hypertension, and CT scan demonstrated no atherosclerosis throughout his aorta. There was no familial history of aortic pathology or connective tissue disease, which is why the trauma is the most suitable cause. Absence of a flap in the descending thoracic aorta caused misdiagnosis of an aortic aneurysm combined with abdominal aortic dissection on initial presentation.

CONCLUSIONS
We report aortic intimointimal intussusception in traumatic chronic type B aortic dissection with associated symptoms of visceral organ ischemia. This uncommon presentation of aortic dissection should be considered if the circumferential flap exists distal to the aneurysmal portion of the aorta.

References

EDITORIAL COMMENTARY
An intimal cylinder in the descending aorta
Yutaka Okita, MD

Intimointimal intussusception is a very rare complication of aortic dissection. Few cases have been reported in the English-language cardiology, cardiothoracic surgery, and radiology literature—only 30 or so since 1980. Most reported cases have occurred in men, in a relatively younger group, aged 31 to 66 (mean: 51.8) years.

This uncommon variation usually occurs in acute type A dissections: the ascending aortic intima flaps around its circumference, detaches from the media, and forms a tube-like structure that may prolapse, either antegrade into the ascending aortic lumen, or retrograde into the left ventricular outflow tract and cavity. Usually, the site of the intimal tear is near the sinotubular junction of the ascending aorta. Although rarely reported, some such cases are complicated by chronic aortic dissection or Stanford type B dissection.

Antegrade intussusceptions can partially or completely occlude the ostia of the branches of the distal or proximal aorta, and might cause dynamic obstruction malperfusion syndrome, such as coronary insufficiency, stroke, transient ischemic attack, paraplegia, or visceral or limb ischemia. Retrograde intussusceptions may severely impair left ventricular filling in diastole, and can worsen aortic regurgitation, as well as produce occlusion of the coronary ostia and acute coronary ischemia.

This complication of aortic dissection seldom causes “static obstruction” of the aortic branches. Most cases have undergone emergent or elective surgery, and clinical results were satisfactory, which, of course, may have depended on patients’ preoperative condition. We experienced the case of a 63-year-old man with acute type A aortic dissection with intimo-intimal intussusception, which caused severe aortic valve regurgitation and coronary ischemia. He was in a state of shock, and emergency hemiarch replacement was performed without any sequelae.

Dr Kim, Dr Ahn, and their associates at Seoul National University1 reported a very rare case of a man aged 38 years who had chronic type B aortic dissection with intimointimal
intussusception in the descending aorta. He had complained of vague abdominal pain, which was caused by dynamic obstruction of the visceral arteries, as evidenced by abatement of these symptoms after surgery.

The possibility of visceral malperfusion was diagnosed preoperatively, using computed tomography images. However, these images failed to definitively demonstrate the objective findings of visceral malperfusion, which would have been shown by abdominal echograms or dynamic magnetic resonance images. This case report brings the added insight that the very rare complication of intimo-intimal intussusception in type B aortic dissection may cause visceral malperfusion syndrome.

Reference

Intraoperative vacuum-assisted closure following in situ graft replacement for an infected thoracic aortic graft

Katsuhiro Hosoyama, MD, Shunsuke Kawamoto, MD, PhD, Naotaka Motoyoshi, MD, PhD, and Yoshikatsu Saiki, MD, PhD, Sendai, Japan

Prosthetic graft infection after surgery to reconstruct the thoracic aorta is a devastating complication, and the management remains a challenge for surgeons. In particular, patients at high risk with anastomotic lesions such as false aneurysms, septic emboli, or ruptured suture lines, which require emergency in situ replacement, are associated

TABLE 1. Patient summary

<table>
<thead>
<tr>
<th>Case</th>
<th>Age (y)</th>
<th>Sex</th>
<th>Risk factor</th>
<th>Primary surgery</th>
<th>Clinical presentation before second surgery</th>
<th>Duration from previous surgery</th>
<th>Pathogen</th>
<th>Operative procedure</th>
<th>Bleeding in postop 24 h (mL)</th>
<th>Period of VAC therapy (d)</th>
<th>Follow-up duration (y)</th>
<th>Recurrent infection</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>54</td>
<td>M</td>
<td>Diabetes mellitus</td>
<td>ATA replacement</td>
<td>False aneurysm</td>
<td>2 mo</td>
<td>Streptococcus species</td>
<td>TAR with allograft and prosthetic graft</td>
<td>650</td>
<td>9</td>
<td>5.7</td>
<td>None</td>
</tr>
<tr>
<td>2</td>
<td>73</td>
<td>M</td>
<td>None</td>
<td>CCA replacement</td>
<td>False aneurysm adjacent to aortic arch</td>
<td>5 d</td>
<td>Unspecified</td>
<td>TAR with prosthetic graft</td>
<td>660</td>
<td>9</td>
<td>2.2</td>
<td>None</td>
</tr>
<tr>
<td>3</td>
<td>75</td>
<td>F</td>
<td>None</td>
<td>ATA replacement</td>
<td>Bacterial vegetation</td>
<td>15 mo</td>
<td>Candida albicans</td>
<td>ARR with xenograft valve and prosthetic graft</td>
<td>640</td>
<td>9</td>
<td>1.9</td>
<td>None</td>
</tr>
<tr>
<td>4</td>
<td>67</td>
<td>M</td>
<td>Steroid use</td>
<td>ATA replacement</td>
<td>False aneurysm</td>
<td>16 mo</td>
<td>Propionibacterium acnes</td>
<td>AAR with prosthetic graft</td>
<td>740</td>
<td>9</td>
<td>1</td>
<td>None</td>
</tr>
</tbody>
</table>

Postop, Postoperative; VAC, vacuum-assisted closure; ATA, ascending thoracic aorta; TAR, total arch replacement; CCA, common carotid artery; ARR, aortic root replacement; AAR, ascending aorta replacement.