

more AV accesses throughout their life (6). In 298 patients, in whom lower-arm radiocephalic fistulas were created, the fistula patency was 74.1%, 64.2%, 49.8%, 33.7%, and 4.1% after 1, 2, 3, 4, and 5 years, respectively (7).

In this presented case with diabetes the patency is much lower, so the risk for CVS is higher. In most of the papers reporting on CVS after fistula creation on the ipsilateral arm of the pacemaker, the authors suggest that phlebography should systematically be performed before the fistula is created, in order to diagnose pre-existing CVS (8). On the other hand the risk of bleeding complications is much higher in ICD implantation in a vein perfused by an AV fistula because of increased blood flow and reduced vessel compliance. With these considerations in mind we decided to implant the device on the side of the fistula protecting the other side for future AV fistula access.

Conclusion

We recommend the implantation of a rhythm device on the side of the AV fistula in relatively young patients with old AV fistula due to the expected requirement for a new AV access, as it seems feasible despite a higher risk of perioperative bleeding complications.

References

1. Crespo EM, Kim J, Selzman KA. The use of implantable cardioverter defibrillators for the prevention of sudden cardiac death: a review of the evidence and implications. *Am J Med Sci* 2005; 329: 238-46.
2. Zuber M, Huber P, Fricker U, Buser P, Jager K. Assessment of the subclavian vein in patients with transvenous pacemaker leads. *Pacing Clin Electrophysiol* 1998; 21: 2621-30.
3. Agarwal AK, Patel BM, Haddad NJ. Central vein stenosis: a nephrologist's perspective. *Semin Dial* 2007; 20: 53-62.
4. Chuang CL, Tarnig DC, Yang WC, Huang TP. An occult cause of arteriovenous access failure: central vein stenosis from permanent pacemaker wire. Report of three cases and review of the literature. *Am J Nephrol* 2001; 21: 406-9.
5. Tourret J, Cluzel P, Tostivint I, Barrou B, Deray G, Bagnis Cl. Central venous stenosis as a complication of ipsilateral haemodialysis fistula and pacemaker. *Nephrol Dial Transplant* 2005; 20: 997-1001.
6. Lynn KL, Buttmore AL, Wells JE, Inkster JA, Roake JA, Morton JB. Long-term survival of arteriovenous fistulas in home hemodialysis patients. *Kidney Int* 2004; 65: 1890-6.
7. Erkut B, Ünlu Y, Ceviz M, Becit N, Ateş A, Çolak A, Koçak H. Primary arteriovenous fistulas in the forearm for hemodialysis: effect of miscellaneous factors in fistula patency. *Ren Fail* 2006; 28: 275-81.
8. Cavatorta F, Campisi S, Zollo A. Subclavian vein stenosis in hemodialysis patients. *Minerva Urol Nefrol* 1998; 50: 55-9.

Persistent atrial fibrillation associated with gastroesophageal reflux accompanied by hiatal hernia

Hiatal herni ile birlikte gözlenen gastroözofajiyal reflü ile ilişkili dirençli atriyal fibrilasyon

Hamza Duygu, Filiz Özerkan, Serkan Saygı, Serdar Akyüz

Department of Cardiology, Faculty of Medicine, Ege University, İzmir, Turkey

Introduction

Atrial fibrillation (AF) may be related to temporary causes and successful treatment of the underlying condition often eliminates AF (1). In this article, we present a case of persistent AF resistant to pharmacological and direct-current cardioversion and converted to sinus rhythm with a proton pump inhibitor (PPI) in a patient with paraesophageal hernia and gastroesophageal reflux (GER).

Case report

A 79-year-old woman presented to our department with complaints of palpitations for 10 days. The patient also complained of long-standing heartburn and daily acid regurgitation. She had history of hypertension and chronic obstructive pulmonary disease. The patient was an ex-smoker and was receiving amlodipine 5 mg once daily and budesonide turbuhaler 200 µg twice daily. Besides, he had not used any anti-acid drug regularly. On physical examination, her blood pressure

was 120/80 mm Hg, and pulse rate 160 beats per minute, arrhythmic. Examination of the heart revealed no abnormality except a rapid rhythm. Her electrocardiography showed AF with rapid ventricular response (Fig. 1A). There were no pathological findings on the laboratory examinations including arterial blood gas analysis. Thyroid disease and any other potential metabolic causes for tachycardia were excluded. Mild tricuspid and mitral regurgitation and mild to moderate pulmonary hypertension (pulmonary systolic artery pressure 40 mm Hg) were detected on transthoracic echocardiography. Left ventricular ejection fraction was 65% and left atrial end-diastolic diameter was 45 mm. Transesophageal echocardiography revealed left atrial spontaneous echo contrast without thrombus in left atrium and left atrial appendage. Upon this, firstly, heparin and intravenous verapamil was administered. But, it failed. Subsequently, electrical cardioversion was attempted. Sinus rhythm could not be achieved and we decided to rate control with verapamil and oral anticoagulation. Computed tomography (CT) scan of the thorax was performed due to complaint of non-productive cough. CT showed large hiatal hernia into the chest and barium swallow confirmed

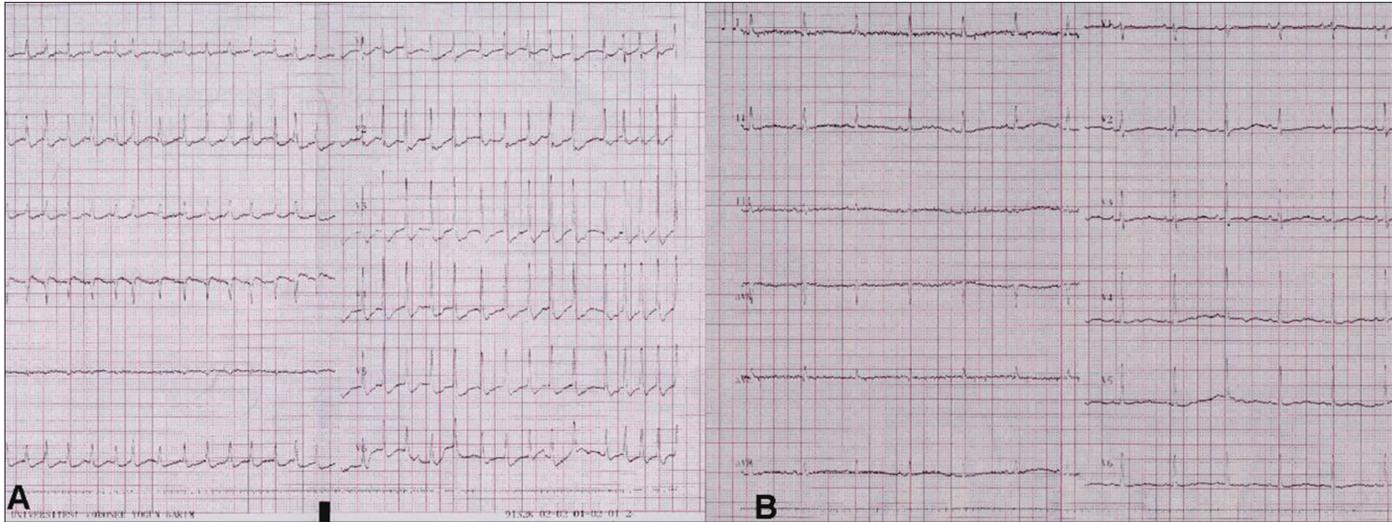


Figure 1. Twelve-lead electrocardiograms of atrial fibrillation (A) and sinus rhythm (B)



Figure 2. Barium swallow (A) and computed tomography scan of the thorax (B) demonstrating large hiatal hernia (arrows)

the paraesophageal hernia (Fig. 2). A 24-hour esophageal pH monitoring study demonstrated severe reflux. She was successfully treated conservatively with dietary control and a PPI (lansoprazole 30 mg twice daily). Two days later, the patient converted to sinus rhythm (Fig. 1B). Verapamil was discontinued and warfarin was used for one month. One year later, the patient felt well on lansoprazole therapy and sinus rhythm maintained.

Discussion

The physiological inter-relationship between the upper gastrointestinal tract and heart is well known (2). Dysrhythmias are common during any procedure that involves manipulation of the upper gastrointestinal tract, such as endoscopy of the esophagus or stomach. Atrial tachyarrhythmias have been rarely reported in patients with hiatal hernia and GER (3, 4). Schilling et al (3) demonstrated suppression of atrial flutter by repair of a large paraesophageal hernia.

A potential reduction in symptoms related to paroxysmal AF after treatment of erosive esophagitis and GER symptoms with PPI therapy has been previously described (5). The exact mechanisms for this potential relationship between hiatal hernia-acid reflux and atrial arrhythmias have not been previously explored. There are a number of possible reasons for this phenomenon. These arrhythmias may be induced by a mechanical effect on the left atrial wall that is related to the passage of food (3). If the hernia remained in the chest for long periods, compression of the left atrium by the hernia may have produced an area of relative

ischemia and anatomical block, resulting in re-entry (3). But no clear distortion of the left atrium was seen on echocardiography in our case. In patients with cardiac arrhythmias and heart disease, both vagal and sympathetic factors are thought to contribute to the onset of the arrhythmia. In the absence of heart disease, however, vagal factors are more likely to be responsible for the onset of arrhythmias (6). Increased vagal tone that might precipitate the onset of tachycardia (7). Finally, hypertension and chronic obstructive pulmonary disease might be contributing factors to development of AF (1).

Conclusion

This case report suggests that AF may be caused by a hiatus hernia and GER that PPI and palliative measures may abolish AF. Hiatal hernia and GER should be investigated as a potential pathogenetic mechanism in patients with AF resistant to therapy. The PPI therapy may reduce not only GER-related but also AF-related symptoms.

References

1. Fuster V, Ryden LE, Cannom DS, Crijns HJ, Curtis AB, Ellenbogen KA, et al. American College of Cardiology; American Heart Association Task Force; European Society of Cardiology Committee for Practice Guidelines; European Heart Rhythm Association; Heart Rhythm Society. ACC/AHA/ESC 2006 guidelines for the management of patients with atrial fibrillation: full text: a report of the American College of Cardiology/American Heart Association Task Force on practice guidelines and the European Society of Cardiology Committee for Practice Guidelines (Writing Committee to Revise the 2001 guidelines for the management of patients with atrial fibrillation) developed in collaboration with the European Heart Rhythm Association and the Heart Rhythm Society. *Europace* 2006; 8: 651-745.
2. Palmer ED. The abnormal upper gastrointestinal vagovagal reflexes that affect heart. *Am J Gastroenterol* 1976; 66: 513-22.
3. Schilling RJ, Kaye GC. Paroxysmal atrial flutter suppressed by repair of a large paraesophageal hernia. *Pacing Clin Electrophysiol* 1998; 21: 1303-5.
4. Landmark K, Storstein O. Ectopic atrial tachycardia on swallowing. *Acta Med Scand* 1979; 205: 251-4.
5. Weigl M, Gschwanter M, Gatterer E, Finsterer J, Stöllberger C. Reflux esophagitis in the pathogenesis of paroxysmal atrial fibrillation: results of a pilot study. *South Med J* 2003; 96: 1128-32.
6. Coumel P. Autonomic influences in atrial tachyarrhythmias. *J Cardiovasc Electrophysiol* 1996; 7: 999-1007.
7. Haughey B. Ingestion of cold fluids: related to onset of arrhythmias? *Crit Care Nurse* 1990; 10: 98-110.