

sodium channels by TCA (6). The use of sodium bicarbonate in tricyclic poisoning has been shown to have beneficial effects (7).

In a review of 91 patients treated with sodium bicarbonate, hypotension was corrected in 20 of 21 patients (96%) within one hour and QRS prolongation was corrected in 39 of 49 patients (80%). The administration of hypertonic sodium chloride to rats with desipramine toxicity has been shown to be as effective as sodium bicarbonate in reversing QRS prolongation and hypotension (8). In the light of this knowledge, the first drug that we administered to our patient was sodium bicarbonate infusion, but it did not make any change in the cardiac rhythm. So, we gave lidocaine and beta-blocker to solve the problem.

Our patient's arrhythmia gave a good response to beta-blocker

therapy. In the literature, use of beta-blockers is reported as being effective in treating arrhythmias both in humans (8) and animals (10), but as a side effect, a marked decrease in blood pressure was reported in all cases. We did not record a marked decrease in our patient's blood pressure. It was either because she did not have hypotension or having been given intravenous isotonic crystalloid solution from the time of admittance.

Because our patient gave a good response to beta-blocker therapy, we suggest that selective beta-blockers may be an effective treatment modality in children with ventricular tachyarrhythmias, which is due to severe amitriptyline intoxication especially when there is no hypotension.

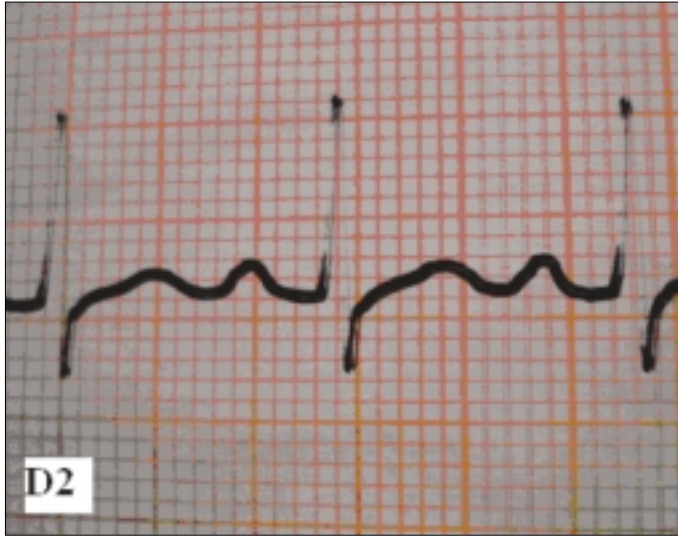


Figure 2. ECG tracing (D2) of the patient after the beta-blocker therapy
ECG- electrocardiogram

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Infective endarteritis in a 2-month-old infant associated with silent patent ductus arteriosus

Sessiz patent ductus arteriozus'lu 2 aylık bir çocukta infektif endarterit

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Introduction

Infective endarteritis (IE), which was a serious complication of clinically apparent patent ductus arteriosus (PDA), has become extremely rare because of early closure of PDA (1-3). However, it is not known if silent PDA, which is not detectable by cardiac auscultation but can be recognized only by colored-flow echocardiography, increases the risk of IE or not (4). Thus, antibiotic prophylaxis or closure

of silent PDA against the risk of IE is not recommended (4-6). There are only a few reports of IE associated with silent PDA in medical literature (7, 8). In this article a case of IE in a 2 month-old infant with silent PDA is reported.

Case report

A 2-month-old male infant was hospitalized because of fever lasting for three days. His physical examination revealed 37.6 C° axil-

lary's temperature, mild pharyngeal erythema, 2 cm hepatomegaly below the right lower costal margin, but no pathological heart sound or murmur and no splenomegaly.

Physical examinations of the other systems were normal. The patient was not anemic, the number of thrombocytes was normal and the white blood cell count was 10600/mm³ with the dominance of polymorphonuclear granulocytes. Erythrocyte sedimentation rate was 34 mm/hour. Urine analysis, serum urea, creatinine, aspartate aminotransferase and alanine aminotransferase, immunoglobulin levels were normal. β-hemolytic, group D streptococcus was grown in his blood culture after three days of his admission to the hospital. The 2-dimensional and color Doppler echocardiography was performed to investigate the origin of bacteremia. It revealed 2 vegetations with the diameters of 2.5 and 2.9 mm on the left side of the wall of the main pulmonary artery, and very tiny retrograde turbulent flow into the left side of main pulmonary artery consistent with PDA (Fig. 1, 2a). Continuous flow pattern that was typical for PDA was hardly demonstrated by HPRF Doppler (Fig. 2b). There was no sign of pulmonary hypertension or widening of the left heart chambers as a sign of left ventricular volume overload. Treatment with penicillin G and gentamicin was started according to antibiogram. He became afebrile two days after the treatment; blood culture that was obtained one week later after the beginning of the treatment became sterile. The patient completed six-week course of antibiotics. The size of the

vegetation decreased and eventually disappeared. Patency of ductus arteriosus that was not detectable by auscultation persisted after the patient was 6 months old at follow-up. Sweat test, employed because of family history of cystic fibrosis in a brother and asymptomatic high level of sweat chloride in his mother, revealed a chloride level of 68 mEq/L. Later development of frequent lower respiratory tract infections, pseudo-Barter syndrome that was recognized by alkalosis associated with hypokalemia and positive F508 mutation test confirmed the diagnosis of cystic fibrosis. Transcatheter closure of the PDA was suggested but the family preferred surgical ligation. Thoracotomy without cardiopulmonary bypass revealed presence of small PDA and uneventful ligation of the PDA was performed at age of six months.

Discussion

During the pre-antibiotic era, IE was a fatal complication of PDA and the annual risk of IE in these patients was estimated to be 0-45% (1, 2). Especially in the industrialized world, infective endarteritis complicating PDA is very rare nowadays and the risk is very low (3, 5). Fatal cases of IE complicating patent ductus arteriosus were associated with large ductus. It is assumed that a small duct to be less susceptible than a large one to IE (5, 6). On the other hand, in developing countries the incidence of IE complicating patent ductus arteriosus is much closer to the natural history of PDA in pre-antibiotic era (9). Every isolated PDA with an audible and typical continuous murmur should be closed irrespective of the size, because untreated persistent ductus arteriosus is a favorable site of IE (5, 6). Surgical or transcatheter occlusion of PDA eliminates the risk of IE.

The term silent ductus arteriosus was coined to describe the preterm infant with respiratory distress syndrome in whom continuous murmur is not heard (4). Doppler ultrasonography has made possible to detect very small PDA that can not be identified clinically. Houston et al (4) suggested that some patients considered to have IE with a normal heart might have a silent PDA or evidence of such an association would justify ligation or antibiotic cover as prophylactic measures. However, they did not recommend endocarditis prophylaxis for every silent ductus.

There is no currently published evidence that the incidence of IE in patients with hemodynamically trivial, silent PDA have increased. But Balzer et al (7) reported a case of endarteritis associated with a clinically silent PDA. They suggested that all cases of PDAs regardless of its clinical characteristics should be closed. We also

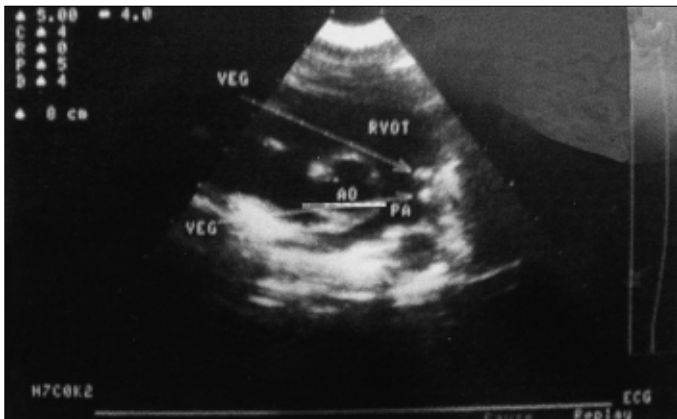


Figure 1. Parasternal short-axis view demonstrates echo dense vegetations (arrows) on the left wall of the main pulmonary artery

AO- aorta, PA- pulmonary artery, RVOT- right ventricular outflow tract, VEG- vegetation

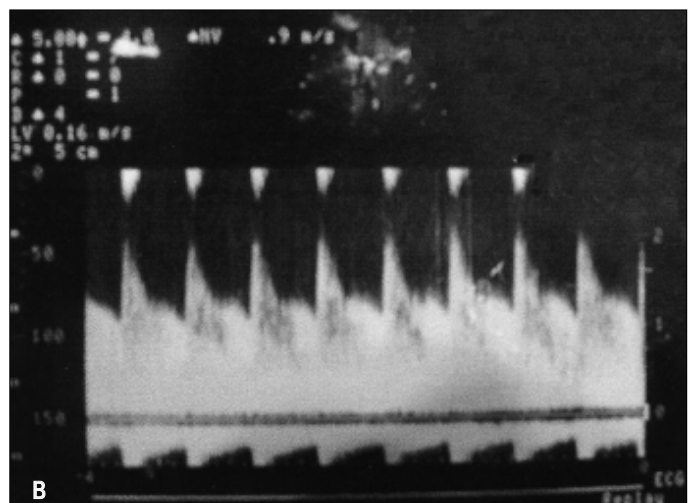
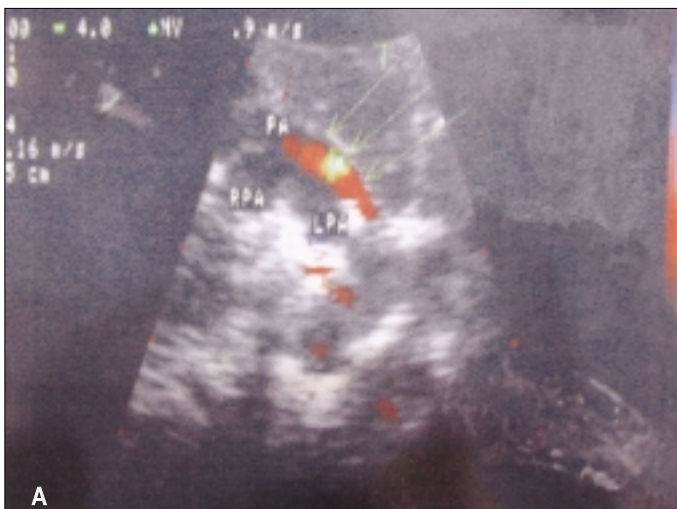


Figure 2. Color flow mapping from high parasternal short-axis view shows tiny retrograde turbulent flow into the left side of the main pulmonary artery (2A) and HPRF Doppler recording demonstrates typical continuous flow pattern (2B)

LPA- left pulmonary artery, PA- pulmonary artery, RPA- right pulmonary artery

report a case of IE associated with silent PDA. We think that cystic fibrosis is a coincidence since it is obvious that the incidence of IE has not been increased in patients with cystic fibrosis. Estimated prevalence of silent PDA's were found as 0.5-1% in normal population, but IE associated with silent PDAs were reported in only few patients before the present case (7, 8). On the other hand residual shunts, which are usually inaudible or associated with nonspecific systolic murmur, are not uncommon in patients who underwent transcatheter closure of PDA. Latson and his associates (10) reported that patients with silent PDA after device occlusion are not at a higher risk for developing endocarditis.

Although the natural history of a small patent arterial duct with a negligible left to right shunt is not known with certainty, this and previous reports may indicate that silent PDA has a risk of developing IE. In cases of unexplained fever and bacteremia, endarteritis with a pre-existing silent PDA should be considered and investigated by 2-dimensional and color Doppler echocardiography. But, it cannot be recommended that antibiotic prophylaxis or closure of every silent PDA routinely unless exact incidence of IE will be cleared out by long-term prospective studies in patients with silent PDA.

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Surgical removal of a migrated guidewire: a safe method

Intravenöz kılavuz telin cerrahi olarak çıkarılması; güvenli bir yöntem

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Introduction

The potential complications of percutaneous venous catheterizations are various and include pneumothorax, subclavian and carotid artery puncture, hematoma, air embolism, catheter malposition, catheter fragment embolization, venous thrombosis, infection and problems of guidewires (1). The rate of broken intravascular catheters has been estimated to be 0.1%, but no definitive data are available for other types of foreign objects such as stents, coils and broken or intact guidewires (2). Serious complication rate associated with foreign bodies in vascular system has been reported to be as high as 71%, and with a high mortality rate ranging between 24-60% (3). Importantly, these patients with intravascular objects are candidates for serious complications such as arrhythmias, perforation, thrombosis, and infection, which may be fatal in some instances. Foreign bodies left accidentally in intravascular compartments during invasive procedures have been reported in various publications. An intravascular foreign body is commonly an iatrogenic complication that occurs during arterial or venous catheterization including interventional procedures, and the foreign body could be either a catheter fragment, a coil or a guidewire. The danger of septic-thrombotic complications and risk of vascular perforation, makes urgent removal of the object by any technique mandatory.

We report a case of an accidental iatrogenic J-wire migration into vena cava during subclavian vein catheter insertion for central venous pressure monitoring in a woman and its unusual, safe and easy way of surgical removal. The guidewire was located in the venous route from the superior vena cava to the beginning of the right common iliac vein. The following report includes a different technique for removing foreign objects like guidewires without invasive radiological intervention.

Case report

A 57-year-old woman in the postoperative period of an abdominal surgery for colorectal cancer followed in surgical intensive care unit, underwent an attempt of subclavian vein catheter insertion by Seldinger technique, ended in inadvertently misplaced guidewire in the venous system. Immediate surgical exploration of subcutaneous tissue at the insertion site by general surgeons revealed no result. After cardiovascular surgery consultation, vascular evaluation was done beginning with routine X-ray. After serial X-rays, we saw that the straight tip was just at the caudal end of the vena cava, and the j-tip part of the catheter was located in right common iliac vein, where the wire was positioned in a route from superior vena cava through right atrium down to iliac vein (Fig. 1).

Because of the fact that the j-wire was intraluminal, anticoagulation with low molecular weight heparin was given. Absence of angiography