dissection area in myocardial perfusion scintigraphy. Stent implantation was not thought to be appropriate because of long segment dissection of a small size (<2mm) side branch coronary artery dissection, which would require multiple overlapping stent implementations. In addition, absence of angina and lacking of ischemic area extension encouraged to do continuing follow-ups under medical therapy.

Conclusion
Spontaneous coronary dissection should be kept in mind as one of the possible causes of acute coronary syndromes even though it rarely exists. Although there is no definitive guideline for optimal treatment of SCAD, from the experiences of reported series in the literature, it is suggested that medical treatment with close follow-ups for stable patients with a preserved good left ventricular function could be beneficial.

References

Pacemaker lead failure due to crush injury

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Introduction
Different techniques for pacemaker implantation such as subclavian, cephalic and transiliac vein are being used today for various circumstances (1, 2). One of the potential complications of pacemaker implantation is the lead fracture or insulation failure due to crush injury. It usually occurs after medial intrathoracic puncture of the subclavian vein and results in damaging of the pacemaker lead body by entrapment within the costoclavicular ligament and/or the subclavian muscle (3, 4). The present case report describes a patient who underwent pacemaker implantation seven years ago and developed lead failure due to crush injury detected by chest X-ray and telemetry data.

Case report
A 33-year-old woman with a history of sick sinus syndrome underwent a dual chamber pacemaker implantation (Ela DR213 Talent, Ela 4068 for atrial, and BT46D for ventricular leads) seven years ago. Because of battery depletion the pulse generator was replaced with a Guidant 1296 generator. PACing threshold, R wave and impedance of ventricular lead during implant were 0.7 V at 0.5 msec, 7 mV and 350 ohms, respectively. PACing threshold, R wave and impedance of atrial lead were 1.2 V at 0.5 msec, 3 mV and 650 ohms, respectively. During follow-up telemetry data revealed intermittent sensing and pacing problem with the atrial lead. Measured impedance values were changing day by day between 540 and 1320 ohms, as shown in Table 1. Intermittent major changes in impedance values were suggestive of a lead malfunction, namely fracture that might be related to crush injury. The intracardiac electrocardiogram recordings revealed multiple artifacts and noise in the atrial channel (Fig. 1). A chest X-ray showed partial thinning and damage to the atrial lead body right at the medial puncture site of the subclavian vein (Fig. 2). All of these findings together with sensing failure probably reflected a partial fracture in the lead body, which was not detected during the implant. Since the patient had good intrinsic sinus rhythm at a rate of 55 bpm and potential complication risk during a subsequent lead revision, which was also refused by the patient, we reprogrammed the generator to VVIR mode that was well tolerated by the patient during follow-up.

Discussion
Subclavian vein puncture is commonly performed to insert the lead for permanent pacemakers and implantable defibrillators. Intrathoracic subclavian vein approach is performed in more than 85% of all endocardial leads (5). However, this medial puncture technique is potentially responsible for increased risk of lead fracture, pneumothorax and...
hemothorax (6). Lead fracture occurs in approximately 1-4% of permanent pacing systems whereas its incidence in transvenous cardioverter defibrillator systems is not well established (7). Subclavian crush syndrome is a well-known cause of pacemaker lead failure, namely conductor fractures and insulation failure by compression of the lead between the first rib and the clavicle (8). Besides cephalic vein cut-down technique, extrathoracic axillary vein puncture is currently suggested as an alternative technique for venous access to avoid crush injury. This novel technique may also be performed by giving some contrast agent through the ipsilateral brachial vein for guidance (5). Furthermore, ultrasound guidance for subclavian vein puncture may also be useful and effective for pacing lead insertion (10). Belott (11), comprehensively described how to safely perform this technique, in a recently published review. Axillary vein can be accessed blindly through the incision with a needle puncture 1 or 2 cm medial and parallel to the deltopectoral groove at the level of the coracoid process. Furthermore, the first rib is a key fluoroscopic landmark. Use of the first rib for orientation is recommended to avoid pneumothorax. The first step in accessing the axillary vein using the first rib is to place the 18-gauge percutaneous needle and syringe on top of the pectoralis major muscle in the superior aspect of the incision. Using fluoroscopy, the needle tip is placed in the middle of the first rib (Fig. 3). The angle of the syringe and needle is gradually increased as the needle is advanced through the pectoralis major muscle. Needle advancement is continued until the first rib is struck. Once the first rib is touched, the needle and syringe are slowly withdrawn under suction until the vein is entered. Once the vein is entered, the guidewire is passed and the sheath applied per standard technique. If axillary vein cannot be found by this technique, the use of radiographic contrast or ultrasound to visualize the axillary vein is recommended (11). As previously proposed by other authors (5,11), we also recommend the extrathoracic subclavian or axillary vein approach for implantation of pacemaker leads and suggest that the classic intrathoracic subclavian approach should be abandoned. Telemetric evaluation during follow-up should always be performed in order to determine the measured data such as impedance and thresholds. Since detection of lead failure may be intermittent, it may be overlooked during pacemaker follow-up. When the lead impedance is measured below 200 ohms, one may suspect of

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Akciğer kist hidatıği ameliyatı sonrasında görülen kardiyak kist hidatik olgusu

A cardiac hydatid cyst case seen after operation on pulmonary hydatid cyst

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Giriş

Ekinokokus granulösis’in etken olduğu kist hidatik siklikla karaciğer ve akciğer yerleşimlir (1). Kardiyak yerleşim oldukça nadirdir. Tüm kist hidatik olguların ancak %0.5–2’si kardiyak yerleşimidir (1). Kardiyak yerleşim bölgeleri içinde de siklikla sol ventrikül, nadiren sağ ventrikül ve interventriküller septum (IVS) yerleşimidir (2). Hastanemizde bilateral akciğer kist hidatik nedeniyle oper edildiğinde sonra interventriküller septum yerleşimli kardiyak kist hidatik sahibi bir olguyumuz mağlup çıktı.

Olgu sunumu

Alts ay önce sol akciğer, üç ay önce de sağ akciğer kist hidatikta tanılıyordu. Olgunun edilen 41 yaşında erkek hasta rutin kontrol amacıyla hastanemize başvurdu. Belirgin bir yakınlama yoktu ve fizik muayene niteliğinde patoloji bulgu sahibi değildir. Rutin kan testlerinde ve elektrokardiografisinde patoloji bulgu yoktu. Çekilen akciğer grafisinde yeni kistik yapışik görüldü (Resim 1). Transtorasik anayşanın ançak %0.5–2’si kardiyak yerleşimidir (1). Kardiyak yerleşim bölgeleri içinde de siklikla sol ventrikül, nadiren sağ ventrikül ve interventriküller septum (IVS) yerleşimidir (2). Hastanemizde bilateral akciğer kist hidatik nedeniyle oper edildiğinde sonra interventriküller septum yerleşimli kardiyak kist hidatik sahibi bir olguyumuz mağlup çıktı.


Tartışma