Spontaneous rupture of nonaneurysmal, noninfected atherosclerotic infrarenal aorta is extremely rare event.1–3 In most of the cases, it is due to penetrating atherosclerotic ulceration (PAU) of the aorta. This is a unique disease with distinct management and prognostic implications, first described by Stanson et al. as a distinct pathologic entity in 1986 in which ulceration penetrated through the internal elastic lamina into the media and was associated with a variable amount of hematoma within the aortic wall. It is an important clinical entity that must be distinguished from classic aortic dissection and rapid expansion or contained rupture of an aortic aneurysm. The disease predominantly affects thoracic aorta. PAU of the infrarenal aorta is a rare entity.1–4 Penetrating atherosclerotic aortic ulcers represent a condition in which an atherosclerotic plaque ulcerates and disrupts the internal elastic lamina, allowing intramural hematoma formation into the aortic media. In the absence of complication, progressive aneurysmal dilatation at the level of the ulcer is a rule.1–4 Although their natural history remains ill-defined, such ulcers may lead to intramural hematoma, aneurysm formation, dissection, embolization, or may present with its life-threatening complication—rupture of the aorta. Recently individualized as a distinct entity from all vascular painful syndromes, they affect preponderantly the elderly patients with positive medical history for hypertension and atherosclerosis and multiple cardiovascular risk factors.1–3 There are few reported cases of such lesions, and most of the published data is in regards to the thoracic aorta. The pathogenesis of PAU in aortic disease remains controversial. Computed tomography scanning and magnetic resonance imaging tend to replace aortography in providing data, informative for the ulceration and of its parietal extent. Moreover, they allow the differential diagnosis with aortic dissection or with intramural hematoma without intimal rupture which outcome and treatment should differ.1–5

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A case of spontaneous rupture through an atheromatous plaque with spontaneous rupture of infrarenal abdominal aorta is reported.
Case

An 80-years old male patient presented at ER with atypical GI (gastrointestinal) symptoms like pain and discomfort for more than 2 months. During this period he admits to more than 10 kg weight loss. There was no history of chest pain, back pain or hypotensive episode. The patient had history of arterial hypertension well controlled. An Echo-Doppler abdominal study showed a 2.3-cm-diameter abdominal aorta without periaortic blood flow. The CT scan showed rupture of atherosclerotic plaque into the aortic wall with formation of intramural hematoma approximately 15 mm wide and 8 cm long starting at the renal arteries, reaching 2 cm proximally to the aortic bifurcation (Figs. 1 and 2). Abdominal aorta presented with normal diameter, approximately 18–20 mm, with massive calcnosis. Hemodynamically the patient was stable. Laboratory findings were within normal range (serum Hgb level was 140 g/dL). Because of his gastrointestinal symptoms, adhesion of the III part of the duodenum to the aorta was suspected, so we decided to perform not endovascular but open surgical repair. After an informed consent was obtained an urgent open surgery operation was performed. Through midline laparotomy the abdominal aorta was exposed. Intraoperatively a firm adhesion of the distal part of the duodenum to the aortic wall was found. The duodenum was released and aortic cross-clamp was placed. The aorta was found heavily calcified, but with normal diameter. A specimen for culture and sensitivity was obtained from the hematoma. Interposition of straight silver Dacron graft No 18 was performed using 3/0 monofilament prolene suture. Postoperatively the patient had palpable pedal pulses. The culture was positive for Salmonella D, sensitive to gentamycin and amoxiclav. An adequate antibacterial treatment was carried out. The patient recovered uneventfully and was discharged from ICU at 7-th postoperative day and from hospital at 9-th postoperative day. The follow-up microbiological tests showed no presence of Salmonella D. At discharge he was hemodynamically stable. Laboratory findings were within normal range except for slight anemia (serum hemoglobin level was 98 g/dL).

Discussion

Intimal defects resulting from atherosclerotic ulcers occur in patients with advanced atherosclerosis. Although the natural history of PAU remains unclear, the intima of the affected aortic wall may distort and lead to pseudoaneurysmal formation, localized dissection, embolization or rupture. Some studies suggest that observation might be considered first because of the low incidence of dissection or rupture, whereas others advocate early surgical intervention for the treatment of PAU. Reports on the treatment of PAU by transluminal endovascular stent grafting have been accumulated. This seems to be a promising alternative to classical surgery, especially in elderly patients in poor condition, as it may reduce postoperative morbidity and mortality. In some cases, however, this endovascular treatment may be less beneficial than classical open surgery. In our case the patient presented with leading gastro-intestinal complaints, caused most probably by adhesion of the duodenum to the anterior aortic wall, which could not be corrected by an endovascular procedure. That is why after CT data for the position of the lesion we preferred to perform an open surgery. Intraoperatively the presence of that kind of adhesion was confirmed and the duodenum was released with a limited dissection. After the repair of the abdominal aorta, gastrointestinal complaints of the patient improved and eventually disappeared. The patient recovered uneventfully and was discharged from the hospital.

Fig. 1. CT angiogram, showing PAU with intramural hematoma of the abdominal aorta (reconstruction). IMH, intramural hematoma; AA, abdominal aorta.
at 9-th day after the operation. At early follow up the
GI symptoms were absent and the patient gained
weight. Unfortunately he passed away at 28-th post-
operative day from nonrelated cause (stroke).

The number of reported cases with PAU of the
abdominal aorta that requires surgical or nonsurgical
treatment is increasing. In most cases it affects thoracic
aorta, while infrarenal abdominal aorta is rare
location. In patients with severe atherosclerosis,
suffering from severe, intractable chest or back pain,
or with obscure abdominal pain without any evidence
of aortic dissection, a PAU should be the first of
differential diagnoses. Once a presence of a PAU is
diagnosed, a decision for surgical, endovascular or
nonsurgical treatment must be taken.

Fig. 2. CT angiogram, showing PAU with intramural
hematoma of the abdominal aorta (axial view).

References
1 Stanson AW, Kazmier FJ, Hollier LH et al. Penetrating
atherosclerotic ulcers of the thoracic aorta: natural history and
2 Welch TJ, Stanson AW, Sheedy PF et al. Radiologic evaluation of
3 Goldstein DJ, Flores RM, Todd GJ. Rupture of nonaneurysmal
4 Cook JP, Kazmier FJ, Arzulak TA et al. Penetrating aortic ulcer:
pathologic manifestations, diagnosis, and management. Mayo Clin
5 Vasquez J, Poultides GA, Lorenzo AC et al. Endovascular stent-
graft placement for nonaneurysmal infrarenal aortic rupture: a
388.

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