than myocardial cells and endocardial fibers may be nourished from cavity blood (9). These surviving Purkinje fibers in infarct region demonstrate enhanced automaticity and triggered activity which may cause polymorphic VT when coupled with prolonged action potential duration (10). In current studies, most of the VPCs originating from Purkinje network were located in the border-zone of MI (5-7).These studies has shown that ablation of these triggers was able to eliminate arrhythmias. Similar results were also demonstrated for patients early after MI (6).

However, it is not always easy to find and abolish Purkinje potentials during electrical storm. For instance, we were not able to localize Purkinje potentials constantly because of repetitive hemodynamically unstable VTs. During the procedure we observed Purkinje like potentials where the earliest endocardial activation regions of VPCs were. After successful RF applications we didn't observe these signals. Our report is result of a single case and more studies are needed to elucidate the mechanisms of polymorphic VT after MI. Unfortunately, follow-up period was too short due to concomitant diseases and this is an obvious limitation of our report.

Conclusion

Catheter ablation plays increasingly important role in management of electrical storm after MI. RF ablation is indicated in recurrent polymorphic VT or VF when specific triggers can be targeted (2). In these cases accelerated ablation approach may help reaching to a safe harbor.

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Successful ablation of cavo-tricuspid isthmus dependent atrial flutter in a patient with Senning operation

Senning operasyonlu hastada kavo-triküspit istmus bağımlı atriyal flutterin başarılı ablasyonu

Introduction

Atrial flutter is a common complication late after atrial switch operation for transposition of the great arteries. It is usually cavotricuspid isthmus (CTI) dependent (1). Radiofrequency catheter ablation (RFCA) targeting CTI region may eliminate flutter (2). Access to targets for ablation may be limited by anatomy and by surgically placed obstacles. We report a case in which bidirectional CTI block achieved under computed tomography (CT) and electro-anatomic mapping (EAM) system guidance terminated the tachycardia.

Case Report

A-16-year old boy with a history of surgical palliation of d-transposition of the great arteries, a normal systolic ejection fraction, and symptomatic drug refractory atrial flutter was referred for an electrophysiological study and ablation procedure. At the age of 3 months he had undergone a Senning (atrial switch) operation. A baffle was surgically constructed within atria for directing systemic venous blood across the mitral valve into the left ventricle (systemic venous ventricle) and

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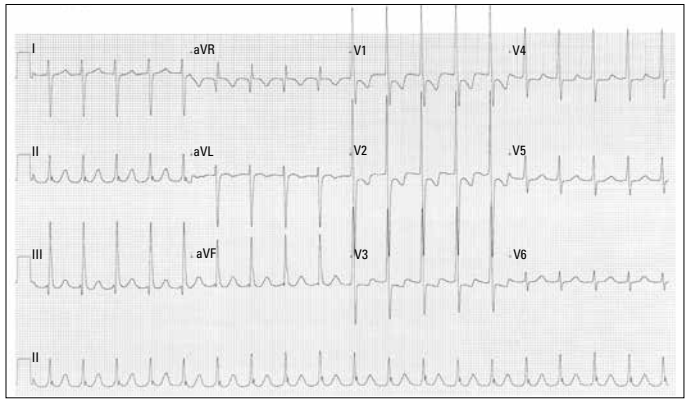

Figure 1. A 12-lead ECG recorded during atrial flutter
ECG - electrocardiogram

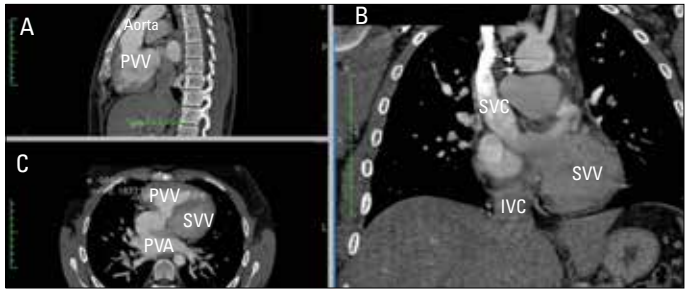

Figure 2. Contrast enhanced computed tomography. A) sagittal axis section shows that aorta originates from the right ventricle (pulmonary venous ventricle). B) coronal axis section shows the baffle connecting inferior and superior vena cava to the left ventricle via mitral valve. C) transverse axis section the baffle connecting pulmonary venous atria to the left ventricle via tricuspid valve
IVC - inferior vena cava, PVA - pulmonary venous atria, PVV - pulmonary venous ventricle, SVC - superior vena cava, SVV - systemic venous ventricle

the pulmonary artery, and pulmonary venous blood across the tricuspid valve into right ventricle (pulmonary venous ventricle) and the aorta. His electrocardiogram (ECG) showed atrial flutter with a ventricular rate of 142 beats per minute (Fig. 1). A transesophageal echocardiography was performed to rule out intra-cardiac thrombus. He underwent a contrast-enhanced computed tomography for detailed anatomic evaluation (Fig. 2).

The procedure was performed under the guidance of the CARTO-XP electroanatomical mapping system (Biosense Webster, Inc., CA, USA). A mapping catheter was placed in the venous baffle and the other in the apex of the systemic ventricle. An atrial flutter with a 214 ms cycle length and 2:1 AV conduction ratio was recorded. The ablation catheter was advanced to the aorta and the pulmonary venous ventricle via femoral arterial access to the pulmonary venous atrium in a retrograde fashion. Electroanatomic maps of the pulmonary venous atrium and systemic venous atrium were created. Voltage map showed large scar area in the pulmonary venous atrium (Fig. 3A and B). Entrainment mapping demonstrated that flutter circuit was CTI dependent. A line of double potentials was found along the septal systemic venous atrium into the baffle. Both ablation lesions were created via transaortic access to the tricuspid annulus and zone between inferior vena cava and the baffle via femoral venous access, which terminated the tachycardia (Fig. 3C). Pacing maneuvers were confirmed bidirectional isthmus block. At the end of the procedure there was no inducible atrial flutter. Echocardiography performed immediately and 1 day after the procedure demonstrated no pericardial effusion. No tachycardias were documented during 2 days of continuous in- hospital ECG monitoring. During a month follow up, he remained in sinus rhythm.

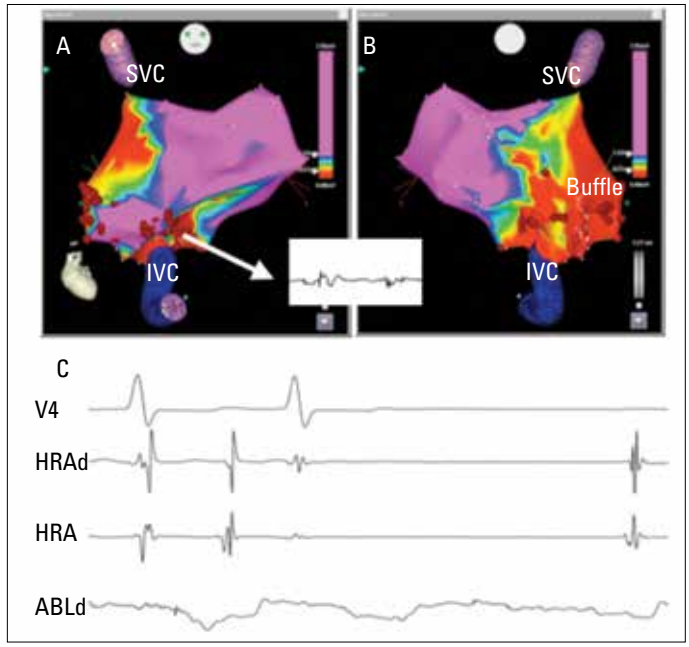

Figure 3. Voltage maps. A-antero-posterior view, B-postero-anterorior view. Arrow shows the point that double potentials were recorded on CTI region. Red area represents scar (<0.5mV), purple area represents healthy tissue (>1.5mV). Red dots represent ablation lines. C-RF energy application terminates the tachycardia
CTI - cavotricuspid isthmus, IVC - inferior vena cava, RF - radiofrequency ablation, SVC - superior vena cava

Discussion

Atrial flutter is common in patients with congenital heart disease, especially in patients with transposition of the great arteries who had undergone palliative atrial switch operations. Most of these tachyarrhythmias are CTI dependent, but the critical zones of slow conduction between suture line and superior vena caval orifice, mitral valve annulus, and pulmonary vein orifice have all been described. Focal atrial tachycardias adjacent to suture lines are also common (1). Cavotricuspid isthmus ablation is a therapeutic option in CTI dependent flutters (2). But it can be limited because of complex cardiac anatomy and large scars due to previous surgery. In the atrial switch population, the coronary sinus (Cs) ostium (os) and tricuspid valve annulus are often on the pulmonary venous atrial side, rather than the systemic venous atrial side. Catheter access to this area necessitates a difficult retrograde approach from the aorta to the right ventricle and then posterior toward the tricuspid annulus and CS os. In our case, after the failure of several ablation attempts from systemic venous atrial side, we decided to reach to the CTI by aortic retrograde access. Bernhardzrenner et al. (3) reported a case in which for successful ablation creation of the ablation lesions to both sides of the baffle line, and creation of bidirectional block were necessary. We also paced the patient from different sides to confirm bidirectional isthmus conduction block. However in some patients, ablating from both sides of the atria cannot eliminate the flutter, and additional ablation lines via transbaffle puncture may be needed (4).

Such procedures may be challenging due to the complex cardiac anatomy and accurate identification of key anatomic locations and landmarks are important for successful ablation. Merging the anatomic data and electrophysiologic data may increase success rates.

Conclusion

In our case both contrast-enhanced computed tomography and electroanatomical mapping system were used for guiding the procedure. Voltage mapping demonstrated a large scar area adjacent to the baffle. Fragmented and double potentials were recorded, indicating slow conduction zones. To achieve CTI block application of the RF energy from the both sides of the baffle was used. Our case demonstrates RF ablation of CTI is a safe and effective therapeutic modality of drug refractory atrial flutters in patients with Senning operation.

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PAİ-1 4G/4G polimorfizmi olan genç bir hastada tekrarlayan miyokart enfarktüsü

Recurrent myocardial infarction in a young patient with PAI-1 4G/4G mutation

Giriş

Kardiyovasküler hastalıkların etiyolojisi multifaktöriyeldir. Geleneksel risk faktörlerinin yanı sıra aterosklerozun moleküler genetiğinin araştırılması, kardiyovasküler hastalıklar için genetik risk faktörlerinin belirlenmesinde önemlidir. Plazminojen aktivatör inhibitör-1 (PAI-1) polimorfizmlerinden 4G allelini içerenler kalıtsal olarak iletilebilen kardiyovasküler risk faktörü olarak bildirilmektedir, plazma PAI-1 seviyesinde ve aktivitesinde artışa neden olarak tromboz riskini artırırlar (1). Burada akut inferiyor miyokart enfarktüsü (ME) tablosu ile başvurup PAI-1 4G/4G polimorfizmi saptanan otuz bir yaşındaki hasta sunulacaktır.

Olgu Sunumu

Otuz bir yaşındaki erkek hasta sabah saatlerinde başlayan iki saattir devam eden yanıcı göğüs ağrısı nedeniyle hastanemize başvurdu. Koroner arter hastalığı için sigara dışında geleneksel risk faktörü yoktu. Öyküsünden altı yıl önce yine kış mevsiminde olmak üzere ME nedeniyle stent takıldığı, ancak medikal tedavisini bıraktığı öğrenildi. Vital bulguları stabil olan hastaya, elektrokardiyografisinde D2-D3-aVF'te ST segment elevasyonu, V1-4 patolojik Q dalgaları olması üzerine akut inferiyor ME tanısı konuldu. Aspirin 300 mg, klopidogrel 600 mg verilerek kateter laboratuvarına alındı. Yapılan koroner anjiyografide (KAG) sol ön inen arter (LAD) proksimalde stent içi trombüs imajı; sirkumfleks arter (CX) plaklı ve sağ koroner arterde (RCA) proksimalde %70 darlığa yol açan trombüslü lezyon saptandı (Video 1, 2.). Göğüs ağrısı ve ST segment elevasyonu devam eden hasta trombolitik tedavi verilmesine karar verilerek koroner yoğun bakım ünitesine alındı. Doku plazminojen aktivatörü (tPA) intravenöz infüzyonu ile birlikte oral metoprolol 50 mg 1x1, ramipril 10 mg 1x1, atorvastatin 40 mg 1x1 ve subkütan enoksaparin 2x 0,6 mL başlandı, aspirin 300 mg 1x1 ve klopidogrel 75 mg 1x1 tedavisine devam edildi. Troponin I değeri 50 ng/mL'ye kadar yükseldi. Hemogram parametreleri ve ortalama platelet hacmi değeri normal sınırlarda saptandı. Yirmi dört saat sonra yapılan kontrol KAG'de trombüslü lezyonların gerilediği saptandı. İkinci günde yapılan transtorasik ekokardiyografisinde sol ventrikül anteriyor duvar ve apikal segmentleri hipokinetik olup ejeksiyon fraksiyonu %40 saptandı. Hastanemizde tromboza eğilimle ilişkili olarak bakılabilen parametreler (antikardiyolipin antikor, antitrombin, protein C ve protein S, homosistein, lupus antikoagülan, faktör 5 Leiden mutasyonu) normal saptandı. Trombofilik faktörlerden protrombin G20210A, MTHFR gen mutasyonları ve t-PA Alu repeat insersiyon/delesyon (I/D) polimorfizmi kurumumuzda bakılamadığından değerlendirilemedi. Hastanın genç yaşta olması ve KAG'de yaygın trombüslü lezyonları olması nedeniyle birinci haftada dış merkezde yapılan genetik incelemesinde PAI-1 4G/4G mutasyonu olduğu tespit edildi. Hastada genç yaşta tekrarlayan ME gelişiminin PAI-1 4G/4G mutasyonu ile ilişkili olabileceği düşünüldü.

Tartışma

Plazmin fibrinolitik sistem ve fibrin pıhtısının eritilmesinde esas enzimdir. Fibrin ağının son yıkımı plazmin tarafından gerçekleştirilir. Plazminojenin plazmine dönüşümü t-PA ile katalize edilir ve PAI-1 ile inhibe edilir (2).

PAI-1 seviyesi sirkadiyen değişim gösterir. Mevsimsel olarak kışın daha yüksek, yazın daha düşüktür, sabahın erken saatlerinde akşamüstü saatlerine göre daha yüksektir. Huber ve ark. (3) tarafından yapılan bir araştırmada, PAI-1 seviyesinin yüksek olduğu durumlarda, PAI-1'e duyarlı doğal veya mutant t-PA'lar hızla inaktive olarak daha düşük etkinlik gösterebileceğinden, eğer trombolitik tedavi uygulanacaksa tenekteplaz gibi PAI-1'e daha dirençli trombolitiklerin tercih edilmesi önerilmiştir. Plazma PAI-1 seviyelerinin sabah saatlerinde pik yapması, akut ME ve iskemik inmelerin sabah saatlerinde daha sık olmasını ve tPA veya ürokinaz ile trombolitik tedavinin sabah saatlerinde etkinliğinin daha az olmasını açıklayabilir (3, 4). Burada sunduğumuz olgunun geçirdiği her iki miyokart enfarktüsünün de kış aylarında ve sabah saatlerinde olması da PAI-1 düzeyinin sirkadiyen değişimi ile ilgili olabilir.

PAI-1 geni çeşitli polimorfik lokuslara sahiptir. Promotorun 675. pozisyonuna lokalize 4G/4G, 4G/5G ve 5G/5G insersiyon/delesyon polimorfizmleri en çok araştırılanlarıdır. Bunlar 4 veya 5 guanin nükleotid dizisine neden olur (4G veya 5G), ortaya çıkan farklı alleller PAI-1 ifadelenmesinde değişikliklere neden olur (5).