Joven atleta de elite afrodescendiente de 23 años que concurre para *screening* – 2010

Dr. Andrés R. Pérez Riera

Este traçado pertence a um jovem atleta de elite que veio para screening.

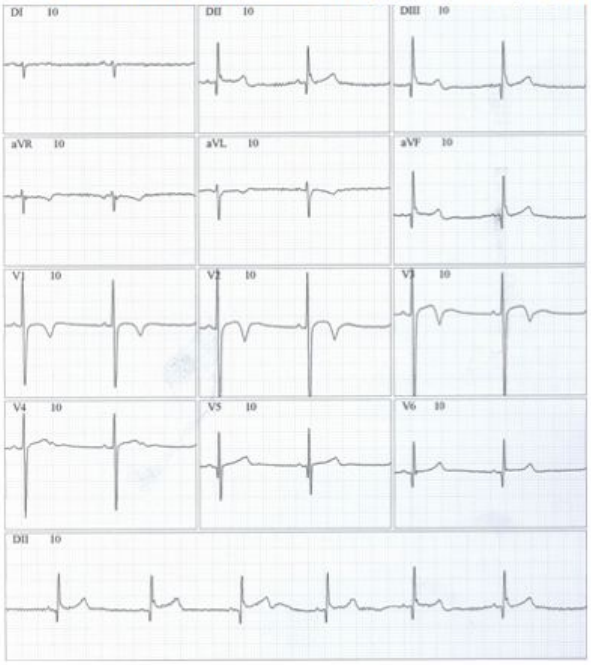
Pergunta: Qual é o diagnóstico eletrocardiográfico?

Podemos liberar assim?

Necessário outro exame?

Andrés R. Pérez Riera

Masculino. Afrodescendente, 23 anos, 77kg, 1,80m. Atleta de elite, modalidade: Futebol, Assintomático
ECG realizado como screening pre-participação



Eco: Espessura miocárdica aumentada em graus discretos:
do VE: Septo: 12mm
Parede posterior: 12mm
FE: 60%
Diâmetro Diastólico: 52mm
(Normal até 50mm)
Diâmetro Sistólico: 35mm

Teste Ergométrico: Normal

Pergunta:
Qual é o diagnóstico ECG?
Conduta? Liberado?

OPINIONES DE COLEGAS

Querido Andrés, a mi entender tiene un Síndrome de Repolarización precoz, donde se describen 3 tipos de patrones distintos:

Tipo 1 con la pseudo onda delta u onda de Osborn en V4 y V5, que lo que más comunmente se encuentra en los atletas de alto rendimiento y hasta ahora en lo reportado en sobrevivientes de MS por FV primaria, este patrón no se ve

Tipo 2: que es la pseudo onda delta en cara inferior y V4 V5, D1 y aVL, este grupo en teoría es el de mayor riesgo de eventos graves y comunmente se encuentra en los sobrevivientes de FV primaria

Tipor 3 que es un tipo 2 más alteraciones en precordiales derechas, y se asocia gravemente a tormentas eléctricas inmanejables.

No se ve bien el ECG por la definición pero creo es un Tipo 2, y el trastorno de precordiales derechas o es el remodelado del atleta, descartarçoa un DAVD asociada y menos frecuentemente creo sea un Tipo 3.

Sin dudas suspenderia el deporte y le haria un EEF.

Saludos

Francisco Femenia

Estimado Maestro Pérez Riera:

Observo RS 53 por min. Eje eléctrico desviado más allá de los 120°. Eje de onda P normal. PR normal. QRS 0,08 seg. QT 0,42 seg.

R altas de V1 a V3 con T negativas.

Llama la atención el punto J en cara inferior (DII. DIII y aVF) elevado y el supra ST (repolarización precoz), no presenta onda epsilon (no es un criterio absoluto para ARDV y no siempre esta presente).

Y no observo en precordiales derechas Repolarizacion precoz concomitante.

El eco informa una HVI excéntrica borderline por su pared posterior para un atleta, desgraciadamente no han referido VD y patrón de llenado diastólico que serían de utilidad para definir una conducta.

Me impresiona que una RMN podría aportar datos acerca de la estructura miocárdica del VD.

No creo el Holter no aportara datos ya que es un paciente asinttomático

Si creo que aportaría el estudio de potenciales tardíos.

Me gustaría conocer sus opiniones si la prueba de ajmalina para descartar Sme de Brugada puede tener alguna utilidad en este paciente y mi otra duda es cuanto me podría aportar el EEF si todo lo demás es negativo (en esto si alguien me puede iluminar estaría muy agradecido).

Un cordial saludo a todos

Martin Ibarrola

Early repolarization syndrome: Is it always benign?

Konstantinos P. Letsas *, Michalis Efremidis, Loukas K. Pappas, Gerasimos Gavrielatos, Virginia Markou, Antonios Sideris, Fotis Kardaras

En un articulo por Ud citado menciona la repolarización precoz en cara inferior espero encontrar su interpretación en este caso.

Idiopathic ventricular fibrillation related to a prominent J wave in the inferior leads

(a variant of BrS with ST-segment elevation in the inferior leads but no coved or saddleback ST-segment elevation in the right precordial leads.) Gussak named this wave as lambda wave due to its morphology;

Arrhythmogenic Right Ventricular Dysplasia: Sometimes, the electrocardiographic phenotype is impossible to differentiate from the electrocardiographic pattern in BrS these cases, observed in the so-called minor or concealed forms, only magnetic nuclear resonance is useful in differentiating both entities.

Estimado Maestro:

Mi humilde opinión es que se trata de un paciente con patrón de repolarización precoz en el ECG asintomático.

No prohibiría la realización de deportes.
No continuaría con más que un estudio de monitoreo Holter para detectar probables extrasístoles con acoplamiento temprano si es que existiera.
Creo que se lo puede "liberar" como usted dice así
Sé que el maestro Femenía me va a matar! Pero a continuación explico por qué es que creo en esta conducta.
En el artículo de Haissaguerre de 2008 NEJM, se dice lo siguiente:

"Case subjects (those with VF) with early repolarization were more likely to be male, with no difference if they belonged to the ethnic group called "Black", were also more likely to have a history of unexplained syncope or sudden cardiac arrest during sleep, and to have a shorter QTc interval than those without early repolarization. In most of the case subjects, ectopy had a positive QRS morphologic pattern in leads V1 to V2, which indicated an origin from the left ventricle and a short coupling interval initiating ventricular fibrillation.
Continúo en inglés....
Another point to have in mind is in the Haissaguerre study is that the confirmation of idiopathic ventricular fibrillation by exercise test was a
difference with statistical significance.
The link between this electrocardiographic pattern and malignant arrhythmias is supported by both the accentuated repolarization before the onset of
arrhythmia in the case subjects and the origin of triggering beats from the region of early repolarization.

THAT'S WHY I THINK A HOLTER MONITORING TEST SEARCHING FOR PREMATURE BEATS WOULD BE USEFUL IN THIS CASE.
Finally, althought to our knowledge no multicenter study has specifically examined the association between early repolarization and sudden cardiac arrest,
anecdotal reports have described patients who had sudden cardiac arrest associated with abnormal J waves.
Another thing to have in mind is that Haissaguerre study conclusions pointed the fact that "althought the cohort included subjects with strictly defined common features, data collection was no uniform among centers. In our study population, we had no subjects with structural heart disease an few athletes or blacks, so the results may no apply to these subgroups."
Most important, althought our results suggest that early reporlarization is a marker of a disorder associated with malignant arrhythmias, natural-history
studies predict a benign course for most of these patients.

UN POCO PARA RESPONDER LA PREGUNTA DE MARTÍN ES LO QUE SE PUEDE EXTRAER DEL ARTICULO DE NAM DE JACC.

In these patients with idiopathic ventricular fibrillation, unlike patients with the Brugada syndrome, the electrocardiographic and arrhythmic abnormalities could not be provoked with intravenous flecainide, were not limited to the right precordial leads, and were not accompanied by abnormalities on the signal averaged electrocardiogram.

PARA TERMINAR EN EL ARTÍCULO DE SAMI VISKIN EN JACC, SE ACLARA QUE
"The Haissaguerre study triggered a "fear of J waves". Electrophysiologist may be consulted about the arrhythmic death risk after an incidental discovery of J waves.

Unfortunately, we do not know how to distinguish "arrhythmogenic" from "normal" J waves.
Continúa de la siguiente manera
"Finally, arrhythmias in idiopathic VF are not provocable by exercise but may be induced with programmed ventricular stimulation. Thus, electrophysiologists may be tempted to recommend electrophysiologic studies for risk stratification of patients with J waves.

THAT WOULD BE UNADVISABLE BECAUSE THE VALUE OF VF INDUCTION FOR PREDICTING SPONTANEOUS ARRHYTHMIAS REMAINS CONTENTIOUS. SINCE IDIOPATHIC VF IS A RARE DISEASE, AND THE ARE NO TEST FOR CONFIRMING OR EXCLUDING IT IN THE ASYMPTOMATIC PATIENT, ASYMPTOMATIC PATIENTS WITH J WAVES SHOULD NOT UNDERGO FURTHER TESTING.
DURING THE LAST DECADE, NUMEROUS YOUNG AND OTHERWISE HEALTHY INDIVIDUALS WORLDWIDE UNDERWENT PROPHYLACTIC IMPLANTATION OF DEFIBRILLATORS BECAUSE OF "ASYMPTOMATIC BRUGADA SYNDROME WITH INDUCIBLE VF". YET, THE CONSEQUENCES OF THAT POLICY ARE ONLY NOW BEING APPRECIATED. HOPEFULLLY, PATIENTS WITH "ASYMPTOMATIC J WAVES AND INDUCIBLE VF" WILL NO SHATE THE SAME FATE.

Jorge Palazzolo Peñafiel

Querido amigo Francisco: tengo la impresion que tentaste escribir una cosa y escribiste en otra.

Sugier que este atleta podría tener un sindrome de repolarización precoz. A seguir, tu expresas que existen 3 variantes de repolarización precóz denominadas tipo 1, 2 y 3 y que el tipo 1 tiene una pseudo onda delta o onda de Osborn en determinada derivación. En este exacto momento comienza la confusión.

¿Por qué digo esto?

Respuesta: porque la onda delta por definición tá localizada en el comienzo del complejo QRS y patrimonio de la pre-excitación ventricular tipo Wollf-Parkinson-White. Contrariamente la mal llamada onda de Osborn se encuentra al final del complejo QRS decir en el punto J (unión del fin del complejo QRS e inicio del segmento ST) .

Toda onda o deflexión extra de inscripción lenta del ECG localizada entre o fin del QRS e inicio del segmento ST, en el lugar correspondiente al punto J puede corresponder a o a la onda epsilon. La primera, denominada onda de la unión (“*junctional wave*”) o deflexión

J ("*the J deflection"*). Por ser una onda y no un punto afecta tambiém la porción inicial del segmento ST ocasionando en él un supradnivel de convexidad superior ("*coved type*") motivo por el cual se la conoce también como potencial de lión ("*injury potential*"). De te perfil de convexidad superior há surgido la denominación de signo de la joroba de camello ("*camel-hump sign*" o "*the camel's hump*") o deflexión semejante a una joroba ("*hump like deflection*"). Por ser semejante en seu aspecto com la onda delta (ð) de la pré-excitación ventricular há sido raramente empleado el término de onda **delta tardia**. La onda δ de pré-excitación ventricular ta localizada en el llamado punto Ja (correspondiente al fin del segmento PR e inicio del complejo QRS).

Finalmente existe la difundida denominación que emplea el epónimo **onda de Osborn** ("Osborn wave") que en nuestro entender es una denominación injusta, porque el primero en describirla fue Tomaszewski[1]. Cinco años después de la primera descripción, el alemán Grosse-Brockhoff, F. et. al la describen en trabajo experimental[2].

La descripción de Osborn ocurrió solo 15 años mais tarde, en 1953[3]. Por ironia, textos conceptuados de Cardiologia denominam a ta onda como siendo de Osborne adicionando la vocal e al nombre original del autor[4].

Esta clasificación que tú adoptas en este momento fue propuesta muy recientemente por Charlie Antezelevich y Yan (5) pero aparte del enorme respeto que me merece estete colosal pesquizador discordo frontalmente con él.

Recientemente el gran maestro de electrocardiologia Dr. Borys Surawitz me envió un e-mail criticando duramente ta clasificación de la repolarización precóz en tres tipos propuesta por Charlie Antezelevitch y Yan (5). El viejo maestro titulaba su comentário asi:

"***birth of illegitimate nomenclature J wave syndrom and early repolarization"*** (nacimiento de una nomenclatura ilegítima

síndrome de y de repolarización) con la que yo concorde en género, número y grado.

Por casualidad Charlie me enviara una réplica hoy,

Las conversacion entre Borys, yo y Charlie tan a seguir (en ingl) pero vale la pena tentar leerlo.

Referências

1) Tomaszewski W. Changements electrocardiographiqu observ ch un homme mort de froid. Arch Mal Coeur 1938; 31:525-528.

2) Grosse-Brockhoff F, Schoedel W. Das Bild der akuten Unterkuhlung in Tierexperiment. Arch. Exp. Pathol Phramakol.1943; 201: 417.

3) Osborn JJ. Experimental hypothermia: rpiratory and blood pH chang in relation to cardiac function. Am J Physiol 1953; 175:388-398.

4) Braunwald E. HEART DISEASE. A Texbook of Cardiovascular Medicine. 5th Edition, 1997; pg 140-141.

5) Antzelevitch C, Yan GX.J wav syndrom. Heart Rhythm. 2010 Apr;7(4):549-58.

De: Borys Surawicz
Enviada em: domingo, 23 de maio de 2010 22:58
Para: Pérez Riera
Assunto: Re:

BIRTH OF ILLEGITIMATE NOMENCLATURE J WAVE SYNDROMES AND EARLY REPOLARIZATION

The term J-wave syndrom appeared first in a paper by Yan et al (1) and is a title of a contemporary review by Antzelevitch and Yan (2). In the Table1 1 of this review, the authors list four inherited and two acquired typ. of J wave syndrom

The inherited typ are:

- Early Repolarization (ER) type 1 where ER is located in lateral leads;

- type 2 where ER is in inferior or inferolateral leads;

- type 3 with global ER, and the forth is Brugada syndrome.

The two acquired typ are associated with ventricular tachycardia or ventricular fibrillation (VT/VF); one is ischemia-mediated, and the other hypothermia - mediated.

What do J stand for in cardiac electrophysiology?

J wave

J wave or Osborne wave frequently appears during deep hypothermia (3). It is a low frequency deflection at the end of QRS complex that is morphologically similar to P wave. It is not clear whether it reprents ventricular depolarization or early repolarization. Similar deflection may be prent in patients with hypercalcemia, Brugada syndrome, and in several other conditions (3)

J point elevation

J point marks the end QRS complex, and is often situated above the baseline with high prevalence in normal younger mal, during myocardial ischemia as a rult of injury current, and in various other patterns of normal and abnormal ECGs.

Defining normal limits of J point elevation in mV above the baseline must consider several variabl, such as the lead, heart rate, age, gender and overall amplitude of the QRS complex. This mak it

difficult to evaluate the significance of increased J point elevation found in survivors of primary ventricular fibrillation (4)

The tale of two J's

It is curious that the letter J in cardiac electrophysiology stands to define two unrelated and totally different events i.e. a rarely occurring long slow deflection of uncertain origin at the end of QRS, r in several typ of abnormal ECG, and a point in time identifying the end of QRS that is prent in all ECGs. The difference between the meaning of two J's is so big that confusion is not expected

What is early repolarization?

The repolarization in ventricular myocardial cells may or may not begin with a notch attributed to transient outward current (ito), and is followed by a plateau (phase2) and rapid phase3 (5). To my knowledge the course of repolarization has not been separated into an early and late phase. Therefore the term ER was generated by electrocardiographers and not by electrophysiologists

Origin of the term and clinical significance of early repolarization

Antzelevitch and Yan (6) reported that in 1961 Wasserburger and Alt (7) defined ER as an elevated takeoff of the ST segment of the QRS complex varying from 1 to from the isoelectric line accompanied by downward concavity of the ST segment and symmetrically limbed T wav.

Kimbaris and Phillips used the term ER in the title of their1976 paper (8), but concluded that it was a normal variant. Haissaguerre et al (9) report that ER is prent in 1-5% of population, but refer to a study of Gussak and Antzelevitch (10) claiming that ER has a potential for arrhythmogenicity .

The Haissaguerre study (9) included 206 subjects who were ruscitated from cardiac arrt caused by idiopathic ventricular fibrillation. ER was prent in 31% of subjects vs 5% in control subjects ($p<0.001$). The ER in this study was defined as an elevation of QRS-ST junction of at least 0.1 mV from baseline in the inferior or lateral lead manifted as QRS slurring or notching. Similarly asssed was the risk of ER in otherstudi of smaller number of patients (11-14)

Morphology of ER

In publications in which ER (mostly in leads III,aF and V4-6)) is identified by an arrow, the 12 lead ECGs were recorded at the speed of 25 mm/s.. When the figur were magnified. one could see that the arrows pointed either at a slow dcent of R wave toward baseline (reminiscent of poor frequency rponse of the recorder), or at notch before or after the approach of R dcent to the baseline,.In one case the terminal deflection rembled one in incomplete RBBB.

Unfortunately, the deflections marked as ER in above studi were not synchronized in several leads, not recorded at higher paper speeds, and not examined by body surface mapping or vectorcardiographic analysis. Thus, it is not possible to know whether the alleged ER is indeed repolarization. The morphology is against it

Wellens in his editorial comment of the study of Haissaguerre et al (15) qutioned "…. whether the dcribed abnormality at the end of QRS complex is indeed early repolarization, or rather delayed activation of the inferolateral wall". A debate about he origin of the terminal QRS portion took place in reference to subjects with Brugada syndrome.(16)

Conclusion: The use of terms J wave syndrom and early repolarization syndrom should be discouraged, The former cannot be defined and the latter probably has a wrong name. To avoid their proliferation, this information should be available to the editors, reviewers and readers, of appropriate journals.

Proper analysis of the ECG in patients with the infrequently occurring patterns discussed above may be rewarding

References:

1.Yan GX,Yan QH,Wang DQ, Cui CC: Electrocardiographic J wave and J wave syndrom. Clin Arrhyth 2004;8:360-365

2.Antzelevitch C, Yan GX: J wave syndrom. Heart Rhythm 2010;7:549-558.

3. Surawicz B, Knilans TK: Chou's Electrocardiography in Clinical Practice, sixth edition, Elsevier Saunders, 2008; pp 547-549

4. Rosso R, Kogan E, Belhassen B, et al: J-point elevation in survivors of primary ventricular fibrillation and matched control subjects. J Am Coll Cardiol 2008; 52;1231-8

5. Surawicz B: Electrophysiologic Basis of ECG and Cardiac Arrhythmias,1996 Williams and Wilkins pp 31-33

6. Antzelevitch C, Yan GX: J wave syndrom. Heart Rhythm 2010;7:549-558

7. Wasserburger RH, Alt WJ. The mormal RS-T segment elevation variant Am J Cardiol 1961;8:184-192

8. Kimbara H, Philips J: Long- term evaluation of early repolarization syndrome (normal variant RS-T segment elevation). Am J Cardiol 1976:33:157-161

9. Haissaguerre M, Derval N, Sacher F, Jsel,et al: Sudden cardiac arrt associated with early repolarization N.Engl.J.Med. 2008;358;216-2005.

10. Gussak I, Antzelevitch C: Early repolarization syndrome:: clinical characteristics and possible cellular and ionic mechanisms. J Electrocardiol 2000;33:299-309

11. Tikkanen JT, Anttonen O,Juntila J Aro Al,et al:Long-term outcome associated with early repolarization on electrocardiography. N.Engl.J Med 2009 361:2529-2538

12. Watanabe H, Mariyama T, Koyama T, Kannankeril Pj et al: High prevalence of early repolarization in short QT syndrome Heart Rhythm 2010;7: 647-652

13. Ghosh S, Cooper DH,,Vijayakumar, Zhang J, et al: Early repolarization associated with sudden death:Insights from noninvasive electrocardiographic imaging. Heart Rhythm 2010;7:534-537

14. Sinner MF, Reinhard W,Mueller M, Beckmann B-M,et al: Early repolarization on ECG is common and is associated with increased cardiovascular mortality Heart Rhythm 7 5 May supplement 2010 (abstract)

15. Wellens HJ: Early repolarization revisited. N Engl J Med 358;2063-65,2008

16. Borggrefe M, Schimpf:J-wave syndrom caused by repolarization or depolarization mechanisms JACC 2010:58:798-800

.

Subject: BIRTH OF ILLEGITIMATE NOMENCLATURE

Importance: High

My deart Prof. Dr. and dear all: I agree totally with you in this particular topic deart teacher (nomenclature).

Please see inside the attachment. In this case with ECG/VCG we demonstrate the prence of the two operative forms (depolarization and repolarization mechanisms) in symptomatic patient with spontaneous type 1 ECG Brugada pattern (to day, we have 11 cas like this one)

What do you think dear Arthur? Do you agree with us?

And what do you think Martin?

Which is your valuable opinion Prof Haissaguerre?

Any reply Charlie?

All the bt.

Andr Pérez Riera

Dear colleagu

It seems that this e-mail fell between the cracks. This is a fascinating debate indeed among individuals that I hold in the hight teem.

The term early repolarization has had many critics over the years. From the cellular electrophysiology vantage point, it would appear to be appropriate nomenclature in that the electrocardiographic maniftations are principally influenced by phase 1 of the action potential, which is the “early” repolarization phase.

In experimental models, we and others have succeeded in recapitulating all of the maniftations of early repolarization pattern via accentuation of this early repolarization phase of the action potential.

To what degree the maniftations are due to depolarization vs. repolarization can lead to a stimulating debate as well. and I and our colleagu have recently been invited to debate this issue as

it pertains to Brugada syndrome in a point/counterpoint that will be published in JMCC in the near future.

Arthur is among the scientist/physicians that I admire most in our field, yet we are deeply divided in our views, which we would be delighted to share with you once this publication is out.

With regards to the nomenclature of J wave syndrom, I think there is general consensus that the syndrom whose phenotypic exprsion rang from ERS to BrS to hypothermia, are associated with accentuation of the electrocardiographic J wave. Their reponse to rate and autonomic influence are similar and consistent with Ito-mediated accentuation of the epicardial action potential notch (early repolarization), as the underlying mechanism. Inhibition of the transient outward current (Ito) with agents like quinidine diminish the epicardial action potential notch, thus reducing the maniftation of the J wave and providing protection against arrhythmias.

Warmt regards to all

Charlie

Charl Antzelevitch, PhD, FACC, FAHA, FHRS
Executive Director/Director of Rearch
Gordon K. Moe Scholar
Profsor of Pharmacology
Masonic Medical Rearch Laboratory

Estimado Maestro:

Mi humilde opinión es que se trata de un paciente con patrón de repolarización precoz en el ECG asintomático.
No prohibiría la realización de deportes.
No continuaría con más que un estudio de monitoreo Holter para detectar probables extrasístoles con acoplamiento temprano si es que existiera.
Creo que se lo puede "liberar" como usted dice así

Jorge Palazzolo Peñafiel

Estimado Maestro Pérez Riera:

En el texo referido por Ud a Charles Antzelevitch, PhD dice: *And what do you think Martin?*

Me pregunta a mi estimado Maestro?? Yo aguardo sus conclusiones de las cuales siempre aprendo y más cuando ni siquiera logro acceder al paper de la clasificación referida por no estar subscripto. Perdón sino se refería a mí.

Yo tengo más preguntas que respuestas en este paciente:

1. En la elevación del punto J en derivaciones inferiores lo publicado en NEJM por Ti K Kanen puede contribuir a un pronóstico?

2. ¿Normaliza el segmento ST en el ejercicio o con isoproterenol.?

3. ¿Le agrega riesgo la práctica deportiva?

4. No tiene antecedentes familiares positivos, es un paciente asintomático. ¿Cuál conducta? (¿CDI? no le encuentro indicación con los datos actuales; ¿quinidina? realmente no me convence; ¿estudios y seguimiento y continuar con su práctica deportiva?)

Como ya dije espero sus conclusiones, he dicho que me parecía, no creo aporte más que los Maestros, solo leer y aprender.

Le envío un cordial saludo.

Martin ibarrola

Coincido ampliamente con lo dicho por Jorge.

Haissaguerre en su artículo lo expresa claramente. No pongamos el carro delante del caballo. Seamos prudentes y "no enfermemos". Saludos.

Oscar Pellizzón

En las "Recomendaciones para la Interpretación del ECG de 12 derivaciones en el Atleta", un ESC Report, European Heart Journal (2010) 31, 243-259, con relación a la Repolarización temprana: es la regla, más que la excepción entre atletas altamente entrenados, y que es un patrón ecgráfico fisiológico y benigno en la población general de jóvenes y atletas y no requiere evaluación clínica posterior, excepto los casos de atletas que se presenten con síncope o arresto cardíaco; y a las inversiones de onda T (igual o + de 2 mm, y en 2 o + derivaciones adyacentes) las clasifican en el grupo de anormalidades no comunes y no relacionadas al entrenamiento y si hay que pensar en descartar ciertas

patologías cardíacas (también se los puedo enviar).

En el Cogreso ESC 2007, el Dr. Antonio Pelliccia, ofreció una tremenda presentación (se la paso gratis al que así lo desee), sobre Patrones de Repolarización Anormal en Atletas (con casos de futbolistas, por supuesto), tratando lo de las ondas T negativas en una cohorte de atletas con anormalidades electrocardiográficas, incluso por grupos etarios, y preguntándose cuál sería el significado clínico y pronóstico de estos deportistas con patrones anormales de repolarizacíón y ausencia de enfermedad cardiovascular detectable: de 12,880 atletas examinados (1979-2001), a 39 se les demostró enfermedad CV, de los cuales, 17 tenían Cardiomiopatía hipertrófica, incluyendo ondas T invertidas en cara inferior; lo cual, también fué asociado a un riesgo incrementado de MCS en sujetos de mediana edad en la población general, ("nuestro futbollista no las tiene") en un artículo del NEJM del 16 de nov. del 2009:

Long-Term Outcome Associated with Early Repolarization on Electrocardiography

Jani T. Tikkanen, B.S., Olli Anttonen, M.D., M. Juhani Junttila, M.D., Aapo L. Aro, M.D., Tuomas Kerola, M.D., Harri A. Rissanen, M.Sc., Antti Reunanen, M.D., and Heikki V. Huikuri, M.D.

Que estén bien y hasta pronto,

Ricardo Pizarro.

Prezados amigos: nosso parecer e o seguinte: Concordamos com todos os comentários realizados pelo colega Pizarrro. Apenas um detalhe gostaria salientar: O paciente possui:

1) um tipo constitucional normolineo (não é longilineo) e eixo elétrico do QRS em aproximadamente +110º

2) Padrão rS em DI e aVL, qR nas inferior com RIII>RII

3) Entalhe na rampa dcendente do R e aVF

4) S profundas em V2 e V3.

Um trabalho recente (vejam abaixo) comenta que para o grupo suspeito de ter doença subyacente om bloqueio do fasciculo póstero inferior foi o mais frequente fato encontrado!!!!! Em 73 membros (20 women and 53 men) do time Olímpico da

Polonia “From 'suspected' (group II) the most frequent was left posterior fascicular hemiblock,

Alguno de voce pensa que te paciente poderia ter um bloqueio divisional postero-inferior (LPFB)?

Swiatowiec A, Król W, Kuch M, Braksator W, Krysztofiak H, Dłuzniewski M, Mamcarz A. Analysis of 12-lead electrocardiogram in top competitive profsional athlet in the light of recent guidelin. Kardiol Pol. 2009 Oct;67(10):1095-102.

Department of Cardiology, Hypertension and Internal Disease, , , .

Comment in:

Kardiol Pol. 2009 Oct;67(10):1103-4.

Abstract

BACKGROUND: One of the most important aims of modern sports cardiology is prevention of sudden cardiac death among athlet. Adequate pre-participation screening is a crucial part of prevention, however, current ACC, AHA or ESC guidelin are not uniform in this context. There is recently ongoing discussion on implementation of 12-lead ECG to the screening protocol. Aim: To asss the prevalence of alterations of rting 12-lead ECG in a population of top-level profsional athlet - members of the Polish Olympic Team - using recently accepted criteria. METHODS: During the period of intensive training before the Summer Olympic Gam in (2008), a 12-lead, rting ECG was performed in 73 members (20 women and 53 men) of the Polish Olympic Team. Commonly accepted criteria were used to asss the ECG, and alterations were divided into two groups according to recent publications: group I - 'benign', common - thought to be consistent with the athlete's heart syndrome (i.e.: sinus bradycardia, 1st degree atrioventricular block, early repolarisation, right bundle branch hemiblock, isolated signs of left ventricular hypertrophy); and group II - 'suspected', uncommon - which may occur due to organic heart disease (i.e. complete bundle branch block, ventricular arrhythmia, inverse T wave or pathological QRS axis deviation). RESULTS: Completely normal ECG was prent in 11% of those examined, common (group I) findings were observed in 65% and 'suspected' (group II) in 23%. The most commonly occurring 'benign' findings were bradycardia incomplete, right bundle branch block and isolated left ventricular hypertrophy, found in 75, 71 and 41%, rpectively. **From 'suspected' (group II) the most frequent was left posterior fascicular hemiblock,** prent in 10% of those examined; other findings were complete right bundle branch block, left atrial hypertrophy, inverse T wav and left anterior fascicular hemiblock in single cas. CONCLUSIONS: 1. Most of the observed alterations in rting ECG of profsional athlet belong to the 'common' group and rult from adaptation to exercise. 2. Frequent occurrence of left posterior fascicular hemiblock, which is thought to be 'potentially malignant', requir further invtigation.

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Andres R. Pérez Riera

Estimado Maestro Perez Riera: desgraciadamente creo no son interpolables ambas poblaciones, en el articulo por Ud citado son deportistas olimpicos y en el articulo de Pellicia es screning precompetitivo en italianos por eso la llamativa diferencia a mi juicio de la frecuenica del hallazgo aislado del hemibloqueo del fasciculo posterior izquierdo (menos del 0,1% en la serie italiana) frente a lo por Ud referido.

Sigo creyendo que si bien es probable se trate de cambios fisiológicos por su práctica deportiva, es necesario completar con estudios a fin de demostrar justamente que estos cambios son fisiológicos y no manifestaciones de otras alteraciones.

No es enfermar....nadie le prohibió aún la práctica deportiva. Sino intentar brindarle la mayor seguridad posible al momento de autorizarle realizarla.

Y probablemente no padezca de una ARVD, o una variante de BS pero en ECG con hallazgos no habituales si uno dispone de los medios y puede realizar exámenes complementarios, aportan no quitan.

Y mil gracias por las explicaciones brindadas Dr Pallazolo un gusto.

Un abrazo

Martin Ibarrola

Estimado Maestro:
En mi opinion el paciente no presenta LPFB, ya que si bien tiene desviación del eje a extrema derecha, no encuentro criterios tales como:

- Tiempo pico de onda R en aVL > 45 msec
- Onda S hasta V6, con tiempo de deflección intrinsicoide en V6<aVF
- Rosembaum y colaboradores sugirieron que una desviación del eje > 120 grados como criterio para el diagnóstico de LPFB, pero se menciona así también que el diagnóstico debería ser considerado con un eje más allá de los 90 positivo.

Pero por sobre todas las cosas, no encuentro algo que marca don Bayes de Luna, allá en el libro que recuerdo en esas noches largas de residencia, que solíamos disfrutar esperando "el ingreso" de algún paciente a la sala.

"Para lograr hacer el diagnóstico de hemibloqueo izquierdo posterior, tanto la morfologia electrocardiografica tipica como las condiciones clinical, principalmente la ausencia de agrandamiento ventricular derecho y el hábito asténico deben estar presentes.
Creo yo maestro, y es como dije anteriormente, mi humilde opinión, que se debe esto a su hábito constitucional, y no a LPFB.

Abrazo

Jorge Palazzolo.

Prezado Palazzolo, Usted escreve:"Para lograr hacer el diagnóstico de hemibloqueo izquierdo posterior, tanto la morfologia electrocardiografica tipica como las condiciones clinical, principalmente la ausencia de agrandamiento ventricular derecho y el hábito asténico deben estar presentes. Eu lhe respondo: Todo o contrario. Para poder fazer o diagnóstico de LPFB o hábito asténico deve estar ausente e no presente. Este paciente é normolineo e não longilineo asténico nem tem SVD nem enfisema nem infarto lateral. O diagnóstico de LPFB sempre deve ser clinico-eletrocardiografico e obrigatoriamente nao pode ter SVD, ex enfisema, habito asténico longilineo ou infarto lateral.
Em referencia ao aumento da deflexao intrinsecoide (R peak time = ou > 45ms) no BDPI ocorre nas inferiores aVF e V6 e no na lateral alta aVL. Lembre-se que o primeiro vetor no LPFB aponta

para acima e a esquerda isto é para aVL e só mais tarde para a região inferior que se inscreve com um atraso medio de 20ms.
Por outra parte os sinais de LPFB não necessariamente devem todos estar presentes. Neste caso por exemplo a voltagem da R de DIII mesmo sendo maior do que a R de DII não atinge os 15mm considerado este um outro critério de LPFB ausente neste caso.
Finalmente fijese em V5 possui S final. A ausencia de S final em V6 pode (e deve) ser por má posicionamento de eletrodo precordial fato extremamente frequente
Lhe envio um artigo interesante em Englsih escrito por Morton Arnsdorf acerca do raro LPFB.

Left posterior fascicular block

In the discussion that follows, it is assumed that the reader understands the general concepts of cardiac vectors, asynchronous activation of the ventricles (delayed as in fascicular or bundle branch block, or early as in preexcitation), and the effects that asynchrony has on the duration, morphology and amplitude of the QRS complex.

FASCICLES OF THE LEFT BUNDLE BRANCH ¡ª The classic hypothesis proposed by Rosenbaum and his coworkers was that the left bundle branch divides into two fascicles of rapidly conducting Purkinje fibers (ie, phase 0 dependent on the rapid inward sodium current) [1]. These fascicles primarily affect the direction of depolarization:

- The left anterior fascicle crosses the left ventricular outflow tract and terminates in the Purkinje system of the anterolateral wall of the left ventricle.

- Th e left posterior fascicle appears as an extension of the main bundle and fans out extensively posteriorly toward the papillary muscle and inferoposteriorly to the free wall of the left ventricle.

In addition, a third fascicle, called the left median or centroseptal fascicle, is found in nearly 65 percent of people [2,3]. This fascicle runs to the interventricular septum, and can arise from the common left bundle or from the anterior, posterior or both fascicles.

Support for the trifascicular nature of the left bundle comes from the observations in animals and humans that depolarization of the left ventricle begins in three areas corresponding to the terminal portions of the anterior, posterior and septal fascicles [4,5]. In the normal heart, the three fascicles of the left bundle are simultaneously depolar ized.

Blood supply ¡ª The proximal part of the left posterior fascicle is supplied by the artery to the atrioventricular (AV) node and, at times, by septal branches of the left anterior descending (LAD) artery. The distal portion has a dual blood supply from both anterior and posterior septal perforating arteries. The left anterior and median fascicles are supplied either by septal branches of the LAD or by the AV nodal artery.
ELECTROCARDIOGRAM IN LEFT POSTERIOR FASCICULAR BLOCK ¡ª Anterior and posterior fascicular blocks mainly affect the direction but not the duration of the QRS complex, because the conduction disturbance primarily involves the early phases of activation. (See "Left

anterior fascicular block"). The electrocardiographic criteria for left posterior fascicular block (LPFB, or left posterior hemiblock) are depicted the ECG (show ECG). The changes that are seen reflect altera tions in the different phases of activation.

- Early activation ¡ª Early activation by the normally conducting anterior and septal fascicles causes the initial vector to be directed to the left, anteriorly, and superiorly producing initial small r waves in leads I, V1, and V6.

- Mid-temporal and terminal activation ¡ª The mid-temporal and terminal vectors in LPFB are directed to the right, posteriorly, and inferiorly due to delayed depolarization of the areas normally activated by the left posterior fascicle. This leads to the characteristic rightward axis of +90º to +180º [6]. As a result, there is a qR morphology in leads II, III, aVF and Y and an rS morphology in leads I and aVL.

The QRS duration usually does not exceed 0.10 sec (100 msec), although the WHO/ISFC Task Force allows up to 0.12 sec or 0.02 sec above the previous baseline [6].

Diagnostic problems ¡ª There are a number of settings which may produce ECG findings similar to LPFB.

- The above criteria apply only in the absence of other causes for a rightward axis, such as right ventricular hypertrophy due to valvular heart disease or lung disease with cor pulmonale.

- A high lateral or anterolateral myocardial infarction (MI) can mimic LPFB. With an infarct, however, the initial r wave in leads 1 and aVL is absent and only a Q wave is seen.

- The presence of right bundle branch block (RBBB) might suggest right axis deviation because of the deep terminal S wave in leads 1,aVL and V5, and V6. However, these S waves reflect delayed right ventricular activation, not left ventricular forces. An additional potential source of confusion is that RBBB can occur in association with LPFB.

- The small q waves in the inferior leads in LPFB may cause confusion with an inferior wall MI.

CLINICAL CONSIDERATIONS ¡ª The left posterior fascicle branch is the first branch of the left bundle and is large in its initial course. It then fans extensively throughout the posterior and inferior left ventricle. The left posterior fascicle is exposed to lower pressures and less turbulence than the left anterior fascicle; it also has a dual blood supply. These characteristics probably explain why isolated LPFB is a rare finding.

Isolated LPFB can, however, be seen in the setting of extensive arteriosclerotic cardiovascular disease, as an association with inferior MI and extensive coronary disease has been suggested [7]. LPFB can also occur with cardiomyopathies, including those which result from hypertension and Chagas disease, myoc arditis, hyperkalemia, acute cor pulmonale, and chronic degenerative and fibrotic processes of the conducting system.

TREATMENT ¡ª There are no symptoms induced by an isolated block in one of the left bundle fascicles. Therapy should be considered only in patients with persistent bifascicular or trifascicular block. (See "Course and treatment of chronic bifascicular and trifascicular block").

1. Rosenbaum, M, Elizari, MV, Lazzari, JO. The Hemiblocks. Tampa Tracings, Tampa, 1970.
2. Demoulin, JC, Kulbertus, HE. Histopathological examination of the concept of left hemiblock. Br Heart J 1972; 34:807.
3. Uhley, HN. Some controversy regarding the peripheral distribution of the conduction system. Am J Cardiol 1972; 30:919.
4. Myerburg, RJ, Nilsson, K, Gelband, H. Physiology of canine intraventricular conduction and endocardial excitation. Circ Res 1972; 30:217.
5. Durrer, D, Van Dam, RT, Freud, GE, et al. Total excitation of the isolated human heart. Circulation 1970; 41:899.
6. Willems, JL, Demedina, EO, Bernard, R, et al. World Health Organization/International Society and Federation of Cardiology Task Force. Criteria for intraventricular conduction disturbances. J Am Coll Cardiol 1985; 5:1261.
7. Godat, FJ, Gertsch, M. Isolated left posterior fascicular block: A reliable marker for inferior myocardial infarction and associated severe coronary artery disease. Clin Cardiol 1993; 16:220.

Querido Maestro:
Lo que digo es que la AUSENCIA de condiciones clínicas tales como agrandamiento ventricular derecho y el hábito asténico debe estar presente. Con error en la palabra "presentes" que debería haber sido "presente" para su correcta comprensión.
Es cierta su corrección sobre el R wake peak time >45 msec ya que pertenece a criterio de hemibloqueo izquierdo anterior, y a todo esto no sé por qué coloqué eso. En el futuro he de chequear lo que escribo al menos dos veces antes de enviar mensaje al foro.
Le agradezco su corrección y aporte, el cual había leído en Uptodate, muy bueno por cierto.
Gracias maestro....

Jorge Palazzolo Peñafiel