Wide complex tachycardia in elderly man with previous anterior MI

Spanish

Presentación del caso

Paciente de 78 años, sexo masculino, con antecedentes de infarto de cara anterior ocho años atrás. Refiere que en la oportunidad fue tratado medicamente sin realización de PCI. Niega diabetes, dislipidemia o disfunción renal. Medicado de mucho tiempo con muy baja dosis de maleato de enalapril (apenas 5mg/dia) + Carvedilol 3.125 2 x dia y espironolactona 25mg/dia en virtud de ser poco tolerante a la medicación (al aumentar la dosis siente mareos atribuido a hipotensión arterial).

El primer ECG-1 es un trazado antiguo que el paciente había realizado en marzo del 2015.

Acude al servicio de urgencia caminando con queja de palpitaciones rápidas precordiales de inicio súbito y acompañado de opresión precordial difusa irradiada al cuello. El examen físico revelaba P.A. 100/60 mmHg, F.C. 185 x' y Sat O2 94% con buena tolerancia a la arritmia, sin trastorno hemodinámico. No hay referencia de ondas en cañón en el cuello, variaciones en la intensidad del primer ruido o variaciones de la PA de latido a latido.

Se realiza el ECG en la sala de urgencias (ECG-2) y se decide intentar la reversión de la arritmia con amiodarona endovenosa (3 ampollas EV de 150mg en 100 cc. de Ringer). Poco antes de concluir el goteo, aproximadamente 30', revirtió a ritmo sinusal (ECG-3). Los parámetros hemodinámicos no sufrieron modificaciones. Biomarcadores no alterados. Se realiza Ecocrdiograma transtorácio

Preguntas:

1. El evento de QRS ancho (ECG-2) es supra ventricular o ventricular? Analice argumentos.
2. Cual es la conducta adecuada?

Dr. Luciano Pereira, MD – Ciudad del Este, Paraguay

English

Case report

Male, 78-year-old patient, with history of anterior infarction eight years ago. He reported that at the time he was pharmacologically treated, with no PCI being made. He denied smoking, diabetes, dyslipidemia or renal dysfunction. Medicated since the MI episode with a very low dose of enalapril maleate (just 5 mg/day) + carvedilol 3.125 twice a day + spironolactone 25 mg/day as he had low tolerance to the medication (when the dose was increased, he felt dizziness attributed to hypotension).

ECG-1 is an old tracing of the patient from March 2015.

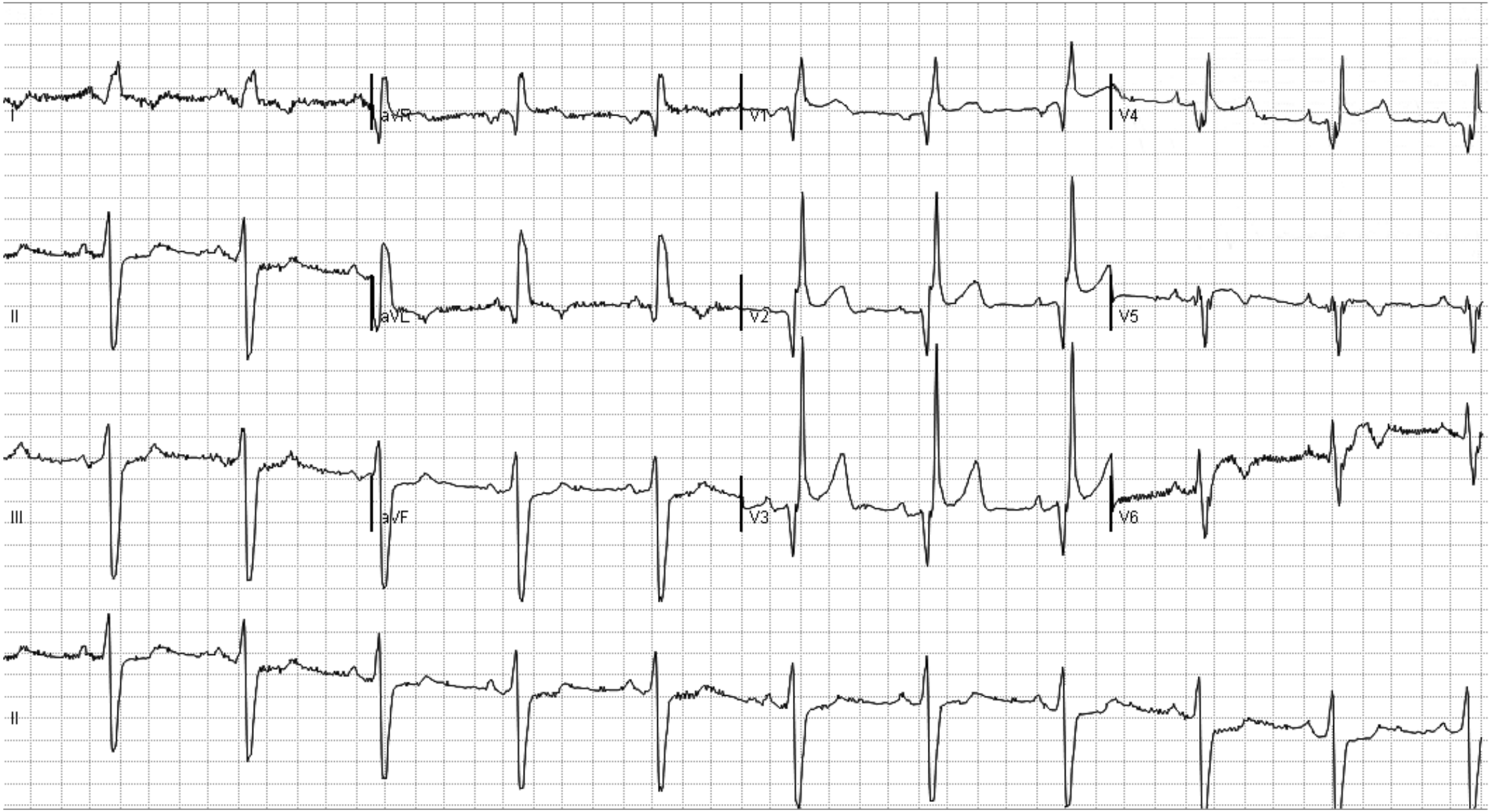
He went to the ER walking, with a complaint of fast precordial palpitations of sudden onset, and accompanied by diffuse precordial oppression radiated to the neck. The physical examination revealed BP 100/60 mmHg, HR 185 bpm and SatO2 94% with a good tolerance to arrhythmia, with hemodynamic disorder. There is no reference to cannon waves in the neck, variations in the intensity of the first sound or BP variations from beat to beat. ECG-2 was made in the ER, and arrhythmia reversion was attempted by IV amiodarone (3 IV ampoules in 100 cc of Ringer by dripping). Shortly before concluding the dripping, approximately 30', he reversed into sinus rhythm (ECG-3). Hemodynamic parameters did not undergo changes. Negative biomarkers.

Questions:

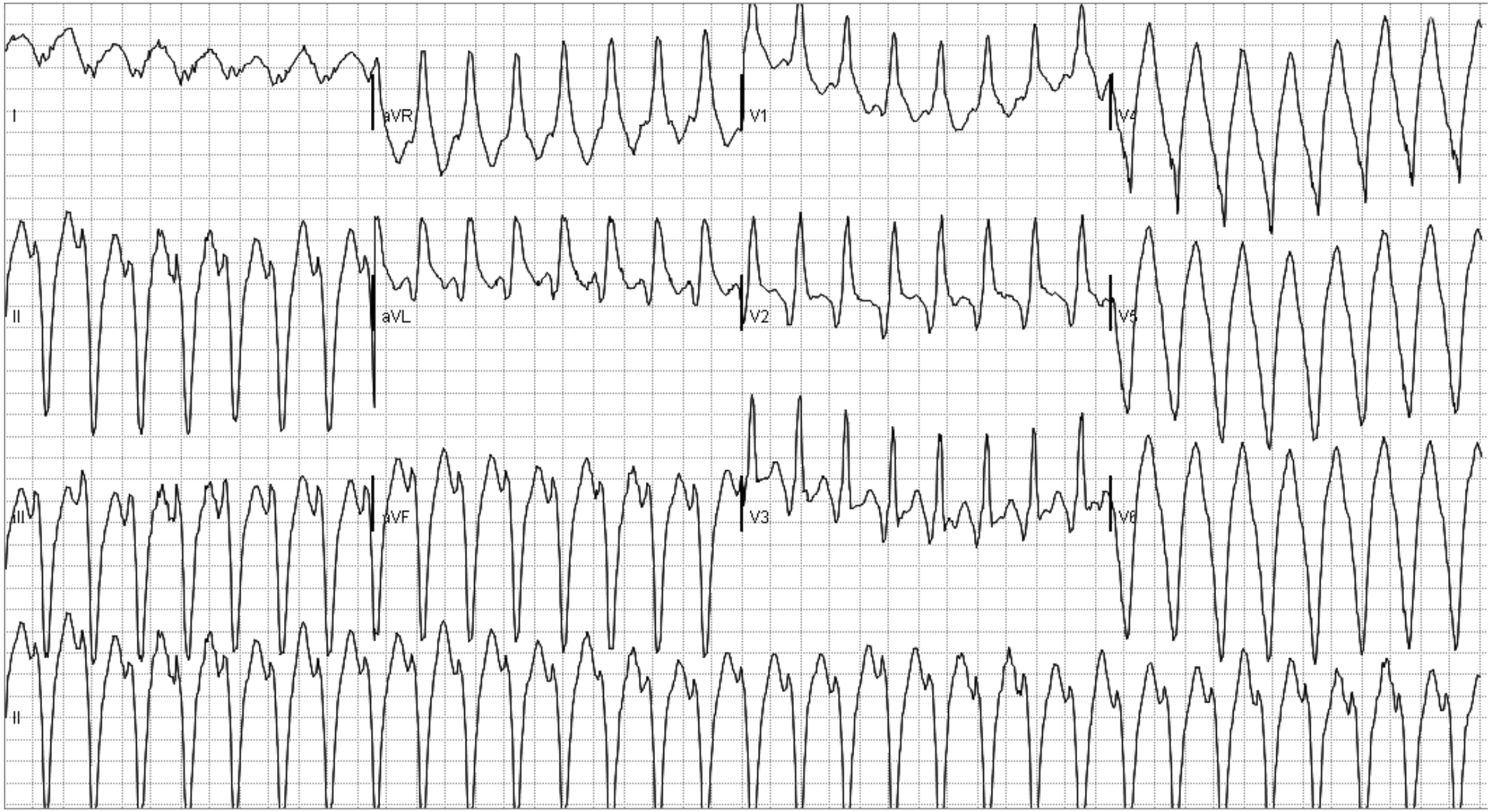
1. Is the event wide QRS (ECG-2) supraventricular or ventricular? Analyze your arguments.
2. What is appropriate approach?

Dr. Luciano Pereira, MD – Ciudad del Este, Paraguay

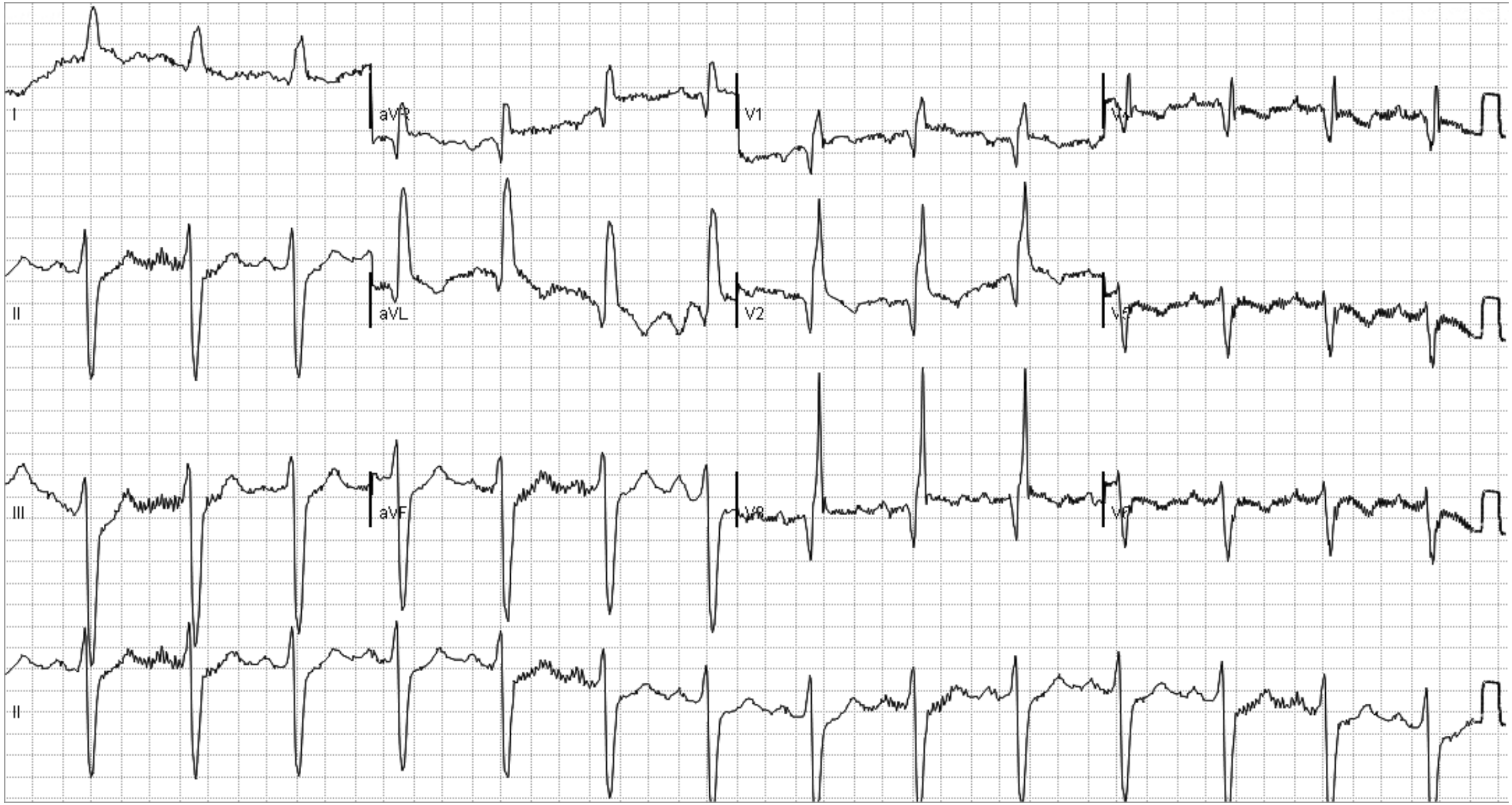
ECG-1



ECG -2 at ED admission



ECG -3 after reversion



Transthoracic Color Doppler Echo Study

First and last name: AV; **Age:** 78 years. **Weight:** 65 kg; **Height:** 1.62 m; **Body surface:** 1.69 m2. **Date:** August 31, 2016.

		Measurements	Normal values (mm)
Left atrium		37	< 40
volume		59 ml (34 ml/m)	Up to 34 ml/m2.
Aorta: Root		30	<40
Right ventricle		28	< 30
IV Septum		6	6-10
Posterior wall		9	6-10
Left ventricle	**EDD**	62	40-59
	ESD	51	Variable
	EDV	193	
	ESV	124	

Left ventricular function

Ejection fraction: 36% (normal value ≥53%) (Teicholz)

Shortening fraction: 17% (normal value >26%)

Simpson: 16%

Strain 2D: 24%

Rhythm: regular

Valves:

- Mitral, tricuspid and pulmonary: no structural alterations
- Aortic: thicker, sclerotic.

Heart dimensions: mild LV dilatation. Parietal thickness: thin IV septum. Parietal motility: globally decreased.

Pericardium: normal. Masses: absent.

TAPSE: 18 mm. Global strain: -4.8%. With overall deficit and area of anterior fibrosis (2 and 1).

Doppler: Type II decreased relaxation pattern (E/e' ratio). Cardiac index: 1.22 l/min/m2. Mitral valve insufficiency. Eccentric jet. MV VTI/LVOT VTI ratio: 1.6. Tricuspid valve insufficiency. Mx V: 2.72 m/s. Mx grad: 29.7 mmHg. Systolic pulmonary artery pressure: 34.7 mmHg.

Conclusions Severely decreased systolic function; type II diastolic dysfunction; mild dilatation of the LV with global diffuse hypokinesis; globally decreased strain, with area of anterior fibrosis; Severe mitral valve insufficiency; preserved right ventricular function; mild tricuspid valve insufficiency; pulmonary hypertension

Ischemic heart disease in dilated phase with severe hemodynamic repercussion.

Colleagues opinions

I have a single diagnosis for this case: Ventricular tachycardia originating from the infarcted area. There are major differences in QRS morphology in several ECG leads that render the diagnosis of SVT almost impossible.

Bernard **Belhasen** MD PhD Israel

Role

Head of the Electrophysiology Laboratory, Department of Cardiology, Tel-Aviv Medical Center, Tel-Aviv. Israel.

Unit

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Hello. Nice case. One would expect ventricular tachycardia in a patient with previous MI and enlarged left ventricle with depressed LV function. Baseline ECG shows Q waves indicating an old large anterior MI, most probably a proximal LAD occlusion at the time of the MI, especially when there is also LAFB + RBBB. There is QRS fragmentation as a sign of scar tissue in the precordial leads. LV mass is increased (deep S in III and high R in aVL). The tachycardia is regular, I can't see fusion beats, escape beats or P waves (not sure about that because of artifacts). The QRS axis in the frontal plane differs from that in sinus rhythm (aVR is positive). I think this is monomorphic VT due to scar tissue in the LV.

Best regards

Kjell Nikus, MD PhD, nickname "the English Lord" or "the Flying Finn"

Tampere, Finland

ECG1: sinus rhythm, anterolateral MI age indeterminate exclude anterior aneurysm.

ECG2: VT from anterior apical lateral focus, there is north west axis, and AV dissociation in V3.

ECG3: similar to ECG1.

Approach: With poor Ejection fraction is candidate for ICD, can assess response of VR to Amio and if VT recurs consider VT ablation. Also consider Mitral clip procedure for mitral regurgitation.

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Dear Luciano and Andrés,

This is a ventricular tachycardia. The site of origin (or exit site if reentrant VT) is located in the inferolateral portion of the LV not too far from the apex.

The QRS during sinus rhythm shows an old anteroseptal MI and a left anterior fascicular block with right bundle branch block. The QRS during VT is completely different that the one recorded during sinus rhythm with all negative QRS in aVL and right superior axis.

Although he had a previous MI with depressed LV function and now presents with hemodynamically tolerated VT, he may do as well with antiarrhythmic therapy as with an ICD. The benefits and risks of each therapy need to be discussed with the patient, so he can make an informed decision.

Mario **González MD PhD**
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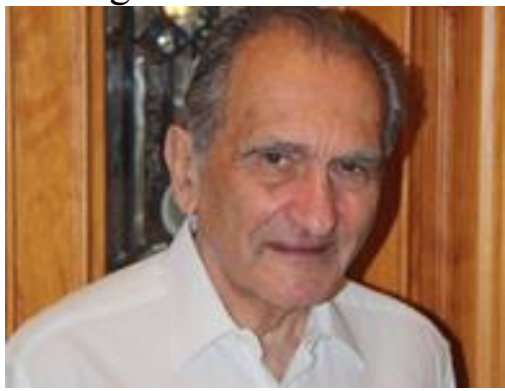
Dear friend, Prof. Edgardo,
I will try to analyze this intriguing and complex case by Dr. Luciano Pereira, and thank you for sending it for discussion.
Limb leads show deep (<25 mm) and wide S (>180 ms). This electronic phenomenon suggests hypertrophic and dilated remodeling of the inferior-posterior muscle. As a reaction to weakened hypertrophy of the cardiac base, this is expressed in a relative decrease of wide complexes and inverted Q and T waves in DI, aVL. Basal hypertrophies are accompanied in a 95% by left anterior hemiblock, while lateral hypertrophies have 1% of anteroseptal block.
Precordial leads: V1 shows Q/R wave with no S waves in DI, aVL, V5, V6; suggesting vertical depolarization of the right septum. From where did we extrapolate this concept? From Brugada syndrome. This suggests a delay in vertical depolarization of the right septum, by a sodium channel mutation or viral infection or type B antiarrhythmic drugs. In this case a delay in vertical depolarization is suggested, by a decrease (downregulation) of connexin 43 in the hypertrophic myocardium. Further, the same pattern exists in V2 and V3. This combination of right basal hypertrophy with biseptal hypertrophy is very frequently seen in clinical practice. From where did I learn this? When I studied and published the electrocardiographic pattern of sudden obstruction in the first diagonal artery, which involved ST elevation with positive T waves in aVL, V1 and V2, and depressed ST-T in DII, DIII, aVF. This also applies to hypertrophies, i.e. increase in aVL, V1 and V2 and reciprocity in DII, DIII, aVF.
Why do biseptal hypertrophies or septal V2 go together with basal hypertrophy in the right segment? Because embryologically, they have the same origin at 18 or 20 days of mammal embryos, when the bulbus cordis is formed, which will originate the future left and right ventricle outflow tract. These areas receive circulation by the first diagonal artery, RCA branch. The elevated ST segment with positive T waves in V1 through V3 is a reciprocal remodeling of inverted T waves in V4, V5, V6, which are not ischemic but hypertrophic. My position is that this ECG indicates an infiltrative cardiac pathology, such as Chagas disease, sarcoidosis, amyloidosis, Fabre-Anderson disease, or eosinophilic myocarditis.
In regard to ventricular tachycardia, they originate in the apex of the posterior wall. In infarctions, according to my investigations, they always appear with frontal axis at the right, and not the left, which appear in posterior infarction or non-ischemic myocardial pathologies.



Warm regards, and the discussion is open,
Samuel **Sclarovsky** MD Israel.

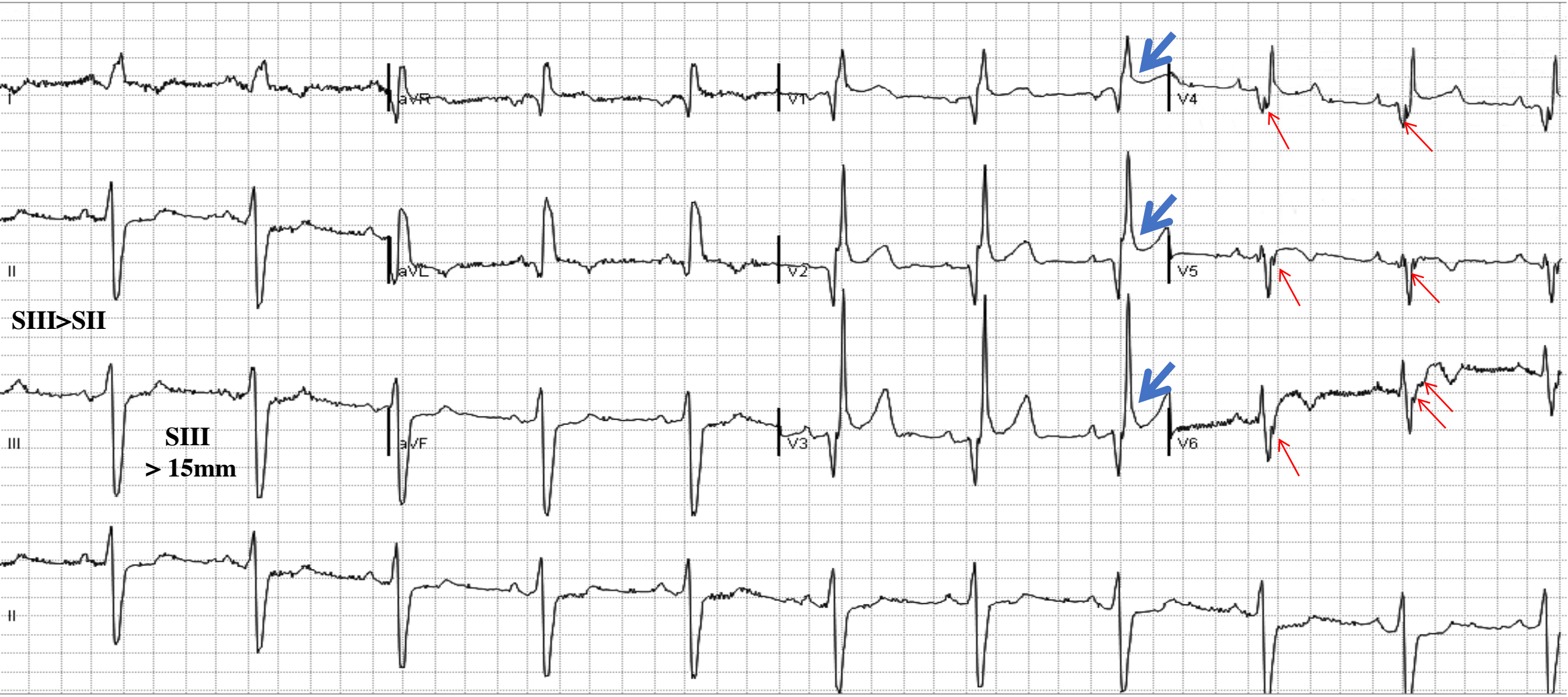
Final Conclusions

Andrés Ricardo **Pérez-Riera M.D. Ph.D.**

ECG / VCG Pérez-Riera | my cardiology site of scientific interests:

https://ekgvcg.wordpress.com

ECG-1



ECG diagnosis: SR, extreme left axis deviation (-75°), rS pattern in inferior leads with SIII>SII and qR in aVL and I: LAFB Rosenbaum´s type IV (SIII ≥ 15mm). QR from V1 to V4, negative T wave in lateral leads, and fragmented QRS(f-QRS) (**red arrows**) and early repolarization with J-wave in V1-V3. **Conclusion:** LAFB Rosenbaum type IV(associated with LVH (**Kukla 2015**) + RBBB (bifascicular block) + anterior myocardial infarction + lateral ischemia+ f-QRS(this is an strong arhythmogenic marker) + early repolarization on right precordial leads (**blue arrow**).

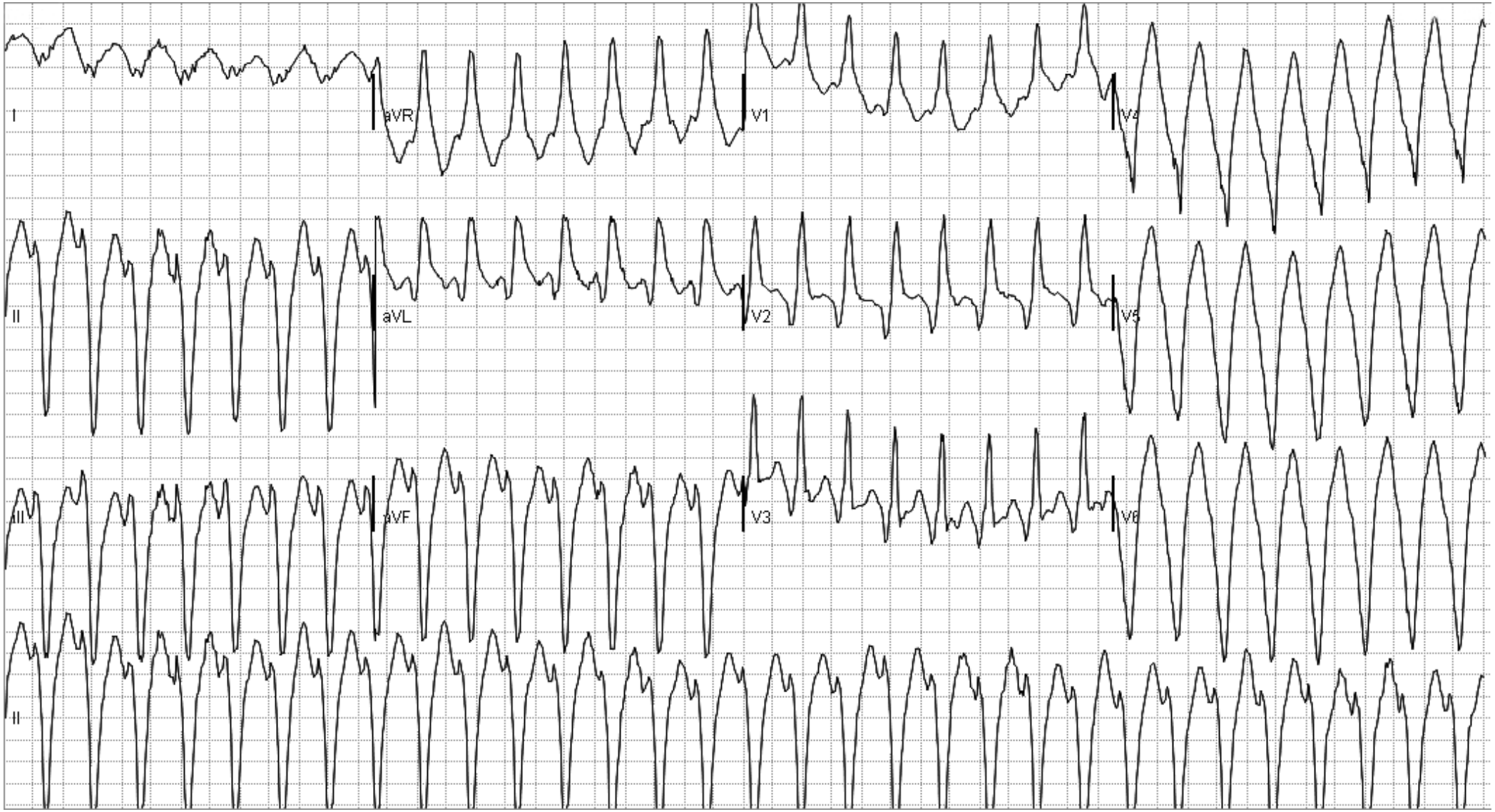
Observation and considerations related f-QRS and ERP

The presence of a fragmented QRS (f-QRS), which is an extra R wave (R′), notching of the single R wave, notching of the S wave **in at least two contiguous leads** on the 12-lead ECG, is associated with a myocardial scar from previous MI. Furthermore, the presence of f-QRS has been shown to be associated with adverse outcomes in CAD and non-CAD patients.

f-QRS in predicting ventricular tachyarrhythmia in many heart diseases, that is, CAD, non-ischemic cardiomyopathy, HCM, BrS (**Kataoka 2016**), ARVC/D, noncompaction cardiomyopathy (**Cetin 2016**), acute pulmonary embolism (**Cetim 2015**), aortic stenosis with hypertrophy and fibrosis (**Açıkgöz 2015**), thalassemia major with iron overload (**Karakulak 2015**), and radiotherapy for breast cancer (**Adar 2005**). In the majority of such cases, VT results in SCD. Diagnosing them beforehand can lead to prevention and/or early treatment of these arrhythmias to prevent potential morbidity and mortality. CAD is the most common cause of HF in developed countries. The extent of CAD by angiography is prognostic ally more significant than sole clinical diagnosis of an ischemic cardiomyopathy. VTS, and SCDs are major sequelae of ischemic cardiomyopathy, the prevention of which reduces the morbidity and mortality. In these patients, the f-QRS complex has been associated with regional myocardial damage, increased cardiac adverse events, and decreased event-free survival. f-QRS has been shown to be associated with significantly greater perfusion and function abnormalities than the Q wave, and, in fact, the f-QRS may be the only evidence of a prior silent MI. In the present era, when aggressive risk factor modification is the center of focus, the sensitivity of the Q wave was shown to be less than half when compared with the sensitivity of the f-QRS to predict a remote MI . Similarly, considering it as a predictive marker of future cardiac arrhythmias, it was also found that the incidence of an arrhythmic event was significantly higher in the patients with f-QRS than those without f-QRS on a 12-lead ECG during mean follow-up of 17 months, and the patients without f-QRS have a higher survival probability after an arrhythmic event. The f-QRS represents delayed activation in a larger ventricular mass (depolarization phenomena) that can cause multiple spikes within the QRS complex; so delayed activation causes delayed conduction (affectation of Vmx of phase 0 in fast monophasic action potentials), then arrhythmia.

Recently, Conte et al (**Conte 2016**) demonstrated that f-QRS and early repolarization pattern are common ECG findings in high-risk BrS patients, occurring in up to 27% of cases. When combined, f-QRS and ERP confer a higher risk of appropriate ICD interventions during a very long-term follow-up. We don’t know if this occurs in the sitting of CAD.

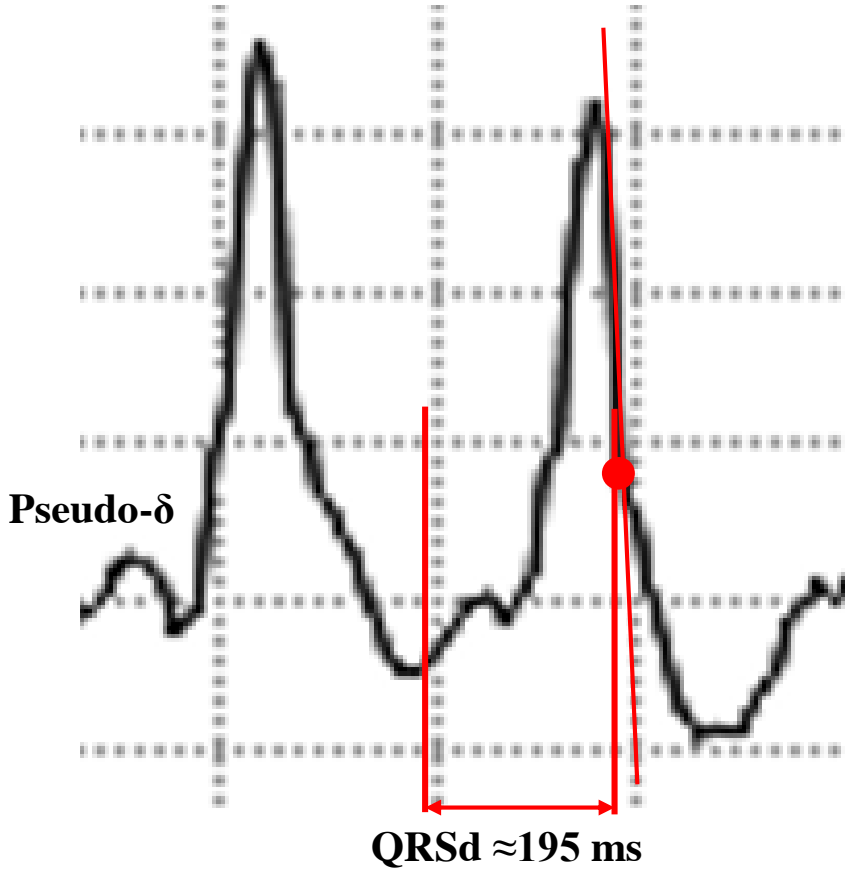
ECG -2 at ED admission



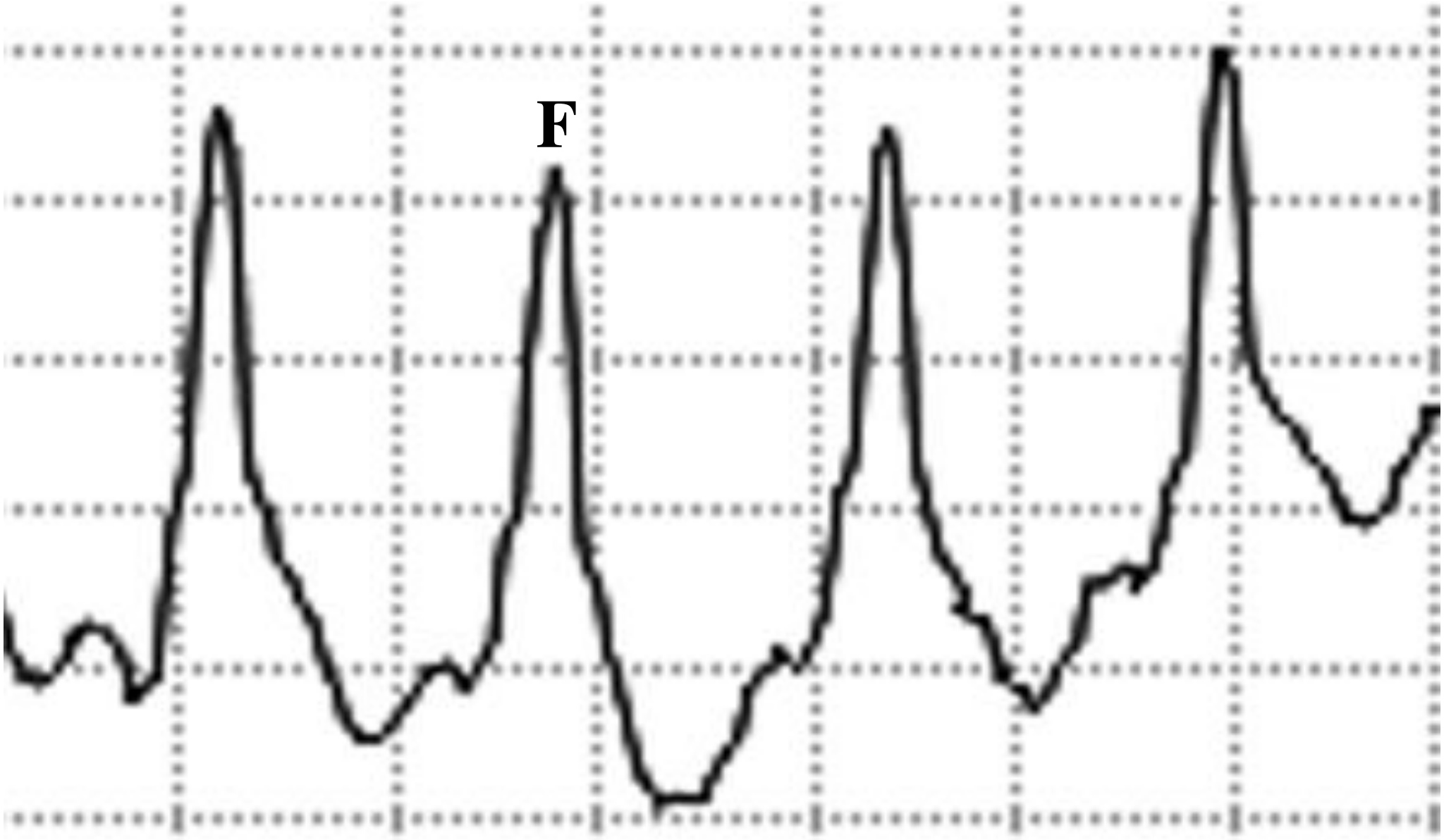
Sustained wide QRS tachycardia; heart rate of 188 bpm, very broad QRS duration ≈ 195 ms, QRS axis on top right quadrant (QRS axis -140°), RBBB-like pattern with monophasic R in V1, and QR in V2-V3; predominantly negative QRS complexes in the inferior and lateral precordial leads (V4-V6), and pure R in aVR and rsR' in aVL. This QRS morphology suggests ventricular tachycardia (VT) with a probable focus in the apical region of the left ventricle. See the sequence in the next slides……

Why VT?

1. Previous history of MI strong for VT
2. Advanced age suggests VT
3. Severe LV dysfunction (LVEF 36%)
4. Very broad QRS duration (≈195 ms) (>140 ms if RBBB pattern; >160 ms of LBBB pattern = VT form epicardial layer): No ECG feature consistently predicted an epicardial LV-VT origin in infarct-related tachycardias (**Berruezo 2004**). The epicardial VTs had longer QRS durations (189±32 ms in epicardial vs 179±37 ms in endocardial) but similar pseudo-δ durations was 38±27 vs 47±27 ms (p=0.2), similar R-peak times 93±35 vs 86±32 ms (p=0.4) and the shortest RS times were not different in epicardial versus endocardial VTs in post MI patients and median deflection index 0.82±0.25 versus 0.87±0.22 . So in post MI VTs these criteria may not help (**Gupta 2013**).

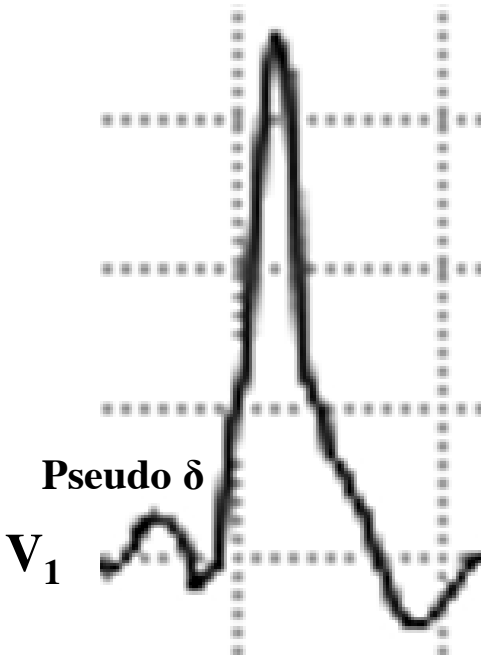


5. Presence of hybrid fusion beat (F). This feature is characteristic of VT.



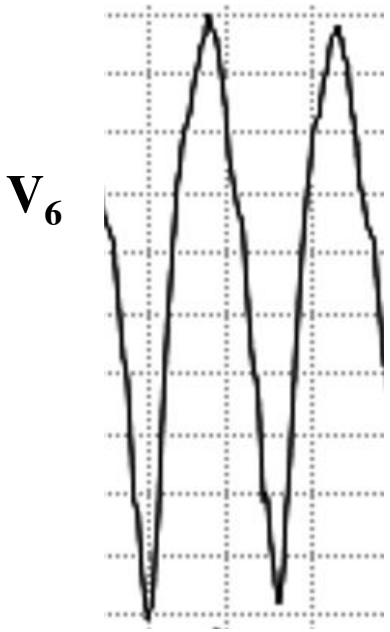
Fusion beats — occur when a sinus and ventricular beat coincides to produce a hybrid complex. An intermediate between the pure sinus beat and the pure ectopic beat. Fusion beat is narrower related to ventricular pure beat.

6. RBBB-like pattern with monophasic positive R-wave QRS pattern in V1= VT

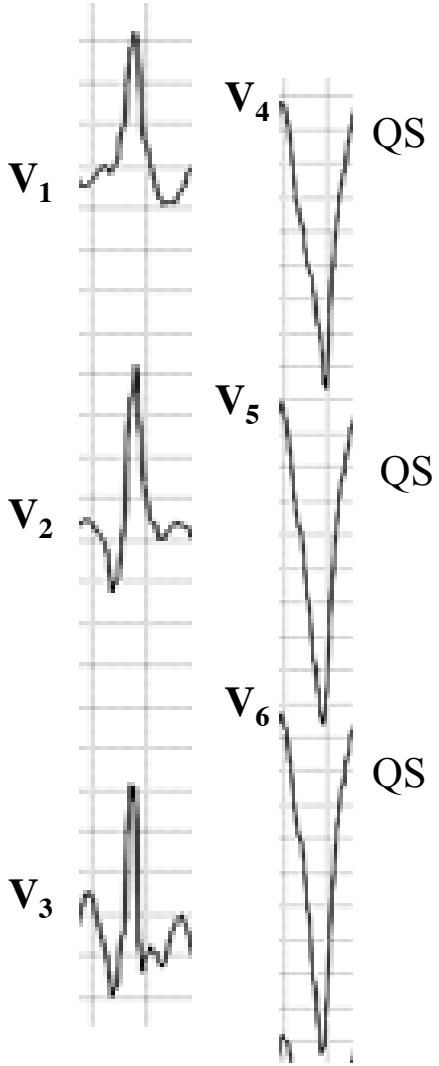


Additionally, pseudo δ-wave at the beginning of R-wave, indicative of VT with focus in epicardial layers.

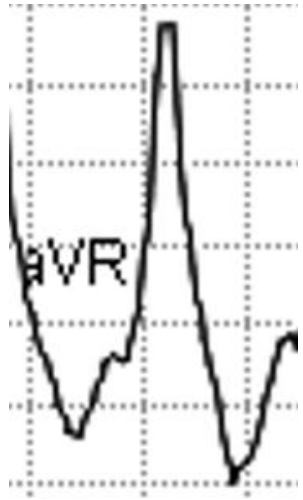
7. QS pattern in V6 = VT



8. Absence of RS pattern in any precordial leads indicates VT

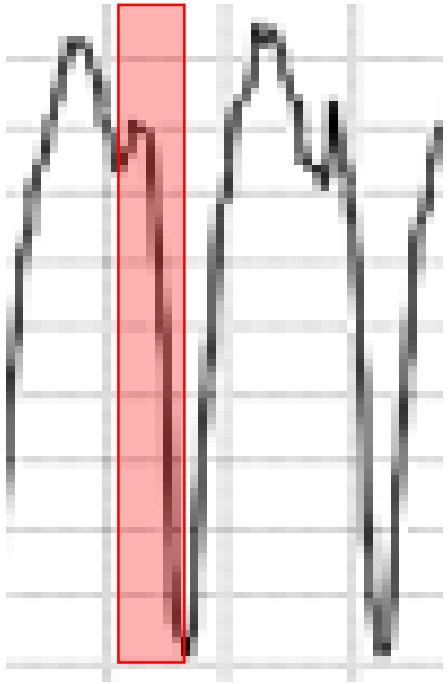


9. Initial R wave in aVR or first step "Vereckei algorithm": Yes = VT (**Vereckei 2008**)

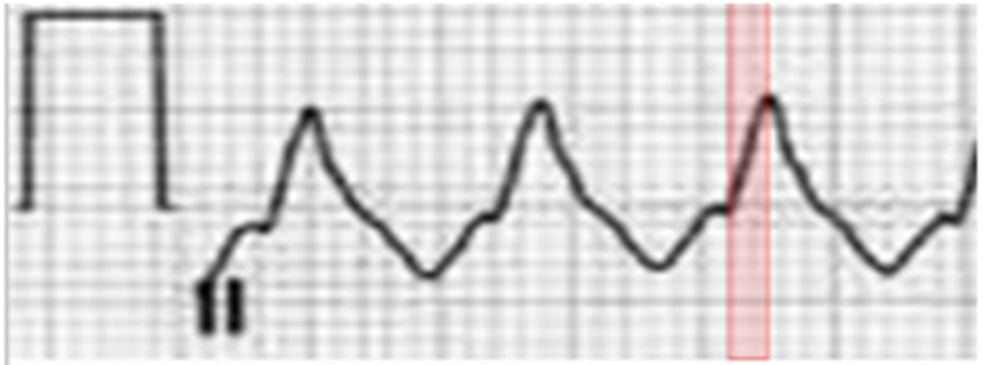


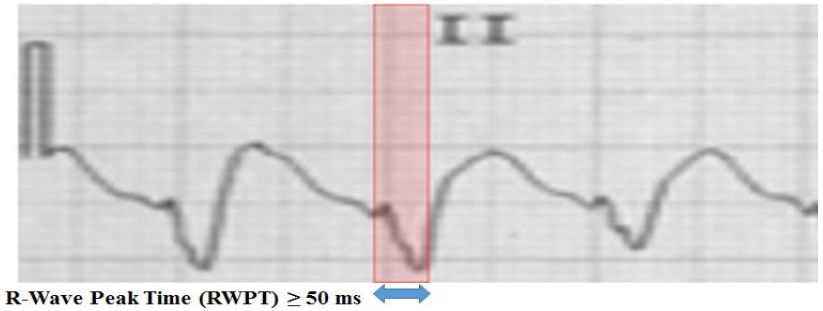
VT with origin in the inferior and apical regions of the ventricle having an initial R wave in aVR. The first step of Vereckei (initial R in aVR) is a simple, reproducible, accurate, and fast tool to use (**Kaiser 2015**).

10. The lead II R-wave peak time (RWPT) criterion, ultra-simple Brugada criterion i.e. the existing duration from QRS onset (R Wave) to the peak of R or S nadir in lead II (**Pava 2010**) = VT



S peak time ≥ 50 ms

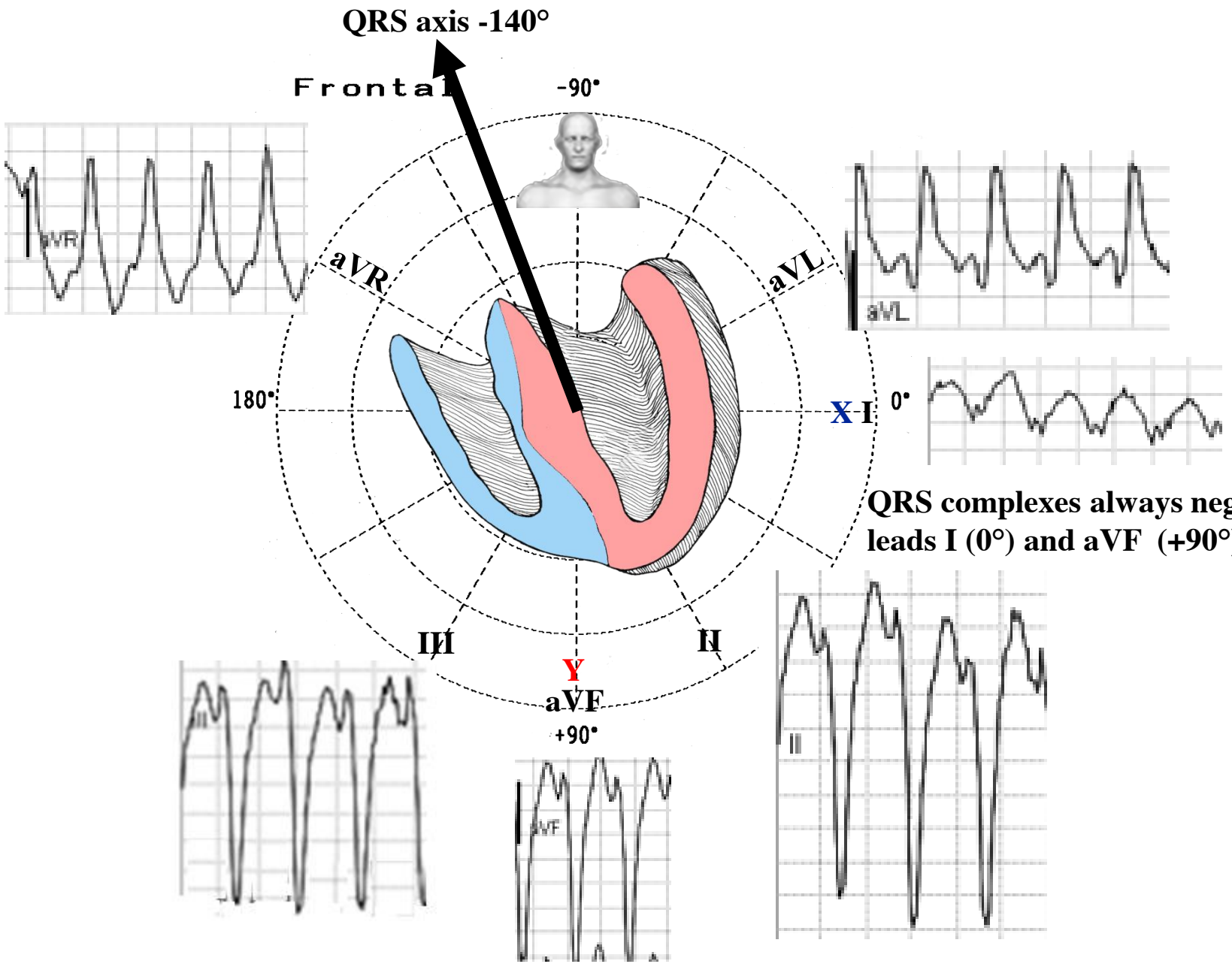




	Vereckei algorithm	RWPT criterion
Accuracy	Superior	Inferior
Sensitivity	Superior	Inferior
Specificity	Inferior	Superior
Negative predictive value (NPV)	Superior	Inferior
Positive predictive value (PPV)	Inferior	Superior

All of these parameters were lower in "real life" than those reported by the original authors for each of the particular electrocardiographic methods (**Szelényi 2013**).

11. QRS axis on upper right quadrant (QRS axis between -90 and 180°) AKA “Northwest axis” or no man's land or extreme right axis deviation = VT



Causes of extreme right axis deviation

1. Emphysema
2. Hyperkaliemia
3. Lead transposition: misplaced limb leads
4. Anatomical malposition
5. Artificial cardiac pacing
6. Divisional block of right bundle branch.
7. Ventricular tachycardia, Ventricular rhythms – e.g. VT, AIVR, ventricular ectopy

QRS complexes always negative in leads I (0°) and aVF (+90°) !!!!

Summary of the main differences between VT and SVT-A

	VT	SVT-A
Focus and etiologies	Bundle branches, Purkinje or ventricular muscle. The causes of VT may be with or without structural heart disease	Atria and/or AV junction
Presence of cannon A waves in the jugular veinous pulse	When present, it is diagnostic	No
Beat by beat variations in the intensity of the first heart sound,	Characteristic	No
Beat by beat variations of systolic blood pressure,	Characteristic	No
History of infarction, angina, CHF, cardiomyopathy, history of correction of congenital heart disease, family history of SCD: suggestive of HCM, ARVD/C, long QT syndrome and Brugada syndrome	Strongly suggestive	No
History of paroxysmal tachycardias responsive to vagal maneuvers or adenosine.	No	Characteristic
Previous ECGs with short PR (<120 ms), wide QRS and delta wave.	No	It indicates pre-excitation as cause.
Previous ECG with bundle branch block pattern identical to the pattern of the event	No	Characteristic
End of event with vagal maneuvers or adenosine	Rare	Yes.
QRS duration	>140 ms if RBBB pattern; >160 ms of LBBB pattern	< 140 or < 160 ms
SÂQRS in the FP	Suggestive when SÂQRS is in the northwest quadrant between –90º and ± 180º	No

	VT	SVT-A
QRS Pattern in V1	In the presence of LBBB pattern, initial r >40 ms and rS interval greater than 70 ms is suggestive. Biphasic or monophasic pattern if RBBB. When biphasic in V1 R' > R (rabbit ear sign) (Figure 29) (**Gozensky 1974**)	Initial narrow r, and clean s, with no notches if LBBB and triphasic pattern if RBBB
QRS Pattern in V6	rS, Qrs, QS, QR or monophasic R. If the pattern was RS R<S.	qRs, Rs or RS with R>S
The distance from the onset of QRS up to the nadir of S >100 ms (Brugada sign) (Fig. 30)	If present, it is diagnostic.	Lower
Notch near the nadir of the S wave (sign of Josephson) (Figure 31)	Characteristic	Absent
QRS complexes of the R or Rs type	Diagnostic	No
Initial q or r wave with duration >40 ms in aVR (qR or rS)	Diagnostic	No
Pattern matching in precordial leads	Strongly suggestive.	No
Presence of fusion beats	Strongly suggestive.	No
Presence of capture beats	Strongly suggestive.	No
Second-degree ventricular-atrial block	Characteristic when present: QRS/P ratio; however, with a greater number of QRS than P.	No
Pattern of LBBB with axis in the right upper quadrant	Nearly always VT.	No
Ratio of duration between the initial and final part of QRS ≤ 1 (Oreto 2009)	Suggestive.	>1

Approach

In this particular case it is mandatory to preform an Electrophysiological Study (EPS). If inducible he will has a greater benefit from prophylactic ICD implantation due to their higher risk of death or arrhythmia (**Zaman 2014**). The most convincing approach seems to be the one combining both noninvasive risk stratification parameters (e.g., PVCs> 10/h or reduced heart rate variability < 70 ms or a positive signal-averaged ECG) followed by a further arrhythmic risk stratification, obtained through EPS. A negative EPS may delineate a subgroup of patients with severely impaired LVEF whose care can be safely managed long-term without an ICD. Severely impaired LV function but no inducible VT have a favorable long-term prognosis without the protection of an ICD (**Zaman S2014**).

References

1. Açıkgöz E, Yaman B, Açıkgöz SK, et al.Myocardial Fibrosis Is the Key Component of Hypertrophied Myocardium That Cause Fragmented QRS in Aortic Stenosis. Ann Noninvasive Electrocardiol. 2015;20(5):513.
2. Adar A, Canyılmaz E, Kiris A, et al.Radiotherapy Induces Development of Fragmented QRS in Patients with Breast Cancer. Breast Care (Basel). 2015;10(4):277-80.
3. Cetin MS, Ozcan Cetin EH, Arisoy F, et al. Fragmented QRS Complex Predicts In-Hospital Adverse Events and Long-Term Mortality in Patients with Acute Pulmonary Embolism. Ann Noninvasive Electrocardiol. 2015 Dec 24. doi: 10.1111/anec.12332. [Epub ahead of print]
4. Cetin MS, Ozcan Cetin EH, Canpolat U, et al. Usefulness of Fragmented QRS Complex to Predict Arrhythmic Events and Cardiovascular Mortality in Patients With Noncompaction Cardiomyopathy. Am J Cardiol. 2016;117(9):1516-23.
5. Conte G, de Asmundis C, Sieira J, et al. Prevalence and Clinical Impact of Early Repolarization Pattern and QRS-Fragmentation in High-Risk Patients With Brugada Syndrome. Circ J. 2016 Aug 25. [Epub ahead of print]
6. Gozensky C, Thorne D. Rabbit ears: an aid in distinguishing ventricular ectopy from aberration. Heart Lung. 1974;3(4):634-6.
7. Gupta PN, Kumar A, Namboodiri N, Balachandran A.What is this? VT versus SVT. BMJ Case Rep. 2013 Sep 23;2013. pii: bcr2013200806. doi: 10.1136/bcr-2013-200806.
8. Kaiser E, Darrieux FC, Barbosa SA, et al. Differential diagnosis of wide QRS tachycardias: comparison of two electrocardiographic algorithms.Europace. 2015;17(9):1422-7.
9. Karakulak UN, Tutkun E, Yılmaz ÖH.Iron overload and fragmented QRS in patients with Thalassemia major: Mechanisms, therapies, and new horizons.Anatol J Cardiol. 2015;15(7):592.
10. Kataoka N, Mizumaki K, Nakatani Y, et al. Paced QRS fragmentation is associated with spontaneous ventricular fibrillation in patients with Brugada syndrome. Heart Rhythm. 2016;13(7):1497-503.
11. Kukla P, Jastrzębski M, Bryniarski L.Total Masquerading Bundle Branch Block.Ann Noninvasive Electrocardiol. 2015;20(6):601-3. Oreto G, Luzza F, Satullo G, et al. Wide QRS complex tachycardia: an old and new problem. G Ital Cardiol (Rome). 2009;10(9):580-95.
12. Pava LF, Perafán P, Badiel M, et al. R-wave peak time at DII: a new criterion for differentiating between wide complex QRS tachycardias. Heart Rhythm. 2010;7(7):922-6.
13. Szelényi Z, Duray G, Katona G, et al.Comparison of the "real-life" diagnostic value of two recently published electrocardiogram methods for the differential diagnosis of wide QRS complex tachycardias. Acad Emerg Med. 2013;20(11):1121-30.

14. Vereckei A, Duray G, Szénási G, et al. New algorithm using only lead aVR for differential diagnosis of wide QRS complex tachycardia. Heart Rhythm. 2008;5(1):89-98.
15. Zaman S, Narayan A, Thiagalingam A, et al. What is the optimal left ventricular ejection fraction cut-off for risk stratification for primary prevention of sudden cardiac death early after myocardial infarction? Europace. 2014;16(9):1315-21.