Niño de 8 años con episodios de TV - 2002

Dr. Oscar Vagnola

Estimados colegas .

Soy cardiólogo pediatra y quiero participarlos de este caso, raro e interesante.

Niño de 8 años, 30 kg, sano (solo a los 4 meses de vida tiene un ECG con extrasístoles supraventriculares aisladas) y hace 1 mes me consultan por lipotimia en tres oportunidades (stress), las dos primeras al asustarse después de ver a un perro agresivo.

La madre refiere se asusta, se pone pálido, pierde el conocimiento y en la segunda presenta un episodio tipo convulsivo de corta duración. La tercera lipotimia la tiene jugando al fútbol, y lo veo a los 3 días,

Examen CV soplo inocente, resto normal. y al ECG frecuentes extrasístoles ventriculares anchas de VD con FC basal de 100. ondas P retroconducidas. (sin WPW)

Ecocardio normal. Holter, casi permanentemente con ritmo ventricular, con taquicardias ventriculares mientras corre con FC 180 l/m y por momentos tiene también ritmo sinusal y tiene taquicardia sinusal. Lo consulté con un colega de Neuquén M.C. y pensó en una taquicardia ventricular. verapamilo sensible. Lo medique con isoptino (90 mg/d), y a los 5 días (siguió refiriendo palpitaciones) le hago un ECG y casi era todo TV, lo suspendí y comencé con Nadolol a 80 mg/d, a las 48 hs había bajado la FC basal pero ante leves esfuerzos el ritmo era ventricular a 100l/m. Aumento a 100mg, mejora, no refiere más palpitaciones, no se marea al jugar al fútbol, al mes le hago otro Holter y tiene 2/3 del día ritmo ventricular, con aislados episodios de ritmo sinusal, pero cundo la FC supera los 106 l/m el ritmo es sinusal sin aparecer ni siquiera una fusión .

Le aumentaré la dosis a 120 mg (4mg/k/d) ya que la mínima FC es de 55-58.

¿Ustedes qué piensan? ¿qué harían? ¿cómo lo encararían?, ¿qué le agrego si no mejora , yo pienso en la amiodarona?

Saluda cordialmente, y el foro es interesante aún para un cardiólogo pediatra.

Dr. Vagnola E. Oscar.

Cipolletti .Rio Negro.

OPINIONES DE COLEGAS

Yo tuve una vez un caso similar. En sus exámenes de rutina detecté G. lamblia en heces, que al parecer ejerce una función cardiotóxica en los niños.

Después de haber tenido poco éxito previo con la amiodarona, traté sgiardiasis. Nunca más presentó extrasístoles ventriculares.

Saludos.

Dr. Luciano Pereira

Estimado Dr Vagnola:

¿Sería posible que escanearas algunos ejemplos de los ECGs de tu paciente para poder analizarlos?

¿Cuánto mide el intervalo QT?

Dr Serra Jose Luis

Cordoba.

¿Cual es el mecanismo "cardiotoxico" de la giardiasis?

Darío A Gómez

Amigos del foro:

No soy arritmoóogo. Es más, siempre les tuve respeto, quizás producto de mi ignorancia sobre ellas. Pero quiero emitir una humilde opinión. Lo que plantea nuestro amigo Luciano Pereira es verdad, pero esto no es privativo de la giardiasis, también se observa en otras parasitosis. Tampoco sería cardiotoxicidad, sino más bien neurotoxicidad, que podrían explicar en parte los trastornos que presentan durante el sueño los niños parasitados. Nunca encontré una explicación para esto, ni en coloquios con parasitólogos ni en publicaciones. Probablemente, los expertos y eruditos que están en la red, como nuestro amigo Pérez Riera de Brasil, puedan opinar al respecto.

Pero quiero aclarar también que todos los casos que vi, que no son pocos, siempre fueron extrasístoles aisladas y polifocales; pero nunca duplas, ni colgajos ni mucho menos taquicardia ventricular como el caso que Oscar nos comenta. Yo vuelvo a repetir, no se de arritmias y ante un caso como este, por temor o por ignorancia yo indicaría un antiarritmico:

CONSULTA CON ELECTROFISIOLOGO PEDIATRA. Afectuosos saludos.

Raul O. Cayre

Resistencia, Chaco

Hola, Dr. Gómez. Desconozco el mecanismo de la cardiotoxicidad, pero obedecería a mecanismos inmunológicos. Se sabe que la producción de inmunoglobulinas IgA e IgE por las células inmunocompetentes es afectadas en la giardiasis. Un trabajo citado más abajo, inclusive menciona que el óxido nítrico sería el elemento que protegería al huésped contra la infección. El hecho de que esta parasitosis tan común afecte más a los niños (en quienes los síntomas son más evidentes) se debe justamente a mecanismos inmunológicos. Estuve realizando una búsqueda en MEDLINE y aún cuando no haya encontrado algo más específico te envío estas citas. Los electrocardiogramas del paciente al cual hice referencia los mostré el año

pasado al Dr. Raúl Cayré quien me sugirió que hiciera un examen parasitológico al niño (Un chinito de 11 años que tenía extrasístoles ventriculares frecuentes habituales, a veces con salvas de taquicardia ventricular. Sólo que esta arritmia en este paciente era frecuencia-dependiente, es decir mejoraba con el ejercicio, a diferencia del paciente presentado). Yo lo había estado tratando con amiodarona con mejoría relativa. Detecté giardiasis y lo traté con albendazol, no volviendo a presentar arritmias hasta la fecha. Tal vez debamos preguntarle al Dr. Cayré la referencia, no?

Abrazos cordiales.

Dr. Luciano Pereira

Aquí van algunas citas:

Faubert G: Clin Microbiol Rev 2000 Jan;13(1):35-54 Immune response to Giardia duodenalis.

J Immunol 2000 Feb 1;164(3):1478-87 Nitric oxide production by human intestinal epithelial cells and competition for arginine as potential determinants of host defense against the lumen-dwelling pathogen Giardia lamblia.

Eckmann L, Laurent F, Langford TD, Hetsko ML, Smith JR, Kagnoff MF, Gillin

FD.: J Trop Pediatr 1998 Aug;44(4):241-6

Immunologic response to infection with Giardia lamblia in children: effect of different clinical settings.

Granot E, Spira DT, Fraser D, Deckelbaum RJ. Arch Med Res 1994 Summer;25(2):171-7

Evaluation of the immune response in symptomatic and asymptomatic human giardiasis.

Perez O, Lastre M, Bandera F, Diaz M, Domenech I, Fagundo R, Torres D,

Finlay C, Campa C, Sierra G.

Estimado Dr. Vagnola:

Creo que vale la pena estudiar el caso que presenta desde el punto de vista etiológico pues de eso dependerá el tratamiento y el pronóstico. ¿Se descartó razonablemente miocarditis?, alguien más ya comentó que parasitosis intestinales pueden asociarse a arritmias graves, pero no sólo hay que descartar parásitos, si no otros tipos de infección. Creo que un ECO podría ser útil. ¿el VD se encuentra dilatado?,¿qué morfología tiene la

taquicardia? ¿es del tracto de salida del VD o se origina del ápex?, todo esto es útil para valorar otras causas, también ya comentadas en el foro. Si bien es importante suprimir por ahora la arritmia, la etiología nos dará el pronóstico y el mejor tratamiento, desde prescripción de ejercicio, valoración de cardioverter o incluso, si es de tipo infeccioso, curación en unas cuantas semanas.

Dra Argelia Medeiros

México.

Luciano

Yo no recordaba este caso que me mostraste. Seria el primero para mi con giardiasis que tiene extrasístoles ventriculares frecuentes y colgajos de taquicardia ventricular. No tengo citas bibliográficas especificas. Si encuentro alguna, te la hare llegar.

Saludos.

R. Cayre.

Prezado Oscar: tenta te dar um norte na orientação de teu pacientinho Andrés Ricardo Pérez Riera de SP Brasil.

Infelizmente como o pede o colega Serra seria importante nos termos acesso ao ECG. Dessa forma poderiamos falar com mais propriedade do caso, porém, na suposição que tua interpretação foi correta, isto é, que se trata mesmo de uma taquicardia ventricular (TV) e não de uma supra com aberrância. Também partirei do presuposto que se trata de uma TV monomórfica (TVM) e não de uma polimórfica porque se nao penso que o terias esclarecido.

As TV monomórficas idiopáticas são aquelas, nas quais não é possível detectar com os métodos convencionais não invasivos e invasivos um substrato anatômico responsável. Por tanto acredito pela descrição que estamos perante esta variante: TVM IDIOPÁTICA ou TVMI.

Nestas variedades na maioria dos casos, não existe qualquer substrato anatômico ou patologia demonstrável, porém, recentes estudos com biópsia endomiocárdica contrariam este conceito, tendo demonstrado anormalidades em mais do 65% dos casos, que aumenta para mais de 80% quando o material é fruto de autópsia. Assim, já foram referidos:

1) hamartomas das células de Purkinje (Garson Jr, A. et. al. J. Am. Coll. Cardiol. 10:619, 1987).

2) forma frustra ou leve de displasia arritmogência do ventrículo direito.

3) microangiopatia associada a fibrose do subendocárdio.

4) miocardite sub-clínica.

5) miocardiopatia focal.

6) miocardopatia isquêmica ateroesclerótica.

7) miocardiopatia isquêmica não-ateroesclerótica.

8) miocardiopatia hipertrófica.

9) Prolapso mitral.

(Wichter, T.; Breithardt, G.; Borggrefe, M.: Ventricular tachycardia in the normal heart. In: Podrid, P. J..: Kowey, P. R. eds. Cardiac Arrhytmia_Mechanism, Diagnosis, and Treatment. Baltimore: Williams &Wilkins, pp.1219-38, 1995).

Se há identificado o gen GNA12 no locus 3-p21.

As TVMI hoje são divididas em 4 grupos bem definidos:

1) TVMI, catecolamino-sensitiva ou adenosina-sensitiva ou por atividade gatilhada "triggered activity": 70%.

2) TVMI verapamil-sensitiva ou por reentrada intrafascicular com QRS estreito

3)) TVI propranolol sensitiva automática

4) TVMI indiferenciada.

Pelos fatos referidos todo me leva a pensar que teu pequeno paciente está acometido da primeira variedade isto é TVMI, catecolamino-sensitiva, adenosina-sensitiva ou por atividade gatilhada ("triggered activity") 70%. encontrada em pacientes sem cardiopatia estrutural demonstrável, com foco de origem predominante na via de saída do ventrículo direito (90%) e morfologia de BIRE ou BCRE com ÂQRS desviado a direita ou normal (de + 300 a + 1200). Em 10% dos casos pode se originar do VE na região da divisão póstero-inferior com morfologia de BRD e extremo desvio do ÂQRS para esquerda.

Outra morfologia que sugere foco de origem no VE é BRE associado a precoce transição na derivação V2. Onda R dominante em V1 e ÂQRS inferior assinala foco de origem superior no VE. Raramente pode ser de origem epicárdica caracterizada por concordância positiva nas precordiais e complexos negativos em DI e aVL. Existe descrição de literatura de TV com ambas morfologias de BRE e BRD cada uma com ÂQRS idéntico o que sugere que o foco se origina no septo interventricular com dupla saída para esquerda ou para a direita.

Mecanismo eletrofisiológico responsável: Atividade deflagrada tardia dependente de pós-despolarizações em fase 4 associada a aumento de AMP cíclico e mediada por catecolaminas: adrenérgico-dependentes. Podem ocorrer com automatismo normal ou anormal.

Eletrocardiograma da TVMI

1) Patente de BIRE ou BCRE com ÂQRS desviado a direita ou normal entre + 300 e + 1200: complexos positivos nas inferiores indicando sua origem na via de saída do ventrículo direito. Raramente o eixo está desviado a esquerda indicando origem na parede livre do ventrículo direito.

2) Caráter repetitivo monomórfico ou TV-S e paroxístico.

3) Invariável reversão com adenosina: adenosino sensitiva: Este fato pode dar a falsa impressão de tratar-se de uma taquicardia supraventricular com condução aberrante, uma vez que esta droga atua apenas no nó sinusal, aurículas e nó AV sem efeito direto no miocárdio ventricular. Mesmo assim, os efeitos da droga podem ser demonstrados durante a estimulação adrenérgica sobre o sistema adenilciclase /cAMP (Bellardinelli, L. e col. in Zipes, D. P.; Jalife, J.; eds. Cardiac Electrophysiology, from cell to bedside.Philadelphia:WB Saunders, 284:1990).

4) Podem ser desencadeadas pela infusão de isoproterenol.

5) Maior frequencia durante a vigília e desencadeada por esforços: adrenérgico-dependentes.

6) Presença no traçado fora das crises de extra-sístoles muito frequentes em salva e monomórficas (comportamento tipo Galvardin). O caráter monomórfico tem valor para o

diagnóstico diferencial com a displasia aritmogênica do ventrículo direito que mostra extra-sístoles polimórficas.

O ECG pode permitir diferenciarla da displasia arritmogenica do VD. Esta entidade, apresenta caraterística onda T negativa de V1 a V4 em menor de 12 anos na ausência de bloqueio de ramo direito e eventual presença de onda épsilon (30% dos casos) localizadas no fim do QRS e inicio do ST (ponto J) ostensíveis de V1 a V3 fato que a TVMI não possue no ECG de base.

Teste ergométrico: Em 25% a 50% dos casos pode desencadear as crises de TV: adrenérgico-dependentes ou esforço induzidas. A iniciaçao do evento tanto pode ocorrer no esforço quanto na fase de recuperação assinalando uma "janela" de FC na qual ocorre o evento.em outras palavras há uma FC sinusal crítica onde ocorre o evento. Um outro elemento de confirmação diagnóstica é a demonstração de dependência do comprimento do ciclo o qual define-se como uma janela ou rango de cumprimento do ciclo do pacing que induz a TV.

Holter/24h: Extra-sístoles ventriculares monomórficas em salvas frequentes o eu bigeminadas. Eventual registro de longos periodos de TV.

ECGAR: o ECG de alta ressolução é de grande importancia para diferencia-la da displasia arritmogênica do VD porque em esta em mais de 80% registrade potenciais tardios no final do QRS filtrado.

Entrainment: negativo. Esta técnica consistente na aplicação de extra-estímulos em série durante a TV-S utilizando-se ciclos pelo menos 20ms menor que o ciclo da TV acelerando a taquicardia para a frequencia estimulada. A presença de entrainment indica que o mecanismo é a reentrada. A TVMI é atomatica.

A ressonancia nuclar tambem pode servir para afastar displasia porque esta tem

a) brilho intenso na parede ventricular direita pela substituição das células miocárdicas (áreas de alta densidade) por tecido fibroadiposo no triângulo da displasia: infundíbulo, região apical e póstero-diafragmática.

b) dilatação ventricular direita e crescimento atrial direito.

c) ectasia na via de saída do ventrículo direito.

d) imágens discinéticas do ventrículo direito.

e) possibilidade de detectar os casos com acometimento do ventrículo esquerdo.

Que fariamos no teu caso:

1) precisar o diagnóstico principalmente com adenosina.

Uma vez que se confirme que se trata desta entidade: me parece que teu caso é para radiofrequencia Por que?

Resposta: Nos casos refratários, com sincope ou pré-síncope associados a TV ou se as freqüentes extra-sistoles são debilitantes a escolha recaerá sobre este método, que procurará ablacionar o foco de origem. O importante é localizar o local de ativação mais precoce realizando o "pace mapping" reproduzindo a configuração da TV no ECG o que permite escolher o local para a emissão da radiofrequência. Pode ser de ayuda para predizer a localização do foco: Uma transição da onda R em V2 durante a TV assinala sua origem imediatamente por baixo do valva pulmonar.

Complicações:

1) BCRD: 2%

2) Perfuração da via de saída e óbito.

3) Obstrução do tronco da coronária esquerda quando a abação é feita justo baixo a valva aórtica.

2) TVMI verapamil-sensitiva ou por reentrada intrafascicular com QRS estreito

Denomina-se assim aquela TVMI originada no sistema de condução ventricular alto: ramo direito (morfologia de BIRE), esquerdo ou nas divisões deste último (morfologia de BIRD) com QRS que não atinge uma duração de 120ms e freqüência não muito elevada (entre 100bpm e 240bpm).

Critérios eletrocardiográficos

1) A freqüência da TV pode oscilar entre 100 e 240bpm, sendo o contingente predominante entre 130bpm e 160bpm.

2) duração do QRS até 110ms: TV com QRS estreito.

3) eventual presença de batimentos de fusão.

4) evidências de dissociação A-V.

5) freqüente alteração da repolarização ventricular no ECG de base na parede ínfero-lateral após a crise prolongada de TV. Explica-se pelo assim chamado fenômeno da memória cardíaca, o qual enuncia que acontecerão alterações de repolarização no local onde se originou quando o evento for prolongado.

Patentes eletrocardiográficas: descrevem-se três variantes na dependência do local de origem:

a) Com morfologia de BIRD: com patente qR ou R em V1-V2 apresenta duas variantes conhecidas:

b) morfologia de BIRD com extremo desvio do ÂQRS a esquerda no plano frontal entre -300 e - 600: quando originada na divisão póstero-inferior do ramo esquerdo: 90% dos casos.

6) Durante a TV registra-se S com entalhe (empastada) na rampa ascendente nas derivações inferiores.

a) Com morfologia de BIRD com desvio do ÂQRS a direita entre + 600 e + 1200 quando originada na divisão ântero-superior do ramo esquerdo: 10% dos casos.

b) Com morfologia de BIRE: originada no ramo direito, na via saída do VE. Eletrocardiograficamente confunde-se com a TV idiopática da via de saída do VD.

Refere-se que a única diferença é a presença de R proeminente em V1 em nosso caso (Darrieux, F. & Scanavacca, M. Taquicardias ventriculares idiopáticas. Rev. Soc. Cardiol. Estado de SP, V8; N01:137-44,1998).

ECGAR: normal.

Ecocardiograma: se há sugerido associada a tendão fibro-muscular ínfero-apical.

Holter/24h: nada característico fora dos eventos de TV.

De escolha o verapamil, o qual na grande maioria dos casos termina o evento e inibe sua inductibilidade além de ocasionar prevenção das recorrências: TV verapamil sensitivas.

A droga administra-se endovenosa rápido 5 a 10mg (0.145mg/Kg) em 10 a 15 segundos (bolo). Na ausência de resposta, pode-se administrar uma segunda dose após 30 minutos.

Em idosos, não ultrapassar a dose de 5mg para evitar hipotensão.

Adenosina: indicada nos casos resistentes ao verapamil.

Posologia: 6mg (uma ampola) em bolo rápido com catéter central grosso. Se não se houver resposta em 2 minutos pode se fazer um bolo de 12mg EV rápido.

Em pacientes transplantados, em uso de dipiridamol ou xantinas deve iniciar-se com bolo de 3mg.

3)TVM idiopática Propranolol sensitiva automática

Caracterização: induzida pelo excercício.ou incesante. Inducção: por catecolaminas.

Morfologia: BRD, BRE ou polimórfica.

Origem: VE ou VD

Entrainment: negativo.

Mecanismo: enhanced automaticidade.

Propranolol, adenosina: terminam o evento/ ou ocasionam supressão transitória

Verapamil: não atua.

4) TVM Indiferenciada

Caracterização: induzida pelo excercício.

Inducção: por catecolaminas. E eventualmente por estimulação venricular programada.

Morfologia: BRE com ÂQRS inferior.

Origem: via de saída do VD.

Entrainment: positivo.

Mecanismo: reentrada.

Propranolol, adenosina e verapamil : não atuam.

Te envio recentes referencias sobre a conduta que te recomendei no caso para atualizacao sua e do foro.

Andrés.

van der Burg AE, de Groot NM, van Erven L, Bootsma M, van der Wall EE, Schalij MJ. Long-term follow-up after radiofrequency catheter ablation of ventricular tachycardia: a successful approach? J Cardiovasc Electrophysiol 2002 May;13(5):417-23

Department of Cardiology, Leiden University Medical Center, The Netherlands.

INTRODUCTION: Radiofrequency ablation (RFCA) of ventricular tachycardia (VT) is a potential curative treatment modality. We evaluated the results of RFCA in patients with VT. METHODS AND RESULTS: One hundred fifty-one consecutive patients (122 men and 29 women; age 57 +/- 16 years) with drug-refractory VT were treated. Underlying heart disease was ischemic heart disease in 89 (59%), arrhythmogenic right ventricular cardiomyopathy (ARVC) in 32 (21%), and idiopathic VT in 30 (20%; left ventricle in 9 [30%]; right ventricle in 21 [70%]). Ablation was performed using standard ablation techniques. Three hundred six different VTs were treated (cycle length 334 +/- 87 msec,

2.0 +/- 1.4 VTs per patient). Procedural success (noninducibility of VT after RFCA) was achieved in 126 (83%) patients (70 ischemic heart disease [79%]; 28 ARVC [88%]; 27 idiopathic VT [93%]). Procedure-related complications (< 48 hours) occurred in 11 (7%) patients: death 3 (2.0%), cerebrovascular accident 2 (1.3%), complete heart block 4 (2.6%), and pericardial effusion 3 (2.0%). Thirty-three (22%) patients received an implantable cardioverter defibrillator (because of hemodynamic unstable VT, failure of the procedure, or abor

Adragao P, Parreira L, Morgado F, Almeida M, Mesquita A, Machado FP, Martins D, Bonhorst D, Seabra-Gomes R. [The radiofrequency catheter ablation of ventricular tachycardia] Rev Port Cardiol 1996 Feb;15(2):119-28, 100

[Article in Portuguese]

Departamento de Arritmologia, Hospital de Santa Cruz.

OBJECTIVE: The aim of this study was to evaluate our results of radiofrequency catheter ablation (RFCA) of ventricular tachycardia. PATIENT SELECTION: We treated with RFCA nine patients, six male and three female, mean age 36 +/- 12 years with ventricular tachycardia (VT), who fulfilled the following criteria; 1) recurrent VT; 2) resistant fo medical therapy despite the use of more than one antiarrhythmic drug; 3) inducible by programmed ventricular stimulation; 4) hemodynamically well tolerated. The VT etiology was coronary artery disease (CAD) in three patients, dilated cardiomyopathy in one, right ventricular dysplasia in one and it was idiopathic in four (being fascicular in three and catecholaminergic right ventricular outflow tract VT in one). METHODS: The RFCA was performed under antiarrhythmic medication. The adequate ablation site was obtained by mapping of the VT, looking for the earliest ventricular activation, identification of isolated mid-diastolic potentials during sinus rhythm or presystolic during VT, good pace mapping (at least 10 of the 12 standard ECG leads), and high frequency short duration spikes, the so called P potentials in fascicular VT. Primary success achieved when occurred termination of VT during application of RF energy and/or VT was no longer inducible by programmed stimulation with the same stimulation protocol. RESULTS: Global primary success rate was 89%, being 100% in idiopathic VT, and 80% in VT associated with structural heart disease. In a follow-up period of 12 +/- 14 months all patients were alive, 75% free of VT in the idiopathic VT group; and 50% in patients with structural heart disease. One of these patients underwent cardioverter defibrillator implantation to treat a fast VT with a new morphology not treated by ablation, and the other two had VT modification with a significant reduction in the number of episodes. CONCLUSIONS: Radiofrequency catheter ablation of VT has shown a good success rate, and it is a valuable alternative in patients with hemodynamically tolerable VT, refractory to drug treatment, highly symptomatic and without surgical indication. In cases of idiopathic VT we

had a high rate success and we think that RFCA will probably become the primary indication in symptomatic patients

ted sudden death). During follow-up (34 +/- 11 months), VT recurrences occurred in 38 (26%) patients (recurrence rate: 19% in successfully ablated patients and 64% in nonsuccessfully ablated patients; P < 0.001). During follow-up, 12 (8%) patients died (heart failure 8, unknown cause 1, noncardiac cause 3). CONCLUSION: RFCA of VT can be performed with a high degree of success (83%). The long-term outcome of successfully ablated patients is promising, with a 75% relative risk reduction compared with nonsuccessfully ablated patients. During follow-up, only one patient died suddenly, supporting a selective ICD placement approach in patients with hemodynamically stable VT.

Petrac D, Radic B, Vukosavic D, Birtic K. Late clinical outcome after radiofrequency catheter ablation of idiopathic ventricular tachycardia: follow-up study. Croat Med J 2002 Feb;43(1):20-4

Division of Cardia Arrhytmias and Cardiac Pacing, Department of Medicine, Sisters of Mercy Hospital, Zagreb, Croatia, **[conectar para ver]**

AIM: To prospectively evaluate long-term clinical outcome in patients who underwent radiofrequency catheter ablation for the treatment of idiopathic ventricular tachycardia (VT). METHODS: Twenty consecutive patients with idiopathic VT resistant to drugs were treated by temperature-controlled radiofrequency ablation. The site of VT origin was localized by pace mapping and endocardial activation mapping during VT. After ablation, each patient was followed up for at least 28 months (median 56, range 28-92). RESULTS: Radiofrequency catheter ablation was successful in 15 out of 17 patients with idiopathic VT originating from the right ventricle, and in all 3 patients with idiopathic VT originating from the left ventricle. The total energy delivered for the elimination of VT was higher in patients with idiopathic right VT than in those with idiopathic left VT (p=0.014). During the follow-up period, clinical VT recurred in 2 patients with unsuccessful ablation. Right and left ventricular ejection fractions did not change and were not significantly different at the end of follow-up period from those determined before ablation. Also, there was no late negative impact of radiofrequency ablation, such as myocardial dysfunction or new VT, in any of the patients. CONCLUSION: Initially successful radiofrequency catheter ablation of idiopathic VT has excellent long-term results without producing late proarrhythmic or cardiodepressant effects. This therapeutical procedure is appropriate in cases of idiopathic VT refractory to antiarrhythmic drugs.

Chinushi M, Aizawa Y, Kusano Y, Washizuka T, Miyajima T, Naitho N, Takahashi K, Shibata A. Radiofrequency current catheter ablation for ventricular tachycardia. Jpn Circ J 1994 May;58(5):315-25

First Department of Internal Medicine, Niigata University School of Medicine, Japan.

Radiofrequency current catheter ablation was attempted for 17 morphologies of ventricular tachycardia (VT) in 14 patients. Five patients had underlying heart disease. The site of VT origin was determined as the earliest site of ventricular activation, or by pacing within the area of slow conduction. In 15 VTs, ablation was performed during VT, and 12 VTs (80%) were terminated within an average of 5.4 +/- 4.2 seconds. After ablation, 14 VTs (14/17 = 82%) of 11 patients (11/14 = 79%) could not be induced by electrical stimulation. Radiofrequency ablation appeared to be more effective in VTs without underlying heart disease (91%), and in VTs originating from the right ventricle (100%). Successful ablation sites usually showed a normal local electrograms during VT. Ablation in the slow conduction area was attempted in 3 VTs, and 2 VTs became noninducible. The mean number of applications of radiofrequency current for each VT origin was 7.7 +/- 6.4 at 20-50 Watts. In 4 patients, application of radiofrequency current was required 10 or more times because of a possible large arrhythmogenic area, or because of reinduction of VT, even though VT was terminated by radiofrequency current. No major complication was observed except for complete right bundle branch block in 1 patient. In conclusion: (1) Radiofrequency catheter ablation was considered to be effective and safe, especially for VT without underlying heart disease or VT originating from the right ventricle. (2) Ablation during VT was considered to be useful for identifying the proper ablation site and to avoid creating an unnecessary lesion.

Raungratanaamporn O, Nutakul T, Chotinaiwattarakul C, Sriyaphai W, Chaithiraphan S, Bhuripanyo K, Mahanonda N, Hongvisitgul C, Kangkagate C. Radiofrequency catheter ablation in symptomatic ventricular arrhythmia. Aust N Z J Med 1997 Aug;27(4):398-402

Her Majesty's Cardiac Centre, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok, Thailand.

BACKGROUND: Radiofrequency catheter ablation (RFCA) is an effective method to cure both supraventricular and ventricular arrhythmia (VA) in certain centres. AIM: To assess the results of RFCA in VA at Siriraj Hospital. METHOD: Electrophysiologic study, mapping, using both earliest endocardial activation and pace mapping, and ablation were performed. RESULTS: Thirty patients with symptomatic VA underwent RFCA. The mean age was 44 years. Eight patients had underlying heart disease (two prolapsed mitral valve, three myocarditis, two dilated cardiomyopathy and one mitral stenosis). Thirty-six morphologies of VA were detected from the study. Thirty-three morphologic tachycardias attempted were successfully ablated; 17, 10 and six were right VT, left VT and premature ventricular contraction (PVC), respectively. Failure of ablation occurred in one patient with left VT. Only minor complications occurred in this study. There was no difference in cycle length

and endocardial activation time between right and left VT. 12/12 identical pace mapping was more easily performed in right VT than in left VT. The fluoroscopic and procedure times in left VT were significantly longer than in right VT. Relapse occurred in six patients. Re-ablation was successfully performed in four patients, giving a final success rate of 93%. CONCLUSION: RFCA is an effective treatment and should be considered as an alternative method to cure VT and refractory PVC.

Cordero Cabra JA, Iturralde Torres P, Lara Vaca S, Colin Lizalde L, Kershenovich S, Carvajal A, Gonzalez Hermosillo JA. [Ablation using radiofrequency in the treatment of ventricular tachycardia]Arch Inst Cardiol Mex 1996 May-Jun;66(3):210-9

[Article in Spanish]

Departamento de Arritmias y Electrocardiografia, Ignacio Chavez, INCICH, Mexico, D.F.

We performed radiofrequency catheter ablation in 14 consecutive patients with Ventricular Tachycardia (VT) 10 of which had healthy hearts, one patient with ischemic heart disease, one with arrhythmogenic dysplasia, one with dilated cardiomyopathy, and one with congenital heart disease. The localization of the VT was: 10 in the left posterior fascicular region, 3 in the right ventricular outflow tract (RVOT), and one patient with ischemic heart disease with the substrate in the left ventricular apex. All of them with VT refractory to pharmacological management, using an average of 2.7 drugs per patient. After all patients underwent Electrophysiological Study (EPS), an intracavitary mapping was performed, in order to locate the arrhythmogenic substrate. Later on, the RF ablation was performed, delivering an average of 15 pulses, using 40 Watts, and an average time of 25 sec. per pulse. The procedure was successful in 60% of the fascicular VT, with a 16% of recurrence; 100% of success with those originated in the RVOT with no recurrence; in the ischemic patient we achieved primary success, but with recurrence, a second session was successful with no recurrence up to date. No major complications occurred in this group. Those patients which showed no success required the use of antiarrhythmic drugs. The total success of the series is 71.4% with 10% recurrence, and no mortality.

Peinado Peinado R, Arenal Maiz A, Almendral Garrote J, Perez Villacastin J, Merino Llorens JL, Martinez-Alday JD, Pastor Fuentes A, Medina Moreno O, Valero Parra R, Delcan Dominguez JL. [Radiofrequency catheter ablation of ventricular tachycardia in patients without apparent structural cardiopathy] Rev Esp Cardiol 1994 Dec;47(12):803-10

[Article in Spanish]

Laboratorio de Electrofisiologia-Clinica Cardiaca, Hospital General Gregorio Maranon, Madrid.

BACKGROUND AND PURPOSE. It has been suggested that the efficacy of radiofrequency ablation of idiopathic ventricular tachycardia (VT) is dependent on the site of VT origin, with the efficacy being greater for VTs originating from right ventricle. The electrophysiologic characteristic and the results of radiofrequency catheter ablation of ventricular tachycardia in patients without structural heart disease are reported. Special emphasis was focused to the differences observed in the pace and activating mapping between VTs originating in the right ventricle and those originating from the left ventricle and its possible implications for radiofrequency efficacy. METHODS AND RESULTS. 14 consecutive patients with idiopathic VT (7 women and 7 men, mean age 35 +/- 16 years), 8 originating in the right ventricle (RV) and 6 in the left ventricle (LTV), underwent catheter ablation using radiofrequency energy. The observation of entrainment with fusion in all LV VT suggested that the electrophysiologic mechanism was a reentry, meanwhile the RV VT were due to focal non-reentrant mechanisms. Sites for radiofrequency energy delivery were selected on the basis of pace and activation mapping in all patients less in two patients with incessant VT in whom only activation mapping was performed. 14 VT were mapped. The activation mapping demonstrated isolated presystolic electrograms in the point of origin in all VT arising from the LV. However in RV tachycardias there was continuous activity between presystolic and systolic electrograms, although the prematurity of these electrograms was similar (31 +/- 16 ms vs 33 +/- 9 ms; p = 0.77). Radiofrequency was successful in eliminating 93% of TV (100% RV TV vs 83% LV TV; p = 0.23). No complications were observed. CONCLUSIONS. The results of this study suggest that radiofrequency ablation is highly successful either in right and left ventricles idiopathic tachycardias when pace and activation mapping are used complementary.

Raungratanaamporn O, Bhuripanyo K, Nutakul T, Mahanonda N, Chotinaiwattarakul C, Hongvisitgul C, Sriyaphai W, Chaithiraphan S. Radiofrequency catheter ablation in monomorphic ventricular tachycardia. J Med Assoc Thai 1996 Jun;79(6):358-64

Department of Anesthesiology, Faculty of Medicine, Siriraj Hospital, Mahidol University, Bangkok, Thailand.

Twenty patients with symptomatic monomorphic ventricular tachycardia (VT) underwent radiofrequency (RF) energy catheter ablation. Four patients (20%) had underlying heart

disease (1 prolapse mitral valve, 1 dilated cardiomyopathy and 2 myocarditis). Five patients (25%) had left sided VT and right sided VT in the remainder (75%). Radiofrequency catheter ablation was initially successful in all patients without major complication. Recurrence occurred in three patients (15%). In conclusion, RF ablation is an effective treatment for symptomatic monomorphic right and left sided VT especially in patients who do not want long term antiarrhythmic agents.

Tsai CF, Chen SA, Tai CT, Chiang CE, Lee SH, Wen ZC, Huang JL, Ding YA, Chang MS. Idiopathic monomorphic ventricular tachycardia: clinical outcome, electrophysiologic characteristics and long-term results of catheter ablation. Int J Cardiol 1997 Nov 20;62(2):143-50

Department of Medicine, National Yang-Ming University, School of Medicine, and Veterans General Hospital-Taipei, Taiwan, ROC.

Ventricular tachycardia (VT) without structural heart disease or any identifiable predisposing causes for arrhythmia is an uncommon but well-recognized clinical entity. The purpose of this study is to assess the results of catheter ablation therapy and the long-term outcome of patients with idiopathic monomorphic VT in a large patient group. Sixty-one consecutive patients (male/female=40/21; mean age 38+/-16 years) with idiopathic VT underwent electrophysiologic study and an attempt of catheter ablation therapy. The 'left VT' group included 31 patients with QRS morphology of right bundle branch block during VT suggestive of the VT originating from the left ventricle (LV), and the 'right VT' group consisted of 30 patients with QRS morphology of left bundle branch block with normal or right frontal axis deviation suggestive of VT arising from right ventricular outflow tract (RVOT). Idiopathic left VT has sustained VT during the clinical attacks, baseline electrophysiologic study or after isoproterenol infusion; it can be entrained by overdrive ventricular pacing, terminated by verapamil, but not by adenosine (except one case with VT focus at left ventricular free wall). Catheter ablation was successful in 22 (84%) of 26 patients, with recurrence rate of 9%. The successful ablation sites were located at LV inferior-apical septum (16 patients), mid-septum (three patients), high septum (two patients) and high anterior wall (one patient). In the right VT group, 20 (67%) of 30 patients presented clinically repetitive monomorphic VT. Most of the idiopathic right VT (22/30) required isoproterenol to facilitate induction of VT, and were sensitive to both verapamil and adenosine. Successful catheter ablation was achieved in 21 (84%) of 25 patients, with

recurrence rate 19%. The successful ablation sites were located at RVOT-septum in 18 patients, and RVOT-free wall in three patients. During a mean follow-up period of 29.2+/- 21.7 months (range 1-76 months) after hospital discharge, all patients were alive but one left VT case died of non-cardiovascular cause. We concluded that idiopathic left side and right side VTs have their distinct clinical, electrophysiologic and electropharmacological characteristics suggestive of different underlying mechanisms, and both have a benign prognosis. Furthermore, catheter ablation can be effective in eliminating idiopathic VT originating from the right ventricular outflow tract and left ventricle.

Jadonath RL, Snow JS, Goldner BG, Cohen TJ. Radiofrequency catheter ablation as primary therapy for symptomatic ventricular tachycardia. J Invasive Cardiol 1994 Nov-Dec;6(9):289-95

Department of Medicine, Electrophysiology Section, North Shore University Hospital, Cornell University Medical College, Manhasset, New York 11030, USA.

Most applications of radiofrequency (RF) catheter ablation for treatment of ventricular tachycardia (VT) have been as a treatment of last resort. The purpose of this study was to determine the efficacy and safety of RF catheter ablation as the primary treatment for symptomatic single morphology VT. Eleven of 81 patients (14%) with inducible sustained monomorphic VT underwent RF ablation as the primary treatment. One of these 11 patients had successful RF ablation of bundle branch reentry VT and was excluded from this series. The remaining 10 patients had a mean age of 58 +/- 19 years (range 20 to 73 years), were mostly men (7 of 10 patients), and all presented with documented evidence of symptomatic sustained monomorphic VT, at a mean cycle length of 340 +/- 60 milliseconds (ms) (range 250 to 430 ms). Six patients had coronary artery disease (CAD), one had surgical repair for tetralogy of Fallot, one had surgical repair of a ventricular septal defect, and two had a normal cardiac substrate. The VT origin was mapped using a combination of activation mapping, mid-diastolic potentials, pace mapping, and concealed entrainment. A mean of 5 +/- 3 (range 2 to 11) RF applications were administered to the putative VT foci. Eight of 10 (80%) clinical VTs were successfully ablated. There were no serious complications. Patients with VT originating from the left ventricle were offered implantable cardioverter-defibrillator back-up; however, only one patient accepted this option. At a mean follow-up of 12 +/- 7 months, only one patient had a possible arrhythmia recurrence.