specificity of vasculitis in this model.