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ANTI-APOA-1 AUTO-ANTIBODIES ARE ACTIVE MEDIATORS OF ATHEROSCLEROTIC PLAQUE VULNERABILITY

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Introduction: Anti-ApolipoproteinA-1 auto-antibodies (anti-ApoA-1 IgG) represent an emerging prognostic cardiovascular marker in patients with myocardial infarction or autoimmune diseases associated with high cardiovascular risk. The potential relationship between anti-ApoA-1 IgG and plaque vulnerability remains elusive. Thus, we aimed to investigate the role of anti-ApoA-1 IgG i! n plaque vulnerability.
Méthode: Potential relationship between anti-ApoA-1 IgG and features of cardiovascular vulnerability was explored both in vivo and in vitro. In vivo, we investigated anti-ApoA-1 IgG in patients with severe carotid stenosis (n=102) and in ApoE-/- mice infused with polyclonal anti-ApoA-1 IgG. In vitro, anti-ApoA-1 IgG effects were assessed on human primary macrophages, monocytes and neutrophils.
Résultats: Intraplaque collagen was decreased, while neutrophil and MMP-9 content was increased in anti-ApoA-1 IgG positive patients and anti-ApoA-1 IgG-treated mice as compared to corresponding controls. In mouse aortic roots (but not in abdominal aortas), treatment with anti-ApoA-1 IgG was associated with increased lesion size as compared to controls. In humans, serum anti-ApoA-1 IgG levels positively correlated with intraplaque macrophage, neutrophil and MMP-9 content, and inversely with collagen. In vitro, anti-ApoA-1 IgG increased macrophage release of CCL2, CXCL8 and M MP-9, as well as neutrophil migration towards TNF-alpha or CXCL8.
Conclusion: These results suggest that anti-ApoA-1 IgG might be associated with increased atherosclerotic plaque vulnerability in humans and mice.