TO THE EDITOR: I read with interest the study by Park et al\(^1\) regarding inhibitory effect of ramosetron on accelerated upper gastrointestinal (GI) transit of male guinea pigs. In the study, GI transit was assessed as the migration of charcoal mixture from the pylorus to the most distal point of migration and expressed as a percentage of charcoal migration through the total length of small intestine. Oral administration of ramosetron significantly delayed GI transit compared with control group. Ramosetron also significantly inhibited 5-hydroxytryptamine, thyrotropin-releasing hormone and mustard oil-induced accelerated GI transit. The authors should be congratulated for completing study, which supports theoretical evidence that ramosetron may be therapeutically useful for the diseases associated with accelerated upper GI transit. I recently reported in other journal that a 49-year-old male with diabetic diarrhea associated with rapid small bowel transit was successfully treated with ramosetron.\(^2\) He had poorly controlled diabetes, orthostatic hypotension, diabetic retinopathy, nephropathy, peripheral neuropathy, autonomic neuropathy and gastroparesis. His diarrhea occurred at a frequency of > 10 bowel movements per day, with fecal urgency following meals. Steatorrhea was absent, and the stool examination was unremarkable. He was prescribed on conventional antidiarrheal agents, including a pancreatic enzyme supplement, rifaximin, probiotics, cimetropium bromide and loperamide, but these were ineffective. Capsule endoscopy was performed to reveal the cause of iron deficiency anemia, and incidentally showed severely prolonged gastric emptying time (13 hours 33 minutes) with rapid small bowel transit time (14 minutes). He was thus diagnosed with diabetic diarrhea associated with rapid small bowel transit and treated with ramosetron 5 μg once daily. Diarrhea completely disappeared after 1 week and recurred right away after cessation of ramosetron. This therapeutic utility of ramosetron for diabetic diarrhea has been documented.\(^3\) Table shows the summary of clinical features of 2 cases with refractory diabetic diarrhea successfully treated with ramosetron. These findings are consistent with the study by Park et al,\(^1\) in which ramosetron inhibited accelerated upper GI transit in male guinea pigs. A recent animal study showed that ramosetron improved soybean oil- and corticotropin releasing factor-induced delay in gastric emptying of rats.\(^4\) Therefore I strongly believe that these results provide therapeutic implication of ramosetron for diabetic diarrhea with accelerated small bowel transit as well as diabetic gastroparesis. Further human study should be needed to confirm the results.

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Table. Summary of Clinical Features of 2 Cases With Refractory Diabetic Diarrhea Successfully Treated With Ramosetron

<table>
<thead>
<tr>
<th>Case</th>
<th>Sex</th>
<th>Age</th>
<th>DM type</th>
<th>HbA1C</th>
<th>Associated diseases</th>
<th>Daily BM</th>
<th>Previous treatments</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Male</td>
<td>49</td>
<td>Type II</td>
<td>17.7%</td>
<td>Orthostatic hypotension, diabetic retinopathy, nephropathy, peripheral neuropathy</td>
<td>&gt; 10</td>
<td>Pancreatic enzyme supplement, rifaximin, probiotics, cimetropium bromide and loperamide</td>
</tr>
<tr>
<td>2</td>
<td>Male</td>
<td>37</td>
<td>Type II</td>
<td>10%</td>
<td>Autonomic neuropathy</td>
<td>&gt; 15</td>
<td>Pancreatic enzyme supplements and loperamide</td>
</tr>
</tbody>
</table>


Conflicts of interest: None.