Brugada syndrome associated to Myocardial ischemia

Sindrome de Brugada associado a isquemia miocárdica

Case of Dr Raimundo Barbosa Barros From Fortaleza - Ceará - Brazil

Caro amigo Dr. Andrés

Gostaria de ouvir a opinião dos colegas do foro sobre este paciente masculino 56anos internado na emergência do nosso hospital dia 03 de Novembro de 2010. Relata que em abril deste ano foi internado por quadro clínico compatível com angina instável (ECG1). Na ocasião foi submetido à coronariografia que revelou lesão crítica proximal da arteria descendente anterior e lesão de 90% na porção distal da artéria coronária direita. Nesta ocasião realizou angioplastia com colocação de stent apenas na artéria descendente anterior (ECG2 pós ATC) A artéria coronaria direita não foi abordada. O paciente evoluiu assintomático(ECG3).

Em 20/09/2010 realizou cintilografia miocárdica de rotina que resultou normal.

No dia 03 de novembro de 2010 procura emergência refirindo ter sofrido episódio de sincope precedido de palpitações rápidas e desconforto torácico atípico.(ECGs 4 e 5). Adicionalmente, informa que 4 horas antes da sua admissão havia apresentado febre (não documentada).

Não há relato de episódio prévio semelhante ou história familiar positiva para Morte súbita em familiar jovem de primeiro grau.

Dosagem seriada de CK-MB e troponina normais.

Qual os diagnósticos ECGs e qual a conduta?

Um abraço para todos

Raimundo Barbosa Barros Fortaleza Ceará Brasil

Dear friend, Dr. Andrés,

I would like to know the opinion from the colleagues of the forum about this patient (male, 56 years old), admitted in the ER of our hospital, on November 3rd, 2010. He claims that in April of this year he was admitted with symptoms of unstable angina (ECG1). He underwent coronary angiography that revealed severe proximal lesion of the LAD and 90% of obstruction in the distal portion of the RCA. Back then, he underwent angioplasty with stent in the LAD (ECG2 post-PTCA); the RCA was not approached.

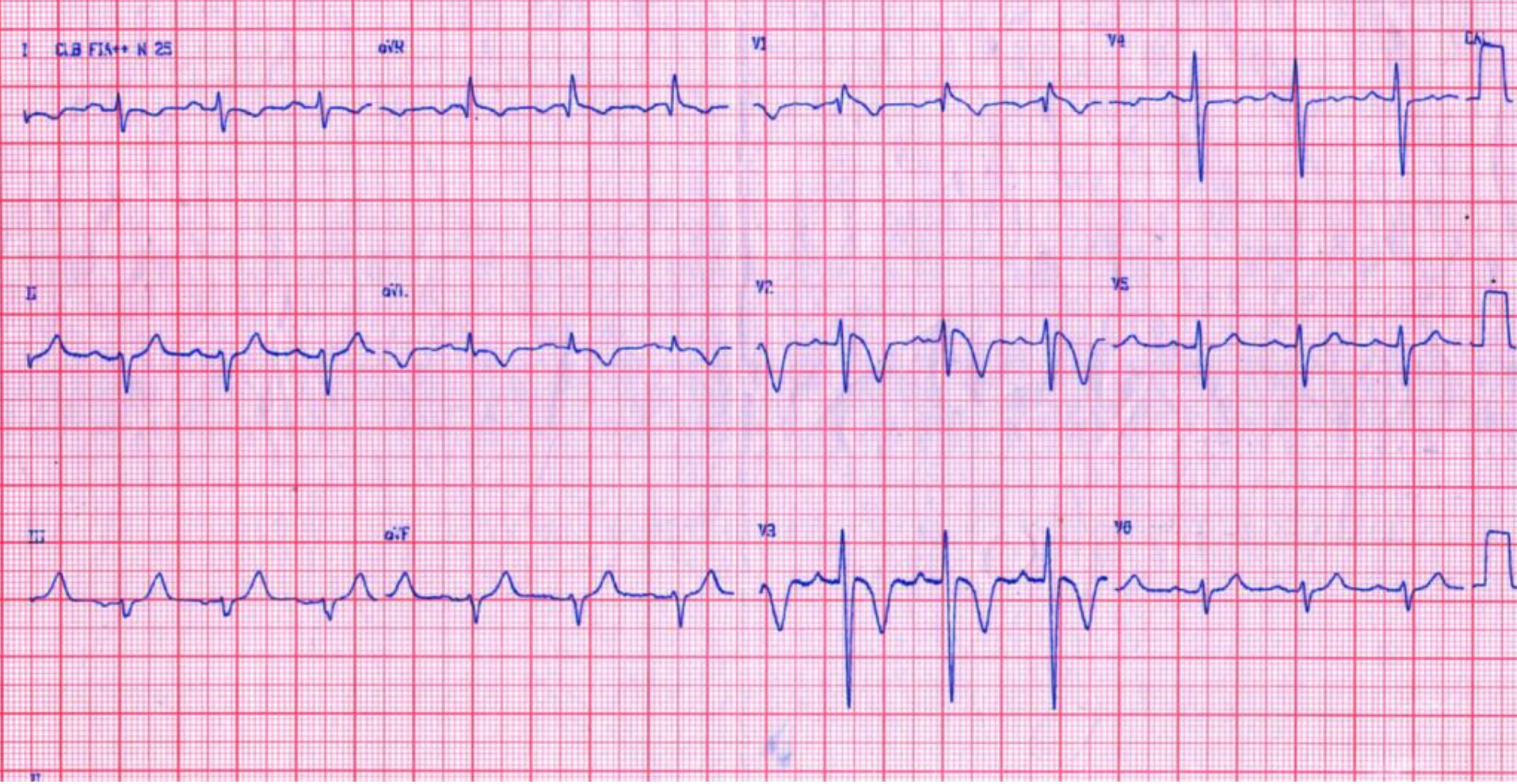
The patient evolved without symptoms (ECG3). On September 20th, 2010, routine myocardial perfusion imaging was made (normal). He went to the ER on November 3rd, 2010 after syncope episode, preceded by tachycardiac palpitations + atypical chest discomfort (ECGs 3 and 4). He says that 4 hours before being admitted, he had presented fever (not documented). He says there was no previous similar episode or positive family history.

Serial measurement of CK-MB and troponin is normal.

What is the diagnosis and the management?

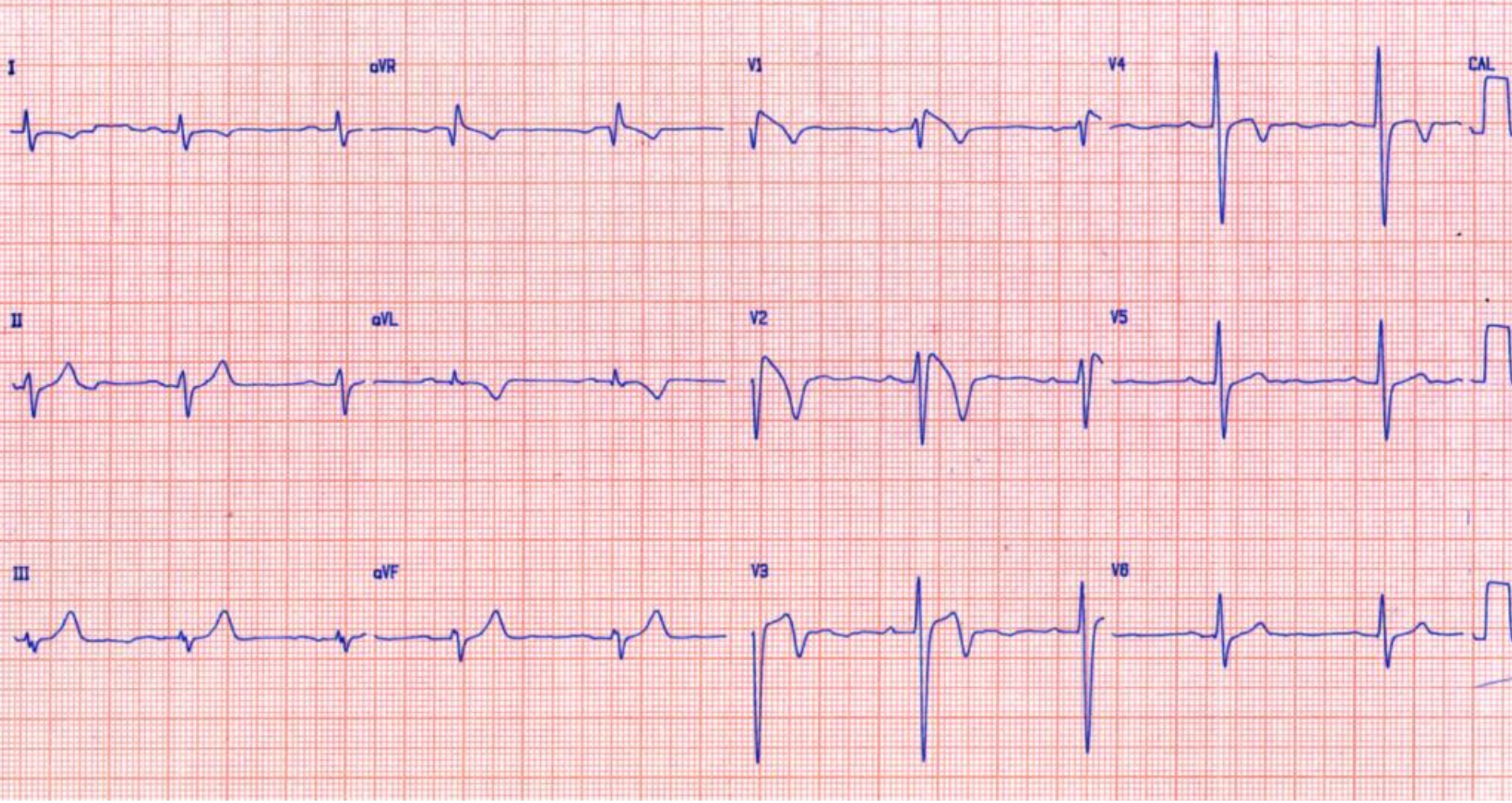
Warm regards,

Raimundo Barbosa Barros



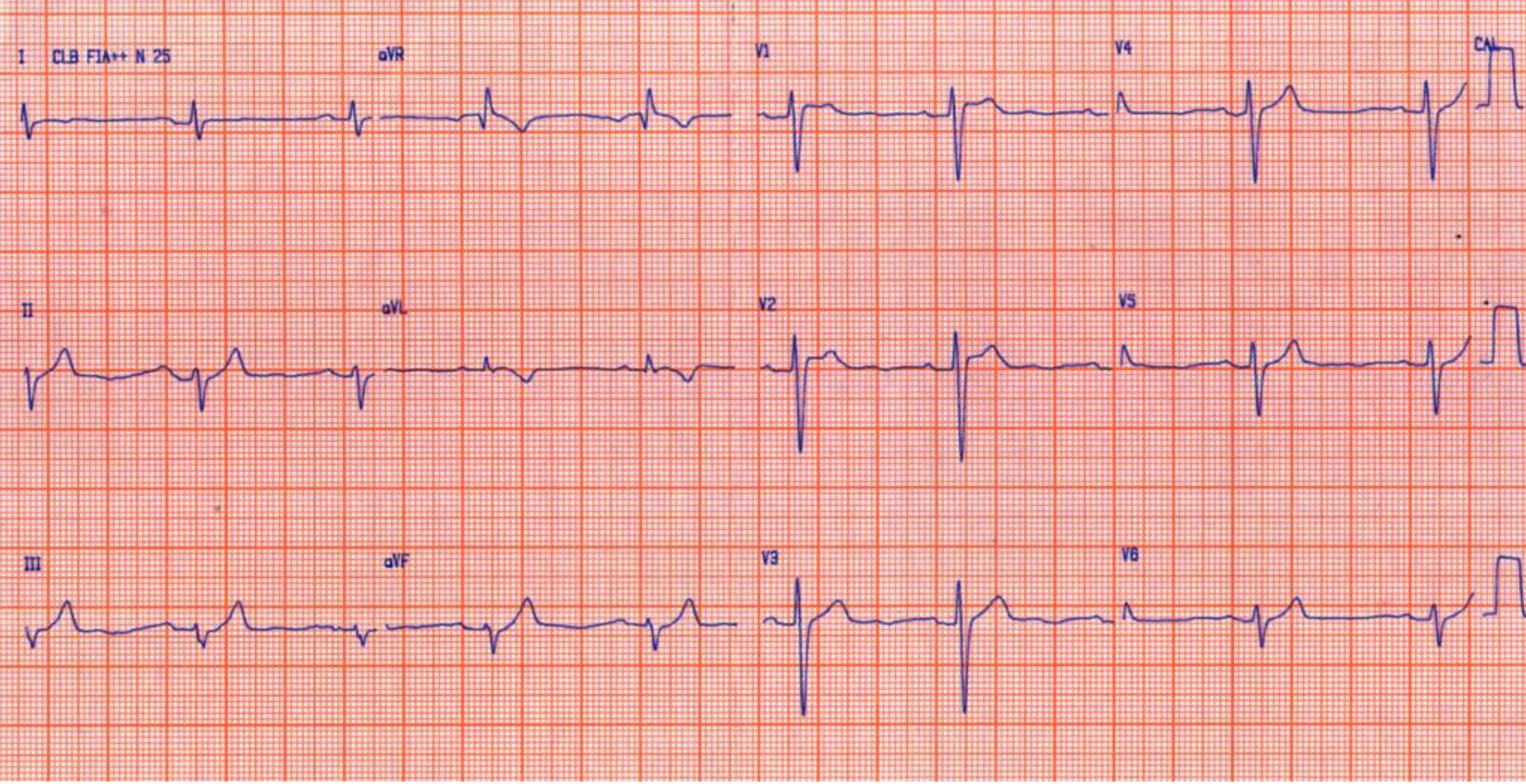
First ECG performed during angina unstable episode. April 2010

Primeiro ECG realizado durante o episódio de angor instável. Abrli de 2010



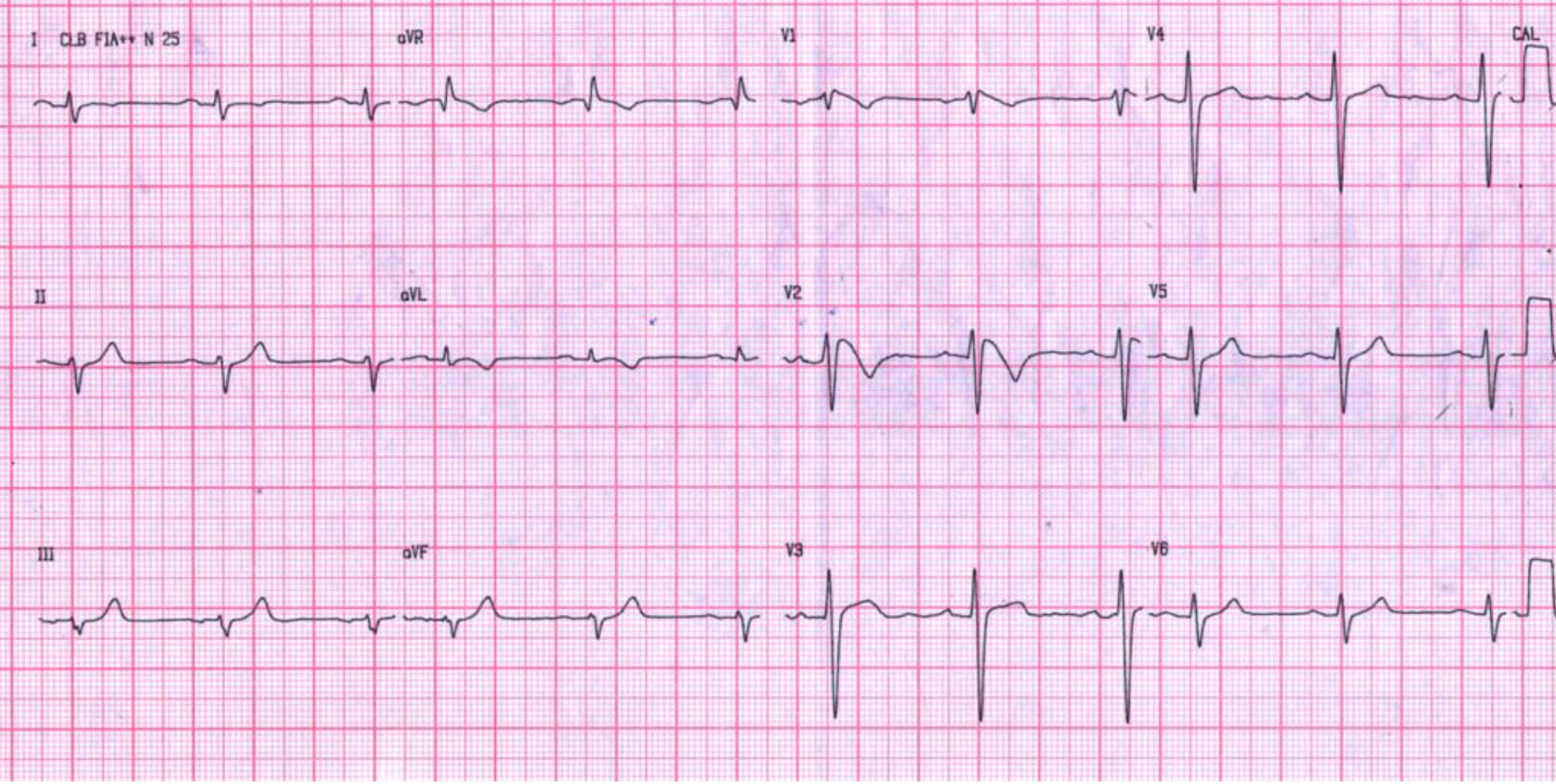
Second ECG preformed immediately after Percutaneous Coronary Intervention (PCI), or coronary angioplasty. A coronary stent was placed in LAD during a PCI procedure.

Segundo ECG realizado logo após a angioplastia na qual se colocou um stent na DA durante o procedimento.



Third ECG late evolution after procedure. Patient asymptomatic

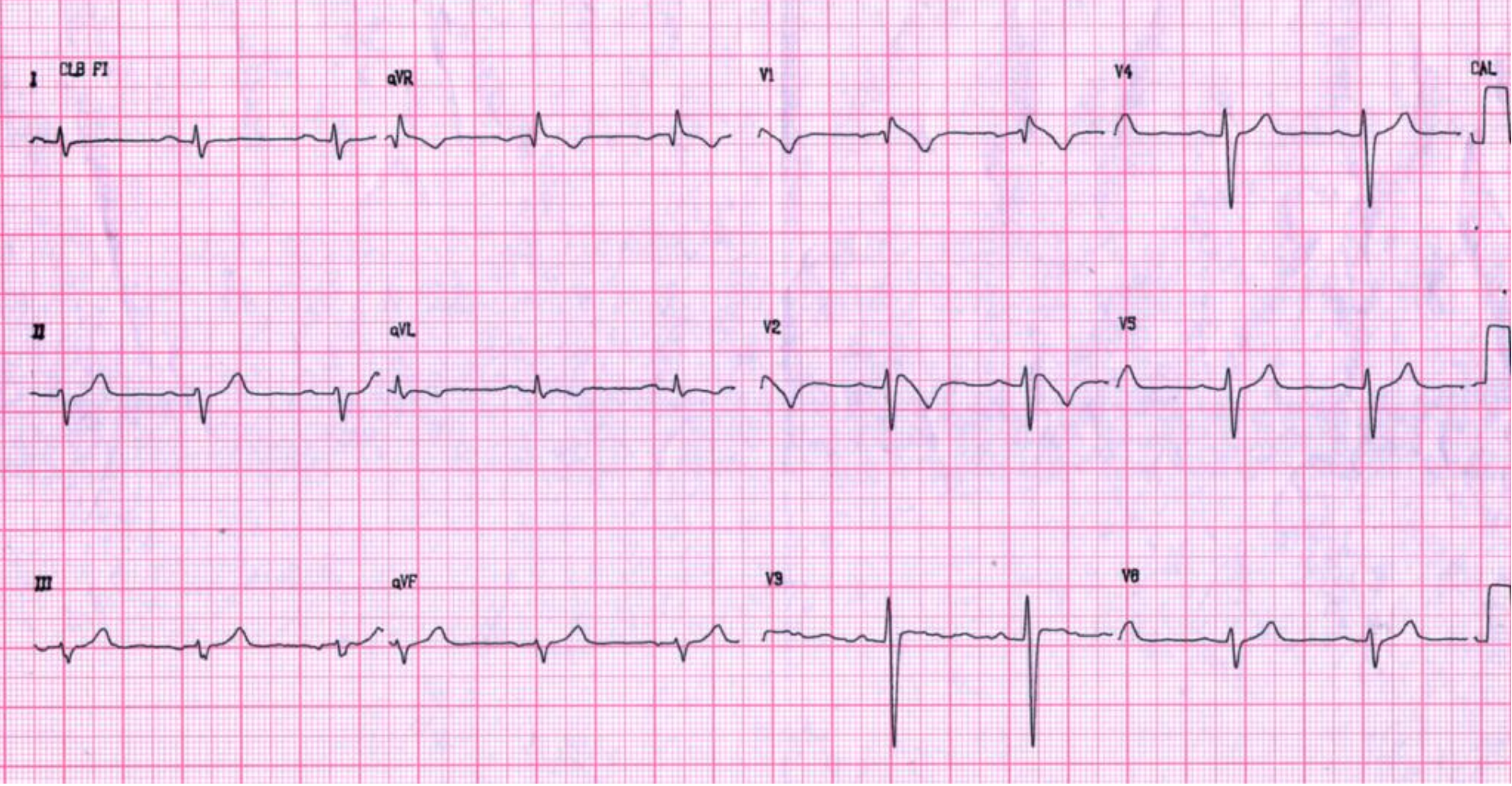
Terceiro ECG da evolução tardia após o procedimento. Paciente assintomático.



Fourth ECG preformed in Emergency room (November 3, 2010) after syncope episode with palpitations an chest discomfort before the episode. The patient informed that four hours before its admission had presented fever (not documented). There are not reference of others events and the familial background is negative for sudden death. Normal serialize biomarkers. CK-MB and troponine.

ECG realizado no dia 03 de Novembro de 2010, quando procurou a emergência após episódio de sincope precedido de palpitações rápidas e desconforto torácico atípico.(ECGs 4 e 5). Adicionalmente, informa que 4 hóras antes da sua admissão havia apresentado febre (não documentada).

Não há relato de episódio prévio semelhante ou história familiar positiva para morte súbita em familiar jovem de primeiro grau. Dosagem seriada de CK-MB e troponina normais.



Fifth ECG: preformed also in November 3, 2010

Quinto ECG: realizado também no dia 03 de Novembro de 2010.

COLLEAGUES OPINIONS

Dr. Samuel Sclarovsky's opinion:

Analisis del fantastico caso enviado por del DR RAIMUNDO BARBOSA

ECG1)abril 2010 registrdo durante un dolor anginotico
Pattern compatible con sindrome SI ,SII ,SIII que va acompaniad por una r' en AVR , r' en V1, ondas S hasta v6, y ausencia de Q en V5 ,V6
El ST deprimido en AVLes un signo muy sensitivo de isquemia inferior (Birenbaum ,Sclarovsky et al ST depression in AVL ;asensitive marker of acute inferior wall infarction European Heart J 1993;14/ 4-7 La onda T puntuda y alta , sin elevacion del segmento ST indica muy probablemente , UNA ISQUEMIA DE PRIMER GRADO , debido a que la arteria derecha recibe circulacion colateral de la anterior descendente
las ondasT invertidas en v2 ,v3 ,y aplanadas en v4 , sugieren reperfusion miocardica por una obstruccion subtotal > mas 75%, de la anterior descendente, el ST elevado en v1 podria ser isquemia del septo derecho en presencia de una R' en v1 por el sindrome SI,SII,SIIIM. Lo que llama la atencion es el notch al principio de la rama descendente de la onda T en V2, Sera un potencial tardio de la septal derecha POR AHORA EL PATRON BASICO DEL ECG , Y UNA OBSTRUCCION REPERFUNDIDA SE LA ANTERIOR DESCENDENTE MAS UNA OBSTRUCCION TOTAL DE UNA ARTERIA DERECHA, CON CIRCULACION COLATRAL DE LA AD , podria explicar este fenomeno electrocardiografico

En el segundo ECG despues de la angioplastia se observa una reelevation del ST en V2 ,V3 con una onda T invertidasugeriendo reperfusion miocardica incompleta ,muy probabl a una embolia periferica
de nuevo se ve el notch en la rama descendente de la onda T en V2 ,v3

El tercer electro me procupa , poque podria ser una normalizacion del ST-T , remodelacion bastante rara , a esta etapa de la evolucion de esta isquemia (no dice la fecha, pero si es antes de los 6 meses , esta presentacion podria ser un seudo normalizacion sugeriendo una obstruccion progresiva y lenta y asimpotomatica del stent(importantismo en descartar por la obstruccion de las dos arteria afectada ,mientrasel area miocardica inferior no esta reperfundida y en sta etapa el eco mostraria una disquinesia inferoposterior

En noviembre se ve este notch que se ve tambien en el ecg anterior antes de la onda T y ahora en la rama descendente, sera el ST elevado una reperfusion incompleta y el sincope una taquicardia polimorfica por reperfusion Birenbaum , Sclarovsky et al polimorphous VT ;arly after acute miocardial infarction Am J os Cardiol1993;71;180

Muy probable un sindrome de Brugada envuelto con isquemias severas reperfudidas y una arteria derecha con circulacion colateral media necrotica pero todavia viva complicada por un sindrome SI ,SII ,SIII
El que va a publicar este caso debera hacer diagnostico diferncial con estos signos que van embarrando un sindrome de btugada
Con gran afecto a los del forum y todavia con la fuerte impresion que me causaron ADRIAN Y LUIS ENRIQUE y el publico de Colombia

Dr. Samuel Sclarovsky

English version Dr Samuel Sclarovsky

I am going to analyze the outstanding case studied by Dr Raimundo Babosa.

The first ECG **shows the basic pattern of a SI ,SII,SIII syndrome , always accompanied by the following signs , R' in aVR , R' in V1 , S waves from V_1 toward V_6 , lack of q waves in V_5 -V_6. Despite it was stressed that the ECG was recorders during an episode of precordial pain, the inverted T with slide ST depression in V_2, and V_3 indicate a repefussed anteroseptal wall. This pattern occurs in the presence of LAD obstruction from 75 to 95%. A complete obstruction of LAD is expressed in the ECG by a ST-T elevation in the same leads. The ST -T depression in aVL indicates an inferior wall ischemia (*BirnbaumY, Sclarovsky S et al ; ST segnent depression in aVL a sensitive marker of inferior wall infarction European Heart J 1993;14 ;4-7*).**

The tall and peaked T waves in III, II, aVF is most probably a first degree inferior wall ischemia. This pattern suggest that the RCA is obstructed and is supplied by collateral circulation. This pattern indicate that the inferior wall is protected, but a slight ST elevation in III suggest the acute ischemia.

The ST elevation in V_1 is probably to an right septal ischemia due to obstruction of the RCA or LAD'S septal perforator arteries.

In the descending limb of the T wave of V_2 is seen a notch, probably a late potential from the right septum.

Second ECG: **after angioplasty and stent shows ST elevation in V1 ,V2 ,V3 with inverted T waves indicating an incomplete reperfusion (complete myocardial reperfusion is manifested by inverted T waves with isoelectric ST segment, and no myocardial reperfusion positive T waves and ST elevation, (THE ELECTROCARDIOGRAM IN ACUTE MYOCARDIAL ISCHEMIC SYNDROMES 1999 MARTIN DUNITS EDITORIAL London Sclarovsky). This pattern was induced most probably due an peripherical LAD emboli. The deep inverted T waves in aVL ,and the increasing peaked T in III is due to the peripherical obstruction of the collateral microcirculation.**

The third ECG recorded later (no date) shows positive T waves in precordial leads , If the recording was performed before 6 months ,must be suspected a pseudo normalization due to a progressive and slow and asymptomatic obstruction of the stent or a progressive normalization of the at waves (less frequent)

The recognition of the situation has clinical importance , taking in consideration that the are 2 areas are involved in the ischemia

The last ECG 3/11 recorded after syncope shows again in the early stages of incomplete reperfusion , V3 is positive

the syncope could be induced by a non sustained PVT occurring during the very early stages of reperfusion (Birenbaum Y, Sclarovsky S ; POLIMORPHOUS VENTRICULAR TACHYCARDIA EARLY AMI AM J OF CARDIOLOGY 1993; 71;745).

I think if the author will publish this case as Brugada syndrome appearing during acute ischemia, must consider this discussion as a high probability

My best regard of all our friend

Shalom
Samuel Sclarovsky

Prof, Dr. Bernard Belhassen's opinion

Superb case indeed. This patient has associated CAD and Brugada-ECG pattern. The first ECG may even suggest this association.
Apparently, he did not have a myocardial infarction (we have no data on LV function) so that I do not think that the palpitations followed by syncope is due to post-MI reentrant VT.
Of course, the most important diagnosis is to rule out the possibility of Brugada-related paroxysmal VT/VF. Another diagnosis to be ruled out is rapid supraventricular tachyarrhythmias frequently associated to Brugada syndrome.
So, practically, what we need first (in case ECG monitoring failed to bring any information) is to perform a comprehensive EPS including both atrial and ventricular stimulation.
It is important to perform repetition of double and triple ventricular extrastimulation in these patients and not avoiding shortest coupling intervals.
My feeling is that polymorphic VT will be induced (sustained ?? nonsustained ???).
I will be happy to discuss the case later after getting the EPS results.
Good luck.

Prof. Belhassen, Bernard
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Portuguese

Fantástico caso. Este paciente tem associação de insuficiência coronária e padrão ECG Brugada.
O primeiro ECG pode sugerir esta associação.
Aparentemente, ele não tem IM (não temos informações sobre a função do VE) então eu não penso que as palpitações seguidas por síncope sejam ocasionadas por TV reentrante pós-IM.
É claro que o diagnóstico mais importante é afastar a possibilidade de TV/FV relacionadas a Brugada. Um outro diagnóstico que deve ser afastado são taquiarritimias supraventriculares frequentemente associadas a Síndrome de Brugada.
Então, praticamente, o que necessitamos primeiro (em caso que a monitorização fracasse em nos dar alguma informação) é realizar um estudo eletrofisiológico com estimulação tanto atrial quanto ventricular.
É importante realizar a repetição com extraestímulos duplos e triplos nestes pacientes com intervalos cada vez menores.
Meu palpite é que as TVP serão induzidas (em forma sustentada? Ou não sustentada?).
Ficaria feliz de discutir o caso mais tarde após os resultados do estudo eletrofisiológico.
Boa sorte.
Prof. Bernard Belhassen.

Hi
Please allow me to respectuflly disagree with Professor Belhassen.
Type 1 Brugada ECG pattern (spontaneous) + Syncope is indication Class I for ICD (despite of EPS results).
If the EPS is negative for VT-VF induction, are you not going to implant an ICD?
The FINGER study, the largest multinational study on Brugada syndrome that I am aware of, has demonstrated the lack of value of the EPS as a predictor of arrhythmic events.
I would recommend an ICD with no further testing.
Best personal regards
Adrian Baranchuk MD FACC
Queen's University

Hola Por fabor me permitan respetuosamente discordar del Profesor Belhassen. El patrón ECG espontáneo tipo 1 associado a síncope es indicación clase I de ICD a despecho del resultado de el estudio electrofisiológico. Si el estudio electrofisiológico resulta negativo para inducción TV/VF no iríamos a implantarle un CDI?.
El mayor estudio multinacional Europeo llamado FINGER há concluido que el estudio electrofisiológico no tiene valor preditivo para eventos arrítmicos.
Yo le recomendaria un CDI sin realizar futuros estudios.
Saludos personales
Adrian Baranchuk MD FACCQueen's University

1. **Probst V, Veltmann C, Eckardt L, Meregalli PG, Gaita F, Tan HL, et al. Long-term prognosis of patients diagnosed with Brugada syndrome: Results from the FINGER Brugada Syndrome Registry. Circulation. 2010 Feb 9;121(5):635-643.**

Prof, Dr. Bernard Belhassen's reply to Dr Barachuk and to Prof Wilde

According to my experience[1;2], EP-guided therapy with quinidine is also a very valuable mode of management of symptomatic patients with Brugada syndrome. I have not yet observed a single Brugada patient with inducible SPVT-VF prevented by quinidine who developed an arrhythmic event during chronic quinidine TX (during follow-up periods up to 17 years). In contrast, I have seen in my experience or that of others several Brugada patients who died wearing an ICD or almost died following ICD implantation. I also have seen several patients with ICD's becoming crazy. Finally, I have seen many patients who had severe ICD-related infection problems.
BB.

Portuguese
De acordo com minha experiencia a terapia com quinidina guiando o estudo eletrofisiológico é uma maneira muito adequada do manuseio de pacientes portadores da síndrome de Brugada . Eu não he observado um único paciente que possa ser induzido quando administrado previamente quinidina em um seguimento de 17 anos. Contrariamente eu tenho observado na minha experiência falecimentos pós implante do CDI com mortes logo a seguir. Eu também tenho observado vários pacientes com ICD ficando locos. Finalmente, eu tenho visto muitos pacientes com sérios problemas de infecção após o implante do CDI.

1. **Belhassen B, Glick A, Viskin S. Excellent long-term reproducibility of the electrophysiologic efficacy of quinidine in patients with idiopathic ventricular fibrillation and Brugada syndrome.Pacing Clin Electrophysiol. 2009 Mar;32(3):294-301.**
2. **Belhassen B, Glick A, Viskin S. Efficacy of quinidine in high-risk patients with Brugada syndrome. Circulation. 2004 Sep 28;110:1731-1737.**

Another “philosophic” intervention of genial Prof, Dr. Bernard Belhassen

Dear Andres:
You certainly remember David vs Goliath !
My fight as far as the Quinidine/Brugada issue is very similar.
Of course I am DAVID !!!!!!!!!!
The ICD supporters are always very happy to find quinidine when their ICD patients developed arrhythmic storms. Once they have cured the storms with quinidine, I am asking them: ""So, now, as you saw the fantastic efficacy of quinidine, you probably will not replace the device in your patient and will continue quinidine??".
Of course you do know the answer: Impossible to get them changing their mind and their misconception of management of this arrhythmia.
Best regards
BB

Querido Andrés: tu ciertamente recuerdas David contra Golias!
Mi lucha tan lejos cuanto Quinidina/brugada son similares. Por supuesto yo soy David!!
Cuando en un paciente a quien se le implantó in CDI desarrolla una tormenta eléctrica ellos curan este evento con quinidina, entonces yo les pregunto a ellos: si la quinidina tiene esta fantastica propiedad ustedes probablemente iran a remplazar el caro CDI por quinidina?. Por supuesto vos conoces la respuesta. Imposible cambiar sus mentes e sus conceptos errados en el manejo de esta enfermedad arritmica.
Saludos
BB

Thank you so very much dear Andres for this show case.
I total agree with Dr. Raimundo's Dx. This is a high risk individual with CAD + BrS. From his ECG I see the scar tissue formation in his heart. SAECG could be positive for late potentials. Fever, a full stomach meal and many drugs can trigger BrS-related cardiac events though events may never return after ICD implantation's. Nevertheless following the guideline for high risk pt management is a safe way to recommend...
Best regards,
Li

Li Zhang, MD
Associate Professor, Jefferson Medical College
Director, Cardiovascular Outcomes Research
Main Line Health Heart Center
Lankenau Medical Center
558 MOB East
100 Lancaster Avenue
Wynnewood, PA 19096

Portuguese
Muito obrigado meu querido Andrés, por este caso show.
Concordo totalmente com Dr Raimundo. Este é um paciente de alto risco com Coronariopatia + Brugada. No ECG eu vejo a formação de cicatriz em seu coração. Um ECG de alta resolução poderia me mostrar potenciais tardios. A febre um estomago cheio de alimento, assim como várias drogas podem ser deflagadores de eventos na síndrome de Brugada, apesar que estes eventos podem nunca repetir-se depois do implante de CDI. Seguindo as guias de pacientes com alto risco, recomendo esta conduta.
Li

Dear Andrés, this is certainly a BrS case (associated with CAD as has been proven by CAG). There is a left axis deviation and the PQ interval is wide suggesting that he is a carrier of a SCN5a mutation (this is almost sure).
So, I don't doubt the diagnosis of BrS and with the presentation of syncope one should strongly consider ICD implant. The presence of critical CAD would tip the balance even further into that direction.
You could consider quinidine but who knows what happens in combination with ischemia then. A final important recommendation is that he should be monitored at the time he has fever, sdo he should know that very best
Arthur

Prof. Dr. Arthur Arnold Maria Wilde M.D. PhD.
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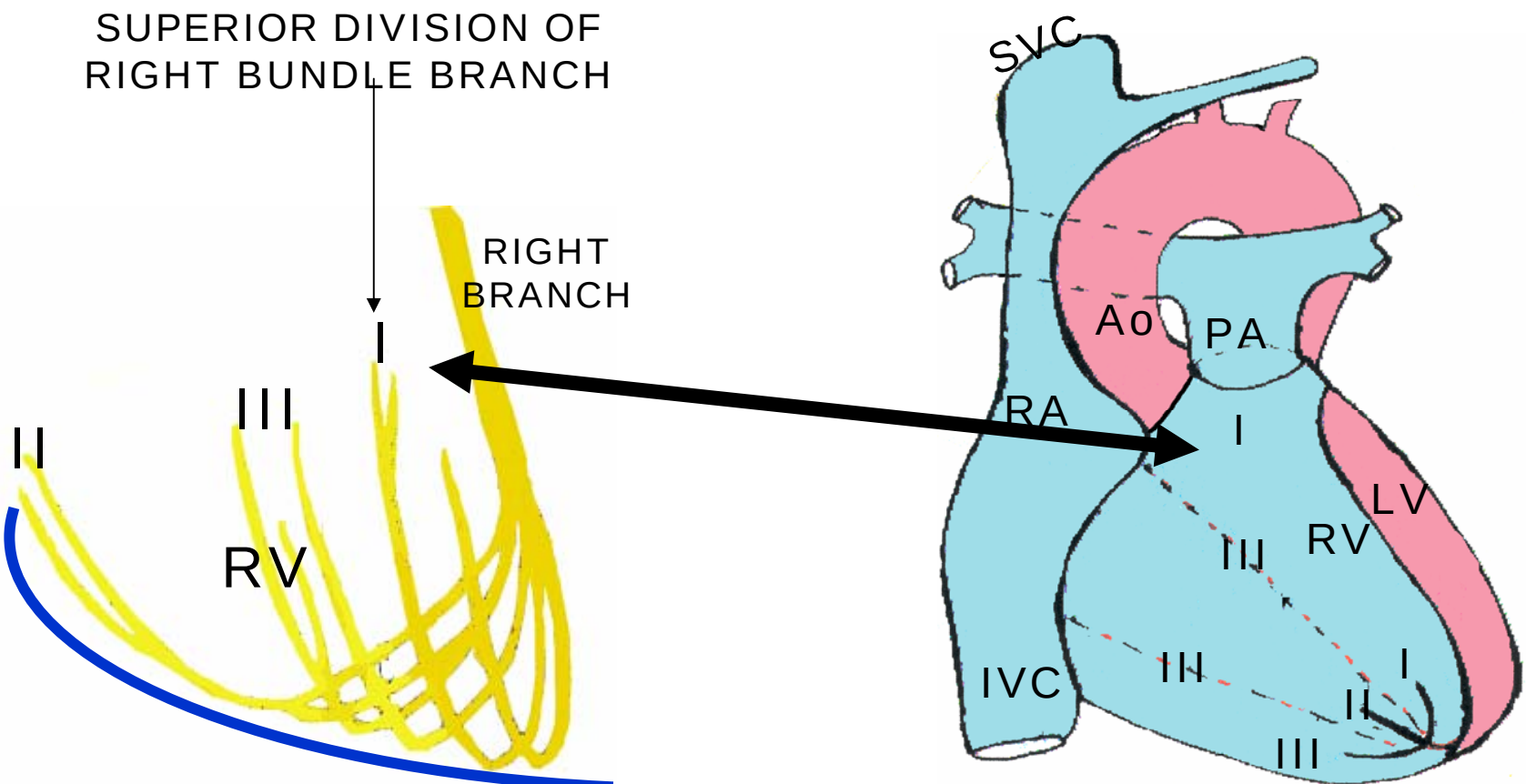
Professor Arthur Wilde was the main research of the first *consensus about the Brugada Syndrome*

Estimado Andrés: este ciertamente es un caso de sindrome de Brugada asociado a enfermedad coronaria como lo ha provado el cateterismo.
Existe extremo desvio del eje elétrico, y el intervalo PR está prolongado lo que sugiere que el paciente es portador de la mutación (esto es casi seguro).

Entonces, yo no dudo del diagnóstico de síndrome de Brugada el cual en presencia de síncope deberia nos hacer inclinar para el implante de un CDI. La presencia de lesión crítica en la LAD balaceará la conducta en esta dirección.
Podriamos considerar quinidina mas no sabemos lo que ocurríria en combinación con enfermedad coronaria.
Una recomendación final es monitorizar la fiebre
Arthur

Andrés Ricardo Pérez Riera commentaries

ECG 1: Sinus rhythm, HR 83bpm, P axis + 25º to front, P duration:110ms.
PR interval 185ms.
QRS axis: near - 90º (extreme superior deviation of QRS axis: isodiphasic in I and negative in aVF) prominent final R wave in aVR: **Right End Conduction Delay (RECD)** on territory of RVOT.
R wave voltage in aVR = **4,3mm**.(aVR sign)
R<S in left leads V_5-V_6. Cause? Right Ventricular Enlargement or RVH? or **RECD** by superior or sub-pulmonary division of right bundle branch? The last one is the correct diagnosis.
Additionally, we have not criteria of type 1 Brugada ECG pattern because J point and ST segment elevation on right precordial leads < 2mm. Negative asymmetrical T wave from V_1 to V_3.



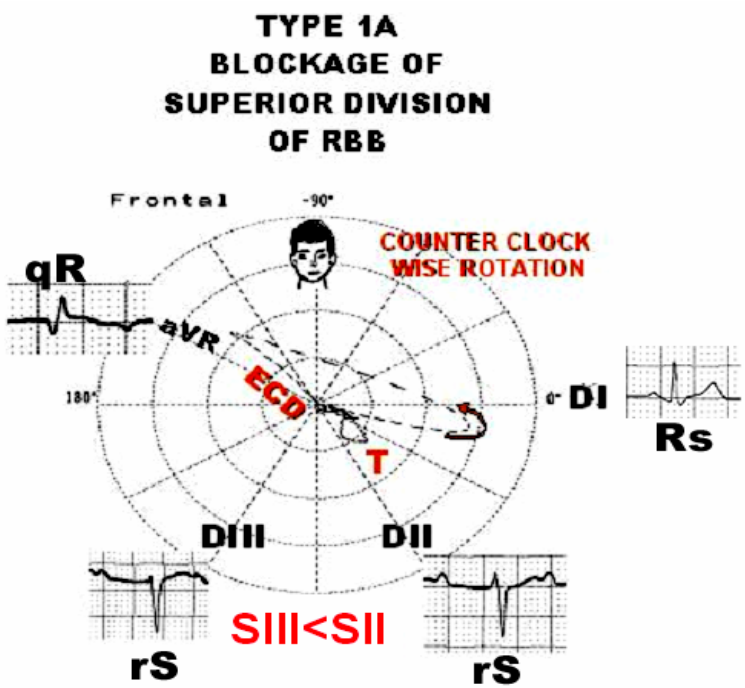
What is the aVR sign? Answer: a prominent R wave in lead aVR (aVR sign) Babai et al [1] show that there is a significant correlation between risk for development of arrhythmic events in BrS and prominent R wave in lead aVR. The feature may reflect more right ventricular conduction delay and subsequently more electrical heterogeneity, which in turn is responsible for a higher risk of arrhythmia. The aVR sign was defined as R wave voltage ≥ 3mm (0.3 mV) or R/q ratio ≥ 0.75 in lead aVR.

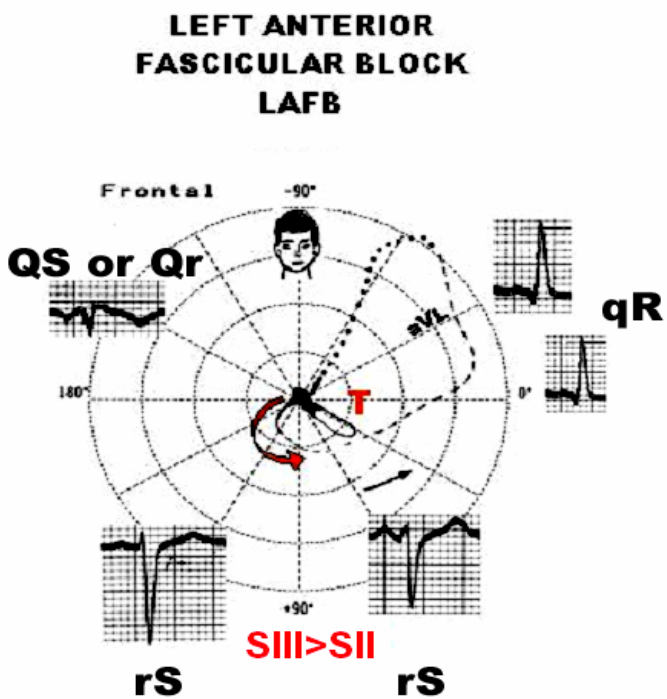
Prof Arthur affirm that *"There is a left axis deviation and the PQ interval is wide suggesting that he is a carrier of a SCN5a mutation (this is almost sure)".* I Respectfully I disagree with Prof. Wilde. Why? Because:
1)First the PR or PQ interval is normal=185ms (< 200ms), and
2)Second the extreme QRS axis deviation is not consequence of Left Anterior Fascicular Block (LAFB) indicative of HV prolongation and probable SCN5A mutation. The QRS axis is near -90º but in right superior quadrant on frontal plane. Additionaly I and aVL have not qR pattern and SII>SII.Consequently, this patient have not LAFB. This patient have righ end conduction delay by the superior or supulmonar division(or contingent) of righ bundle branch block. The QRS axis is not on left superior quadrant on frontal plane. The QRS axis is located on right superior quadrant
The table on the next slide summarize this concepts:

1. **Babai Bigi MA, Aslani A, Shahrzad S. aVR sign as a risk factor for life-threatening arrhythmic events in patients with Brugada syndrome. Heart Rhythm. 2007 Aug;4(8):1009-12.**

ECG/VCG DIFFERENTIAL DIAGNOSIS ON FRONTAL PLANE BETWEEN SUPERIOR DIVISIONAL BLOCK OF RIGHT BUNDLE BRANCH (SDBRBB) AND LAFB

	SDBRBB	LAFB
Initial 10 to 20 ms vector	Downward ant to left	Downward ant toright
Leads I and aVL QRS pattern	R or RS	qR
II/III relation	SII>SIII	SII<SIII
R wave aVR lead	Prominent	May be low or absent
QRS rotation on frontal plane	CCW or others	CCC
End Conduction Delay	On top right quadrant	On top left quadrant





Second ECG analysis

Sinus rhythm, HR 55bpm, QRS axis -95º(on top right quadrant). Additionally we have an association never described before:

1. Type 1 Brugada ECG pattern in V1-V2 and Type 1
2. Wellens syndrome pattern in V3 (plus-minus T wave).

Fantastic ! We have two syndromes together: The Brugada syndrome associated with the Type 1 Wellens syndrome!!! (Tremendous coincidence: Brugada was pupil of Prof Wellens!!!).
Please Dr Andrés Pérez-Riera(self question) could you explain what is the Wellens syndrome?

WELLENS' SYNDROME; LAD CORONARY T-WAVE SYNDROME OR "LEFT ANTERIOR DESCENDING T WAVE SYNDROME".

Definition – ECG pattern of T waves in the precordial leads that are associated with a critical stenosis of the proximal left anterior descending coronary artery (LAD). The syndrome is also referred to as LAD coronary T-wave syndrome. He described the “Wellens Syndrome” is a characteristic, easy to identify cardiac syndrome, which indicates a critical, high-grade occlusion of the LAD artery. If not identified, and properly treated, the mean time from onset of symptoms to wide scale anterior wall myocardial infarction is 8.5 days (Conover, 1994)

Differential characteristics of Wellens syndrome include:

1. Prior history of chest pain

2. Progressive, symmetrical, deep T wave inversion in V_2 and V_3.

3. Plus-minus T waves + inversion of the terminal portion of the T waves in leads V_2 - V_4 are near-pathognomonic of that syndrome

4. Deep T-wave inversion is seen, especially in V_2 and V_3 leads

Cases can be classified according to the ECG pattern into type 1 (biphasic T waves) or type 2 (deeply inverted T waves, especially in leads V2 and V3). Biphasic T waves in leads V_2 and V_3 (Type 1) or symmetric, often deeply inverted T waves in leads V_2 and V_3 (Type 2);
Rarely changed from type 2 to type 1 during observation is possible, and the coronary lesion located in the middle rather than the proximal part of the LAD
Characteristic critical myocardial ischaemic with minor ECG changes[2]
The T-wave abnormalities are persistent and may remain in place for hours to weeks[3].

1. **Kardesoglu E, Celik T, Cebeci BS, Cingozbay BY, Dincturk M, Demiralp E. Wellens' syndrome: a case report.J Int Med Res. 2003;31:585-590);**
2. **Goor Y, Magal R, Goor O, Frimerman A, Cabili S.Critical myocardial ischemia: minor electrocardiograph changes--Wellens' syndrome.Isr Med Assoc J. 2003;5:129-130.**
3. **Rhinehardt J, Brady WJ, Perron AD, Mattu A. Electrocardiographic manifestations of Wellens' syndrome. Am J Emerg Med. 2002; 20:638-643.**

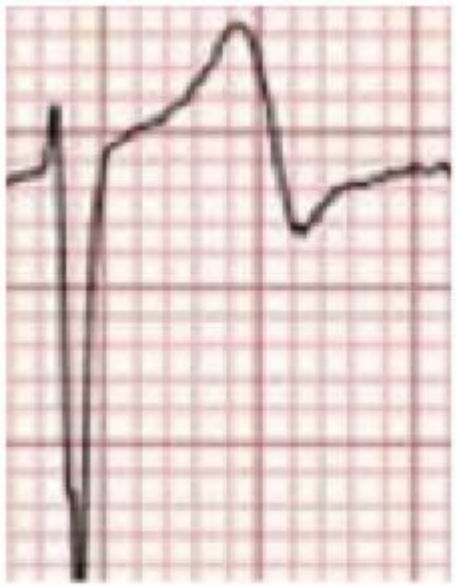
History of unstable angina chest pain without serum marker abnormalities: Little or no enzyme and troponin elevation[1]. Patients lack Q waves[2].
Little or no ST elevation[3].
Normal precordial R-wave progression: No loss of precordial R waves. We and others authors observed a variant with prominent R waves in V_2 to V_4 associated sometimes with initial q wave[4].
Little or no enzyme elevation
Note these EKG changes usually occur during a pain-free interval when other evidence of ischaemic or unstable angina may be absent;
The natural history of Wellens' syndrome is anterior or Antero-septal wall acute myocardial infarction;
Wellens' Warning (Terminal Inversion of T waves anteriorly)
"Wellens' warning" Published by de Zwaan C, Bar FW, Wellens HJ. in 1982 the finding that Terminal Inversion of T waves anteriorly associated with critical proximal LAD lesions. These patients should not have stress tests but rather emergent Cardiac Catheterizations.

1. **Tandy TK, Bottomy DP, Lewis JG. Wellens' syndrome. Ann Emerg Med. 1999; 33:347-351.**
2. **Kahn EC, Keller KB. Wellens' syndrome in the emergency department.J Emerg Nurs. 1991; 17:80-85**
3. **(Conover M. Wellens' syndrome: identification of critical proximal left anterior descending stenosis. Crit Care Nurse. 1990; 10:30-36.**
4. **Riera AR, Ferreira C, Ferreira Filho C, Dubner S, Schapachnik E, Uchida AH, et al. Wellens syndrome associated with prominent anterior QRS forces: an expression of left septal fascicular block? J Electrocardiol. 2008 Nov-Dec;41(6):671-4**

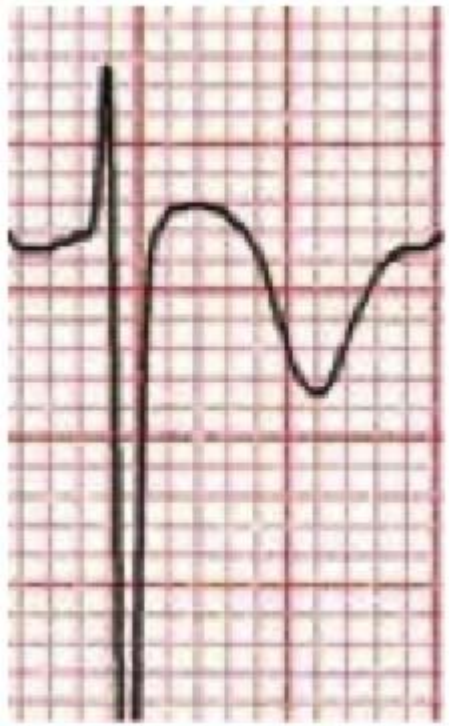
Second ECG preformed immediately after Percutaneous Coronary Intervention Has similar pattern of T wave only in V3: Plus/minus

Hein J Wellens

Type 1



Type 2



1. de Zwaan C, Bär FW, Wellens HJ. Characteristic electrocardiographic pattern indicating a critical stenosis high in left anterior descending coronary artery in patients admitted because of impending myocardial infarction. Am Heart J. 1982 Apr;103(4 Pt 2):730-6.

Third ECG late evolution after procedure. Patient asymptomatic

Very similar only Brugada type 2 J point and ST segment elevation = 2mm (0.2mV) with saddleback appearance, and remains at least 1 mm above the isoelectric line, followed by positive T wave.

Fourth ECG preformed in Emergency room (November 3, 2010) type 1 Brugada ECG pattern only in V2.

Fifth ECG: preformed also in November 3, 2010 similar.

Conduct:

1. CDI or quinidine(polemic issue)
2. Pharmacotherapy: , as the stent is a foreign object (not native to the body), it incites an immune response. This may cause scar tissue (cell proliferation) to rapidly grow over the stent. In addition, there is a strong tendency for clots to form at the site where the stent damages the arterial wall. Since platelets are involved in the clotting process, patients must take antiplatelet therapy afterwards, usually clopidogrel for six months and aspirin indefinitely. In order to reduce the treatment, new generation of stent has been developed with biodegradable polymerHowever, the antiplatelet therapy may be insufficient to fully prevent clots; these and the cell proliferation may cause the standard ("bare-metal") stents to become blocked (restenosis). Drug-eluting stents were designed to lessen this problem; by releasing an antiproliferative drug (drugs typically used against cancer or as immunosuppressants), they can help avoid this *in-stent restenosis* (re-narrowing).
3. Caution with fever: Since temperature affects permeability, temperature change forces the Na+ channel and other channels to modify their functional state. INa+ kinetics depends strongly on temperature[1].

1. **Nagatomo T, Fan Z, Ye B, Tonkovich GS, January CT, Kyle JW, Makielski JC. Temperature dependence of early and late currents in human cardiac wild-type and long Q-T DeltaKPQ Na+ channels. Am J Physiol 1998; 275:H2016-H2024**.

Thus, an increase of 10ºC increment the voltage or width by a factor of 1.3 to 1.6 and increases the time of opening and the number of times that the channel is opened by a factor of three. Activation and inactivation kinetics for early INa+ are twofold faster at higher temperature, and shift activation and steady-state inactivation**1**. Then, the fever is considered triggers to PVT/VF in the Brugada Syndrome (BS) jointly with other causes that causing modifications in the degree of elevation of the J point and of the ST segment. The other factors capable to get worse the ventricular repolarização are: *antimalarial agents; tricyclic antidepressants; class IA (ajmaline and procainamide) and 1C (flecainide, pilsicainide) anti-arrhythmic agents; hyperglycemia; nocturnal bradycardia by vagal predominance; alcohol consumption; mental stress and cocaine use*.

In BrS, fever is associated with a greater chance of tachyarrhythmic events; this suggests that the increase in temperature affects the Na+ channel conductance. Mutations in a cardiac sodium channel gene have been linked to this syndrome and some experimental data suggest that the dysfunction of the mutated channel can be temperature sensitive**2**.

Dumaine et al**3.** hypothesized that at more physiological temperatures; the missense mutation may change the gating of the sodium channel such that the net outward current is dramatically augmented during the early phases of the right ventricular AP. The authors tested this hypothesis by expressing Thr1620Met in a mammalian cell line, using the patch-clamp technique to study the currents at 32 degrees C. They concluded that Thr1620Met current decay kinetics are faster when compared with the wild type at 32 degrees C. Recovery from inactivation was slower for Thr1620Met at 32 degrees C, and steady-state activation was significantly shifted. These findings explain the features of the ECG of BrS patients, illustrate for the first time a cardiac INa+ channel mutation of which the arrhythmogenicity is revealed only at temperatures approaching the physiological range, and suggest that some patients may be more at risk during febrile states.

1. Gonzalez Resolo JM, Hernandez Medina A, et al. Recurrent ventricular fibrillation during febrile illness in a patient with the Brugada syndrome. Rev Esp Cardiol 2000; 53:755-757
2. Saura D, Garcia-Alberola A, Carrillo P, Pascual D, Martinez-Sanchez J, Valdes M. Brugada-like electrocardiographic pattern induced by fever. Pacing Clin Electrophysiol. 2002; 25:856-859.
3. Dumaine R, Towbin JA, Brugada P, et al. Ionic mechanisms responsible for the electrocardiographic phenotype of the Brugada syndrome are temperature dependent. Circ Res 1999; 85:803-809.

Nagatomo et al[1] characterized early INa+ (the peak and initial decay) and late INa+ of the wild-type hH1 channel and a mutant channel (DeltaKPQ) associated with LQT3.
Channels were stably transfected in HEK-293 cells and studied at 23 and 33 degrees C using whole cell patch clamp.
Activation and inactivation kinetics for early INa+ were two fold faster at higher temperature for both channels and shifted activation and steady-state inactivation in the positive direction, especially for DeltaKPQ. For early INa+ (<24 ms), DeltaKPQ decayed faster than the wild type for voltages negative to -20 mV but slower for more positive voltages, suggesting a reduced voltage dependence of fast inactivation.
Late INa+ at 240 ms was significantly greater for DeltaKPQ than for the wild type at both temperatures. The majority of late INa for DeltaKPQ was not persistent; rather, it decayed slowly, and this late component exhibited slower recovery from inactivation compared with peak INa+.
Kinetic changes for early and peak INa+ for DeltaKPQ compared with the wild type at both temperatures were:
Reduced voltage dependence of steady-state inactivation with no difference in midpoint;
Positive shift for activation kinetics, and;
More rapid recovery from inactivation.

1. Nagatomo T, Fan Z, Ye B, Tonkovich GS, January CT, Kyle JW, Makielski JC. Temperature dependence of early and late currents in human cardiac wild-type and long Q-T DeltaKPQ Na+ channels. Am J Physiol 1998;275:H2016-H2024.

There are references of ST-segment elevation, spontaneous T wave alternans1, PVCs, severe2 PVT with syncope and incessant MVT3 with fatal electrical storm related in febrile states

- **Morita H, Nagase S, Kusano K, Ohe T. Spontaneous T wave alternans and premature ventricular contractions during febrile illness in a patient with Brugada syndrome. Cardiovasc Electrophysiol. 2002; 13:816-818.**
- **Porres JM, Brugada J, Urbistondo V, Garcia F, Reviejo K, Marco P. Fever unmasking the Brugada syndrome. Pacing Clin Electrophysiol. 2002; 25:1646-1648**.
- **Dinckal MH, Davutoglu V, Akdemir I, Soydinc S, Kirilmaz A, Aksoy M. Incessant monomorphic ventricular tachycardia during febrile illness in a patient with Brugada syndrome: fatal electrical storm. Europace. 2003; 5:257-261**.