P-selectin gene -825 polymorphism is associated with risk of Henoch-Schönlein purpura nephritis

Sirs,

In recent years the morbidity of Henoch-Schönlein purpura (HSP) has risen, but its exact pathogenesis is unknown. One manifestation of HSP that can continue to cause lifelong problems is renal involvement. Some Henoch-Schönlein purpura nephritis (HSPN) patients have poor prognosis. Davin et al. (1) found that 20% of the patients developed chronic renal failure. Although the exact mechanisms governing the fate of renal function in HSPN are also unclear, genetic factors are presumed to participate in its development and progression. In particular, the correlations between the development of HSPN and single-nucleotide polymorphisms (SNPs) in genes that encode nephritis-related molecules have become the focus of attention (2, 3).

P-selectin is a cellular adhesion molecule belonging to the selectin family. After its activation, P-selectin molecules are released from the a-granules of platelets and participate in the rolling and tethering of platelets at the surface of endothelial cells. P-selectin is also redistributed at the cell membrane of activated endothelial cells from Weibel-Palade bodies and therefore mediates the adhesion and rolling of leukocytes on the vascular wall. P-selectin therefore plays an important role in the recruitment of leukocytes on the vascular surface at inflammatory foci, in their extravasation, and in the adhesion of platelets to the endothelium (4). Because of its biological functions, this molecule is postulated to be involved in the development of vasculitis and its complications. Supporting this concept are the observations that P-selectin participation in the rolling and tethering of neutrophil-mediated glomerular injury. A role for P-selectin in complement-independent neutrophil-mediated injury has been shown. The P-selectin promoter was amplified using specific primers

<table>
<thead>
<tr>
<th>Group</th>
<th>n.</th>
<th>Genotype, n. (%)</th>
<th>Allele, n. (%)</th>
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<tbody>
<tr>
<td>Control</td>
<td>70</td>
<td>47 (67.1)</td>
<td>117 (83.6)</td>
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<tr>
<td>HSP without nephritis</td>
<td>46</td>
<td>33 (72)</td>
<td>79 (86)</td>
</tr>
<tr>
<td>HSPN</td>
<td>40</td>
<td>37 (92.5)</td>
<td>77 (96.3)</td>
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*p-value<0.05 vs. the control and the HSP without nephritis groups.

In summary, this study showed that the P-selectin -825 AA genotype and A allele frequencies increased in children with HSPN compared to HSP children without nephritis and healthy controls. Also, the AA genotype of the P-selectin -825A/G polymorphism was associated with an increased risk for the development of nephritis in HSPN patients. This suggests that P-selectin -825A/G gene polymorphism may be associated with the development of HSPN and that A allele may be a susceptible gene of HSPN.

Competing interests: none declared.

References