Junctional Adhesion Molecule-1 May Have a Wider Role in Cardiovascular Disease

To the Editor:

Junctional adhesion molecule-1 (JAM-1; or F11 receptor) was first discovered as a surface protein on human platelets. It was later recognized as a receptor in the tight junction between cells involved in leukocyte migration in inflammation and platelet adhesion in thrombosis. The recent study by Waki et al suggested that JAM-1 may play a novel role in the etiology of hypertension through the autonomic nervous system. They demonstrated that expression of JAM-1 was generally increased in spontaneously hypertensive rats and that adenoviral-mediated transfection of JAM-1, increasing its expression in the nucleus tractus solitarii in the brain, increased blood pressure in the rat. This ties in with the concept of the activation of the sympathetic nervous system as one of the mechanisms by which obesity induces hypertension. However, expression of JAM-1 in spontaneously hypertensive rats increased not only in the brain but also in other organs, such as heart, kidney, and skeletal muscle. JAM-1 is also expressed in atherosclerotic plaques, suggesting a wider role in cardiovascular disease. The gene encoding human JAM-1 is located at chromosome 1q21, a susceptibility locus for type 2 diabetes and the metabolic syndrome. Therefore, it would be of interest to study the role of JAM-1 in insulin resistance, dyslipidemia, endothelial dysfunction, and atherothrombosis. Interestingly, the increased expression of JAM-1 precedes the onset of hypertension, which is consistent with the contemporary view that inflammation is involved in the pathogenesis of hypertension. Although JAM-1 may be a significant determinant of hypertension in spontaneously hypertensive rats, its role in human hypertension awaits confirmation. Furthermore, its influence on the cardiovascular system beyond the autonomic nervous system merits investigation.

Disclosures

None.

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