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 implantations. 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

1. Basso C, Morgagni GL, Thiene G. Spontaneous coronary artery dissection: a neglected cause of acute myocardial ischaemia and sudden death. Heart 1996; 75: 451-4.
2. Hamilos MI, Kochiadakis GE, Skalidis EI, Igoumenidis NE, Chrysostomakis SI, Vardakis KE, et al. Acute myocardial infarction in a patient with spontaneous coronary artery dissection. Hellenic J Cardiol 2003; 44: 348-51.
3. Dorros G, Cowley MJ, Simpson J, Bentivoglio LG, Block PC, Bourassa M, et al. Percutaneous transluminal coronary angioplasty: report of complications from the National Heart, Lung, Blood Institute PTCA registry. Circulation 1983; 67: 723-30.
4. Pretty HC. Dissecting aneurysm of coronary artery in a woman aged 42. Br Med J 1931; 1: 667.
5. Lakshmi V, Umesan CV, Radhakrishnan N, Dhas L, Ajit SM, Cherian KM. spontaneous coronary artery dissection: An institutional review. Indian Heart J 2001; 53: 567.
6. Celic SK, Sagcan A, Altintig A, Yuksel M, Akin M, Kultursay H. Primary spontaneous coronary artery dissections in atherosclerotic patients. Report of nine cases with review of the literature. Eur J Cardiothoracic S 2001; 20: 573-6.
7. Maehara A, Mintz GS, Castagna MT. Intravascular ultrasound assessment of spontaneous coronary artery dissection. Am J Cardiol 2002; 89: 466-8.
8. Zampieri P, Aggio S, Roncon L, Rinuncini M, Canova C, Zanazzi G, et al. Follow up after spontaneous coronary artery dissection: a report of five cases. Heart 1996; 75: 451-4.
9. Vale PR, Baron DW. Coronary artery stenting for spontaneous coronary artery dissection: case report and review of the literature. Cathet Cardiovasc Diagn 1998; 45: 280-6.
10. DeMaio SJ, Kinsella SH, Silverman ME. Clinical course and long-term prognosis of spontaneous coronary artery dissection. Am J Cardiol 1989; 64: 471-4.

Pacemaker lead failure due to crush injury

Ezilme hasarına bağlı kalıcı kalp pili elektrod kusuru

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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 65% of all endocardial leads (5). However, this medial puncture technique is potentially responsible for increased risk of lead fracture, pneumothorax and

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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

Table 1. Stored data of atrial lead impedance measured automatically at various intervals through the generator

Date	Impedance, Ohm
29 May 2005	1070
28 May 2005	600
27 May 2005	1070
26 May 2005	540
25 May 2005	540
24 May 2005	1320
23 May 2005	580
18 May 2005	650
11 May 2005	910
04 May 2005	790
27 Apr 2005	640
20 Apr 2005	550

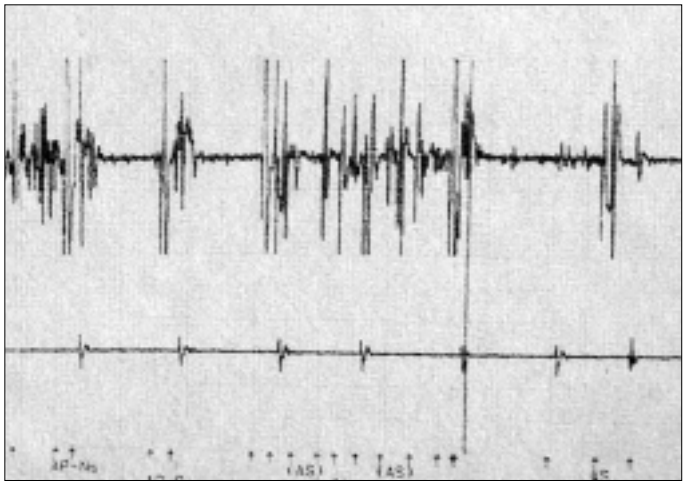

Figure 1. Demonstration of intracardiac noise in the atrial channel

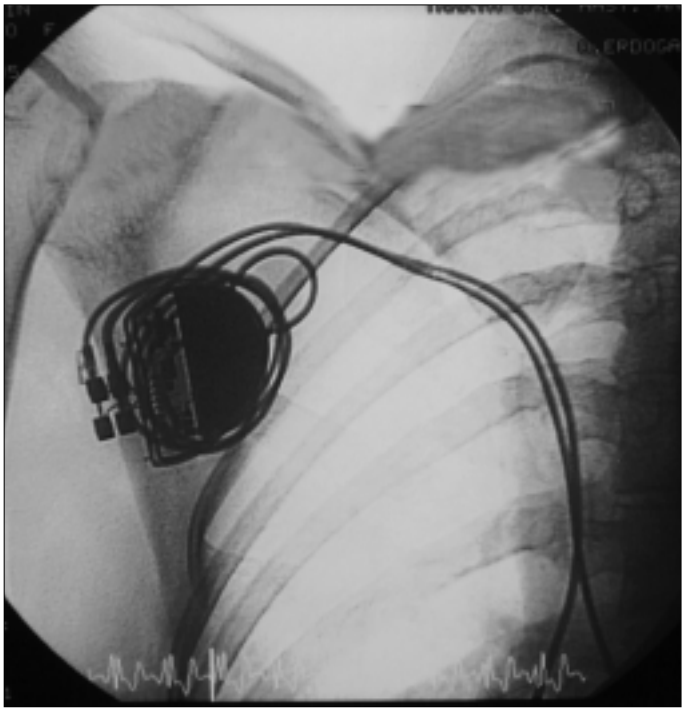

Figure 2. Chest X-ray showing lead damage and thinning at the medial sternoclavicular junction due to crush injury

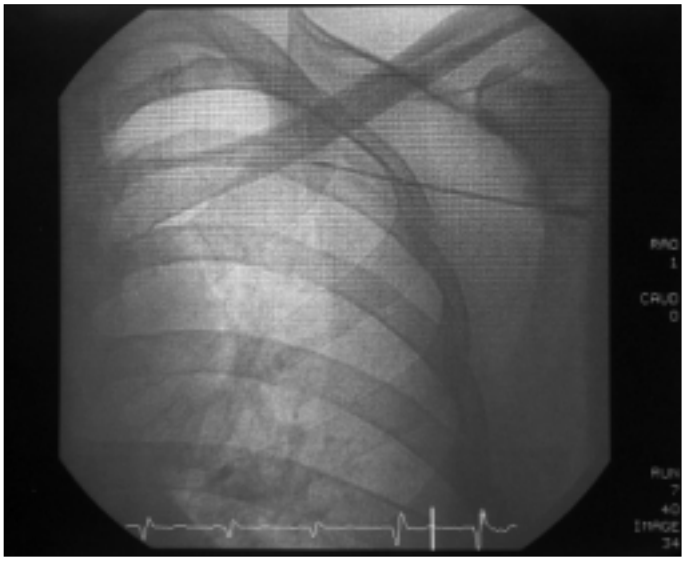

Figure 3. Fluoroscopic view demonstrating appropriate puncture site of the axillary vein aiming the first rib as a landmark at the lateral 1/3 border of the line between the corocoid process and manubrosternal angle

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an insulation failure; whereas an impedance measured more than 1000 ohms is suggestive of lead fracture. Intracardiac electrocardiograms should also be checked for detection of noise. As it was shown in the present case, chest X-ray may reveal damage to the lead body right at the sternoclavicular region and intracardiac electrocardiographic detection of noise should not be neglected.

Conclusion

In conclusion, we propose to perform extrathoracic subclavian or axillary vein approach for implantation of pacemaker leads and always check the lead status by telemetry during follow-up.

References

1. Belott PH. Implant techniques. In: Kusumato FM, Goldschlager NF, editors. Cardiac Pacing for the Clinician. Philadelphia: Lippincott Williams and Wilkins, 2001: p, 91-162.
2. Erdogan O, Augostini R, Saliba W, Juratli N, Wilkoff BL. Transiliac permanent pacemaker implantation after extraction of infected pectoral pacemaker systems. Am J Cardiol 1999; 84: 474-5.
3. Altun A, Erdogan O. Pacemaker lead failure suggestive of crush injury. Cardiol Rev 2003; 11: 256.
4. Suzuki Y, Fujimori S, Sakai M, Ohkawa S, Ueda K. A case of pacemaker lead fracture associated with thoracic outlet syndrome. Pacing Clin Electrophysiol 1988; 11: 326-30.
5. Ramza BM, Rosenthal L, Hui R, Nsah E, Savader S, Lawrence JH, et al. Safety and effectiveness of placement of pacemaker and defibrillator leads in the axillary vein guided by contrast venography. Am J Cardiol 1997; 80: 892-6.
6. Chauhan A, Grace AA, Newell SA, Stone DL, Shapiro LM, Schofield PM, et al. Early complications after dual chamber versus single chamber pacemaker implantation. Pacing Clin Electrophysiol 1994; 17: 2012-5.
7. Gallik DM, Ben-Zur UM, Gross JN, Furman S. Lead fracture in cephalic versus subclavian approach with transvenous implantable cardioverter defibrillator systems. Pacing Clin Electrophysiol 1996; 19: 1089-94.
8. Magney JE, Flynn DM, Parsons JA, Staplin DH, Chin-Purcell MV, Milstein S, et al. Anatomical mechanisms explaining damage to pacemaker leads, defibrillator leads, and failure of central venous catheters adjacent to the sternoclavicular joint. Pacing Clin Electrophysiol 1993; 16: 445-57.
9. Burri H, Sunthorn H, Dorsaz PA, Shah D. Prospective study of axillary vein puncture with or without contrast venography for pacemaker and defibrillator lead implantation. Pacing Clin Electrophysiol 2005; 28: 280-3.
10. Orihashi K, Imai K, Sato K, Hamamoto M, Okada K, Sueda T. Extrathoracic subclavian venipuncture under ultrasound guidance. Circ J 2005; 69: 1111-5.
11. Belott P. How to access the axillary vein. Heart Rhythm 2006; 3: 366-9.

Akciğer kist hidatiği 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 granülosis'in etken olduğu kist hidatik sıklıkla karaciğer ve akciğer yerleşimlidir (1). Kardiyak yerleşim oldukça nadirdir. Tüm kist hidatik olgularının ancak % 0.5–2'si kardiyak yerleşimlidir (1). Kardiyak yerleşim bölgeleri içinde de sıklıkla sol ventrikül, nadiren sağ ventrikül ve interventriküler septum (İVS) yerleşimlidir (2). Hastanemizde bilateral akciğer kist hidatiği nedeniyle opere edildikten sonra interventriküler septum yerleşimli kardiyak kist hidatik saptanan bir olguyu sunmayı amaçladık.

Olgu sunumu

Altı ay önce sol akciğer, üç ay önce de sağ akciğer kist hidatiği tanılarıyla ameliyat edilen 41 yaşındaki erkek hasta rutin kontrol amacıyla hastanemize başvurdu. Belirgin bir yakınması yoktu ve fizik muayenede patolojik bulgu saptanmadı. Rutin kan testlerinde ve elektrokardiyografisinde patolojik bulgu yoktu. Çekilen akciğer grafisinde yeni kistik oluşum gözlenmedi. Göğüs tomografisinde daha önceki tomografilerinde saptanmayan interventriküler septumda 1.5-2 cm boyutlarında kist hidatikle uyumlu olabilecek lezyon görüldü (Resim 1). Transtorasik ekokardiyografide sağ ventrikül içinde İVS'den köken alan kistik oluşum saptandı (Resim 2). Hastaya albendazol tedavisi başlandı ve kardiyak kist hidatik tanısıyla operasyona alındı. Standart kanülasyon, hafif hipotermi ve kan kardiyoplejisi takiben sağ atriyotomi yapıldı. Triküspid kapak ekarte edildiğinde sağ ventrikülde İVS'un büyük kısmını işgal eden kistik yapı görüldü (Resim 3). Hipertonik salin solüsyonu (%15) kist içine enjekte edildi beş adet germinatif membran ve iki adet kist çıkarıldı. Kist kavitesi hipertonik salin solüsyonu ile yıkandı ve 4/0 prolen ile poş kapatıldı. Ameliyat sonrası problemsiz seyreden hasta altı ay albendazol kullanımı önerilerek 9. günde taburcu edildi.

Tartışma

Kist hidatik ekinokokus granülosis'in neden olduğu bir doku enfeksiyonudur. Özellikle tropikal ve bazı subtropik bölgelerde, hayvancılıkla uğraşılan yörelerde sık görülen önemli bir sağlık problemidir. Bizim hastamız da hayvanlarla yakın temas içinde bulunan bir çiftçiydi.

Kist hidatik başta karaciğer olmak üzere akciğer, dalak, santral sinir sistemi ve kaslara yerleşebilir (3). Kardiyak yerleşim çok nadir olmasına karşın, ekinokoklar koroner dolaşımla miyokarda da ulaşabilirler. Kalpteki yerleşim yerleri bölgesel kanlanma miktarına bağlıdır. Sağ

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