therapies. In one third of the cases atypical chest pain was reported and only 9 of 51 cases led to cardiac tamponade [1, 2]. In 7 of 9 described tamponades, prodromes such as thoracic pain, coughing, palpitations, and hiccups prior to hemodynamic instability were observed [1, 4]. Extremely rare late acute tamponade, occurring without prodromes, as in our case, may prove fatal if not promptly diagnosed.

Reviewing the literature we have found descriptions of two pathomechanisms responsible for the development of late cardiac tamponade as a complication of device implantation: myocardial perforation and lead-related exudative pericarditis. It has been shown that late perforations occur more frequently when active fixation leads are used [5]. In such cases the most plausible explanation for delayed cardiac wall perforation is gradual protrusion of the helix of the active fixation lead [6].

In our case late acute tamponade occurred due to erosion of the parietal pericardium with an intact visceral layer. The ulceration penetrated deep enough to damage one of the adjacent arteries, which was identified as the source of bleeding. The erosion probably arose from a "pressure sore" mechanism. We propose that the bulging end of the atrial lead led to repeated irritation of the peritoneal layer, causing damage and hence life-threatening bleeding.

Management strategies for cardiac tamponade caused by device implantation are not covered by current guidelines. Hence, the treatment has to be individualized in each case. After pericardial drainage it needs to be decided if the lead should be repositioned or left in place. If intervention is chosen, according to a recently published review, percutaneous techniques compared with surgical methods are more commonly used [7]. Surgical approach is limited to cases with severe clinical presentation or after percutaneous extraction failure [8]. One well known predictor of percutaneous extraction failure is the length of the post-implantation period. Therefore, some authors suggest that in cases of late perforation without lead malfunction its extraction is not obligatory, even when tamponade complicates the perforation [2, 4].

On the contrary, the case presented shows that conservative strategy may not be sufficient to prevent a relapse of pericardial bleeding and could lead to fatal consequences. Therefore, unless the underlying pathomechanism of late implantable device-related tamponade is evident (such as clear protrusion of the tip of the electrode on computed tomography, or drainage of typical inflammatory fluid from the pericardial cavity) sternotomy and pericardial exploration should be regarded as the first-choice treatment.

Moreover, the presented complication points to a lifelong risk of the use of atrial active fixation leads. It involves acute bleeding and therefore is unpredictable and difficult to prevent.

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Infected False Aneurysm of the Aortic Arch After Endoscopic Transurethral Instillation of Bacillus Calmette-Guérin

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The case of an 81-year-old male operated on for an infected false aneurysm of the aortic arch by *Mycobacterium bovis* (*M. bovis*) is described. Arch reconstruction with cryopreserved aortic patch was successfully performed under hypothermic circulatory arrest. Antituberculous chemotherapy was given for 12 months and presently the patient is leading a normal life. Vascular infection after bacillus Calmette-Guérin bladder therapy is uncommon and aortic arch involvement near exceptional. This diagnosis has to be considered in patients with such previous urologic interventions.

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Bacillus Calmette-Guérin bladder therapy is widely used as an effective treatment for different stages of bladder cancer. Vascular infection secondary to this attenuated live strain of *Mycobacterium bovis* (*M. bovis*) is uncommon and aortic arch involvement near exceptional. Nevertheless, to enhance a successful treatment this

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Abbreviations and Acronyms

ALAT = aspartate aminotransferase
AP = alkaline phosphatase
ASAT = aspartate transaminase
BCG = bacillus Calmette-Guérin
COPD = chronic obstructive pulmonary disease
CT = computed tomography
GGT = gamma-glutamyl transpeptidase
PAS = periodic acid-Schiff stain
TB = total bilirubin

differential diagnosis has to be considered in patients with such previous urologic interventions.

An 81-year-old man was admitted to our institution because of a 2-month history of malaise and 10-kg weight loss. He had a 60 pack/year smoking history, chronic obstructive pulmonary disease, and an in situ transitional cell carcinoma of the bladder, successfully treated 1 year earlier with transurethral endoscopic instillations of bacillus Calmette-Guérin (BCG).

On physical examination, there was a 4-cm tender hepatomegaly. The patient was not icteric. Laboratory tests revealed a mild elevation in transaminase count (alanine aminotransferase 67 IU/L, aspartate aminotransferase 76 IU/L) and dissociated cholestasis (total bilirubin 0.7 mg/dL, gamma-glutamyl transpeptidase 585 IU/L, alkaline phosphatase 701 IU/L). Regular aerobic and anaerobic blood cultures were negative. Transthoracic echocardiography showed good biventricular function and no signs of endocarditis. An ultrasound-guided liver biopsy was performed because of enzyme abnormalities. Granulomatous hepatitis with negative periodic acid-Schiff and Ziehl-Neelsen staining was confirmed.

Thoracoabdominal computed tomography showed neither recurrence of bladder cancer nor other malignancies. There were no signs of pulmonary infection, empyema, or abdominal abscess. An incidental 32 × 25 mm aneurysm of the aortic arch involving the origin of the left subclavian artery was found and the diagnosis of a mycotic aneurysm was suspected (Fig 1A, 1B).

The association of granulomatous hepatitis, a possibly infected aneurysm, and previous history of BCG therapy 1 year earlier suggested the diagnosis of a vascular *M. bovis* infection. Empiric chemotherapy with isoniazid, rifampicin, and ethambutol was started. A control computed tomography scan performed 2 weeks later showed rapid aneurysm growth, now measuring 52 × 45 mm (Fig 2A, 2B). Surgical treatment was indicated on an urgent basis.

Under general anesthesia with single-lung ventilation, a left thoracotomy through the fourth intercostal space was performed. Cardiopulmonary bypass was established through femoral arterial and venous cannulation. Dense adhesions between the left superior pulmonary lobe and the aneurysm required a pulmonary wedge resection. Circulatory arrest was established at 18°C core temperature. Opening of the false aneurysm wall and thrombus removal revealed a 10-mm defect in the anterior aspect of the aortic arch at the origin of the left subclavian artery. The surrounding area was severely calcified. Resection of the proximal left subclavian artery and regional arch resection were performed. The aortic arch was reconstructed with a patch from a cryopreserved thoracic aorta from our institutional tissue bank. Hypothermic circulatory arrest and cardiopulmonary bypass times were 14 and 147 minutes, respectively. A short arrest time was related to limited segmental resection and homograft patch reconstruction of the aortic arch. Homograft interposition to the left subclavian was considered but deemed technically difficult in view of the extensive regional inflammatory reaction; thus the left subclavian artery was ligated.

Cultures of the false aneurysm wall and intraaortic thrombus grew *M. bovis*. There was postoperative mild acute non-dialytic kidney injury and the patient was discharged uneventfully on postoperative day 18. The patient has completed a 12-month course of prednisone, isoniazid, rifampicin, and ethambutol and currently leads a normal life. Left upper limb ischemia has not been detected during follow-up and CT angiography 8 months

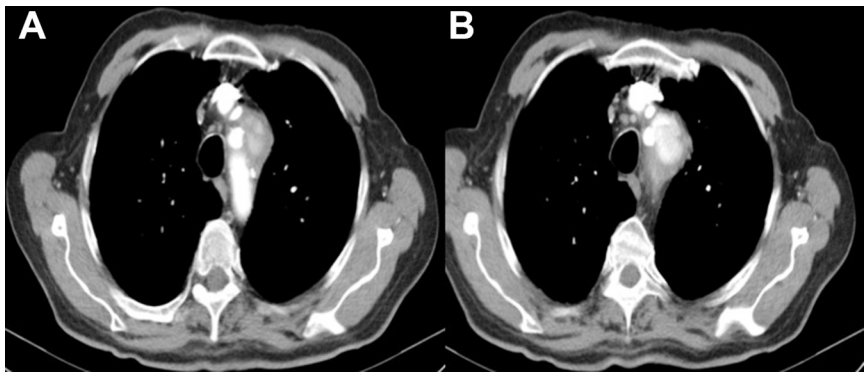

Fig 1. (A) and (B) Computed tomography on admission shows aortic arch dilatation.