Caucasian man with acute coronary syndrome and peculiar ECG

Homem branco com síndrome coronariano agudo e ECG peculiar

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Case report

A 61-year-old Caucasian male presented to the emergency room with severe crushing chest pain of 3-hour duration. The pain was retrosternal, was crushing, and was associated with profuse cold sweating and vomiting. There was no radiation of pain, breathlessness, or nausea. He is a known hypertensive on olmesartan 40mg + hidroclorotiazide 12,5mg + orlistat 120 mg 3x/day. He has centripetal severe obesity class II (BMI 38). Waist-to-hip ratio > 0.95. He did not have any other risk factors for coronary artery disease (CAD). He was not a smoker, and there was no family history of CAD.

The admission ECG in showed particular characteristics (ECG1). On physical examination, his blood pressure was 120/80 mm of Hg and heart rate was 52/minute, and there were no abnormal findings on physical examination.

Questions

1. Which is the electrocardiographic particular diagnosis?
2. Which is the culprit artery?
3. Which is the appropriate approach?

Portuguese: Homem branco de 61 anos se apresentou na sala de emergência com dor intensa retroesternal com caráter de esmagamento, sem irradiação iniciada havia 3 horas atrás. A dor estava associada com sudorese fria profusa e vômitos. Não estava associada a falta de ar ou náusea. Referia ser hipertenso em uso regular de olmesartan 40mg + hidroclorotiazida 12,5mg + orlistat 120 mg 3x/dia.

Obeso centrípeto grave grau II (IMC 38). Relação cintura/cadeira > 0,95. Não tem outros fatores de risco para doença arterial coronariana (DAC). Não é fumante, e não tem história familiar de DAC.

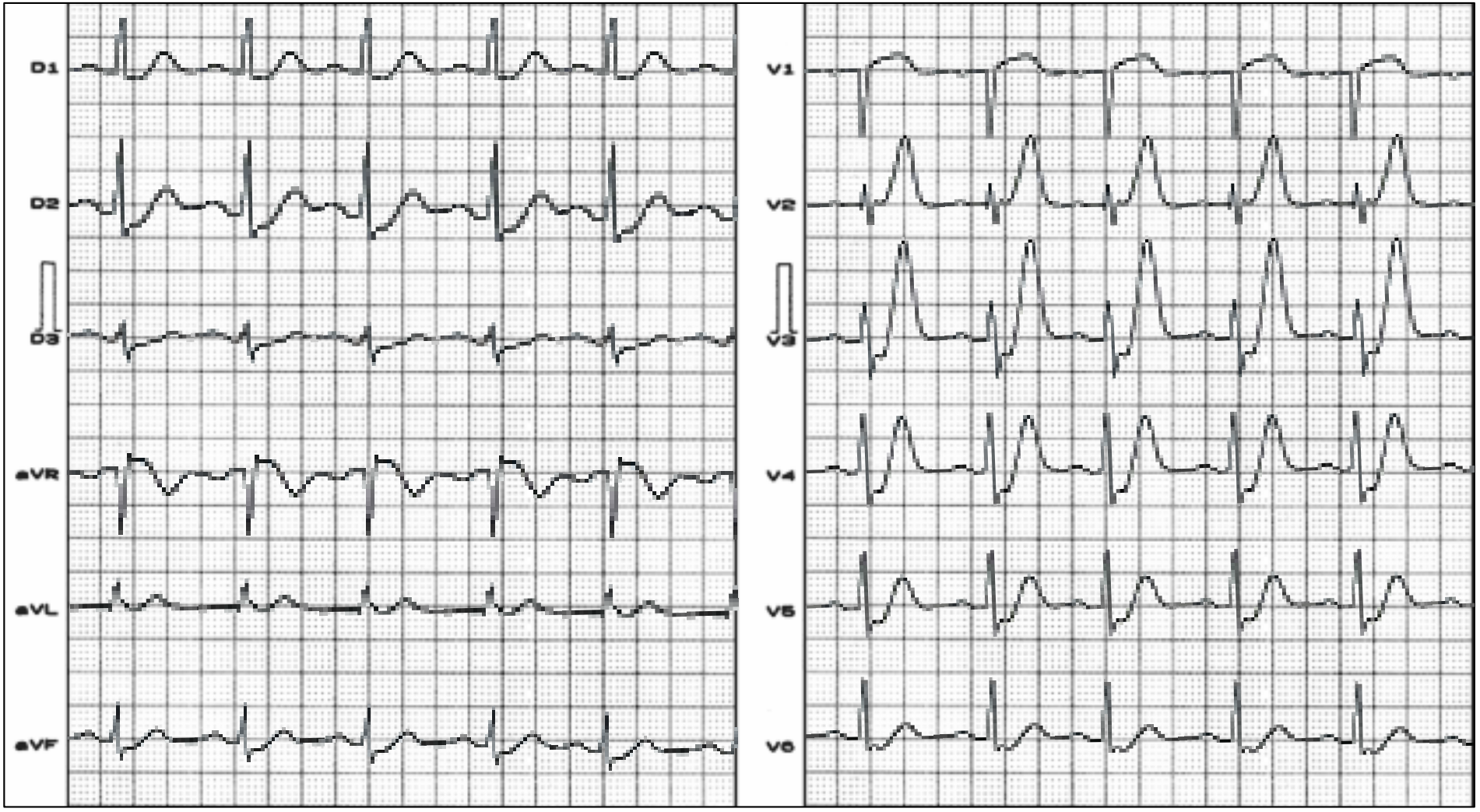
O ECG de admissão possui características particulares (ECG1).

Ao exame físico, pressão arterial de 120/80 mm Hg e freqüência cardíaca 52 bpm, e não foram achadas outras anormalidades.

Perguntas:

1. Qual é o diagnóstico eletrocardiográfico em particular?
2. Qual é a artéria culpada?
3. Qual é a abordagem adequada?

ECG1 - Name: STA; **Age**: 61 y/o; **Sex**: M; **Ethnic group**: Caucasian; **Weight**: 111 kg; **Height**: 1.69 m; **Date**: Jul 07, 2015; **Medication in use**: Olmesartan medoxomil 40 mg + hidroclorotiazide 12.5 mg + orlistat 120 mg 3x/day.



Colleagues opinions

Spanish

Maestro Andrés
Depresiones del ST con altas ondas T positivas es una presentación inusual del SCA (sólo el 2% según la publicación de Winter et col). Es un posible signo de isquemia subendocárdica. Esto patrón puede evolucionar rápidamente a STEMI debido oclusión proximal de la DA(elevación del segmento ST en aVR asociado). Angiocoronariografia urgente está indicada.

Raimundo Barbosa-Barros Fortaleza Ceará Brasil

English

Master Andrés: ST depressions with high positive wave T is an unusual presentation of the ACS (only 2% as published by Winter et al). It is a possible sign of sub endocardial ischemia. This pattern can quickly evolve to STEMI due to proximal occlusion of the LAD (ST-segment elevation in aVR associated). Angiocoronariography is urgently indicated.

Raimundo Barbosa-Barros Fortaleza Ceará Brazil

Caro Maestro El Potro O amigo Raimundo tiene razón en sus colocaciones, (exceto que es "downsloping" of ST), e envio las respuestas(1) así:

1, **Diagnóstico eletrocardiográfico:** Isquemia aguda regional subendocárdica

2, **Artéria culpada:** Possivelmente DA com obstrução sub total ou obstrução total do 1º Diagonal

3. Abordagen: Cateterismo coronário.

1. Electrocardiography of Acute Myocardial Ischaemic Syndromes - Sclarovsky,S. 1999 pg.13,17.

Adail Paixão Almeida M.D. Vitória da Conquista Bahia Brasil

English

Dear Master Pony: My friend Raimundo is correct in their placements, (except in the ST segment shape: "downsloping"), and I am sending the responses (1) as follows:

1 Electrocardiographic Diagnosis: acute regional subendocardial ischemia

2 **Culprit artery:** subtotal or total obstruction of LAD or obstruction of first diagonal

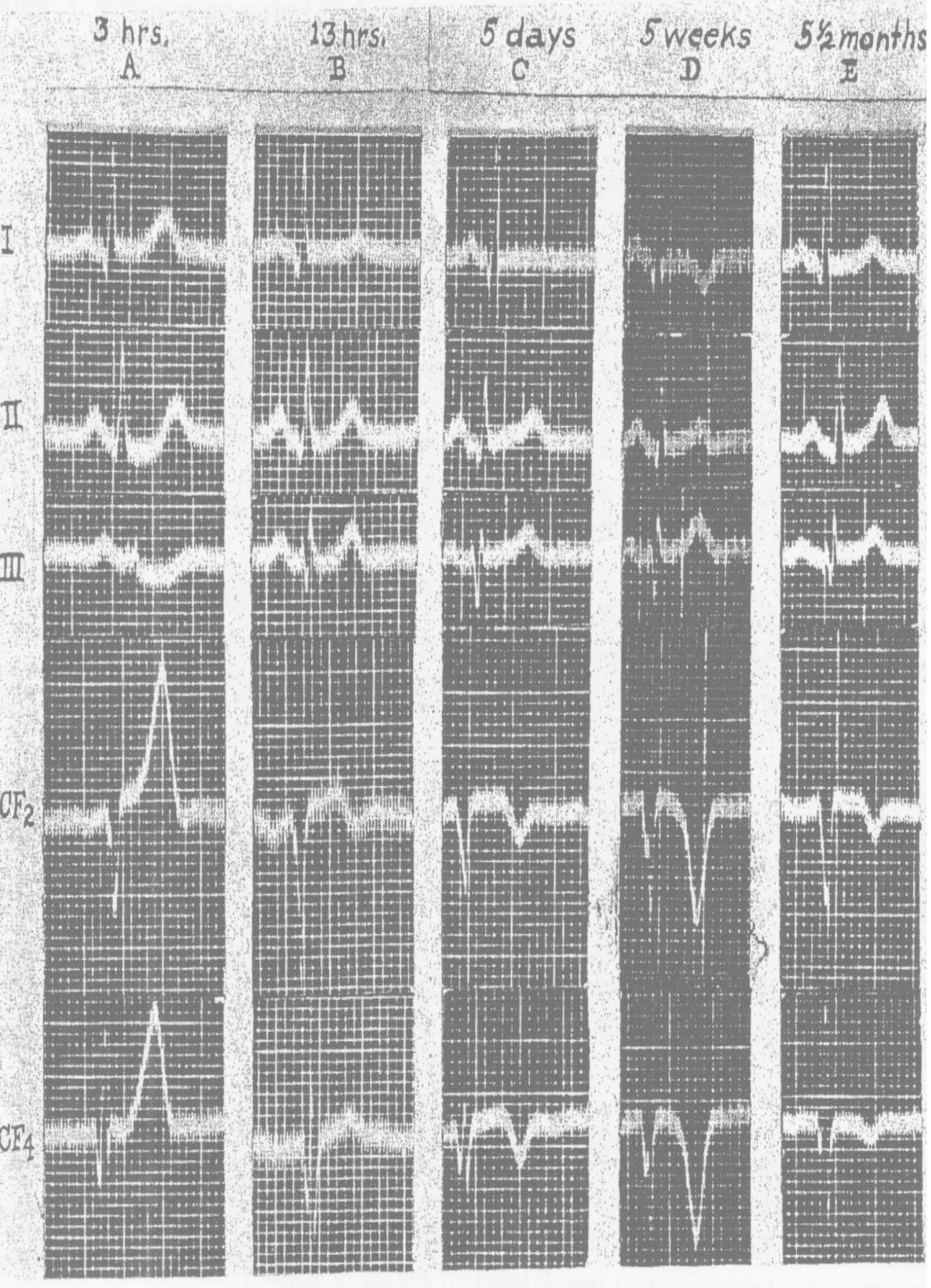
3. Approach: coronary catheterization.

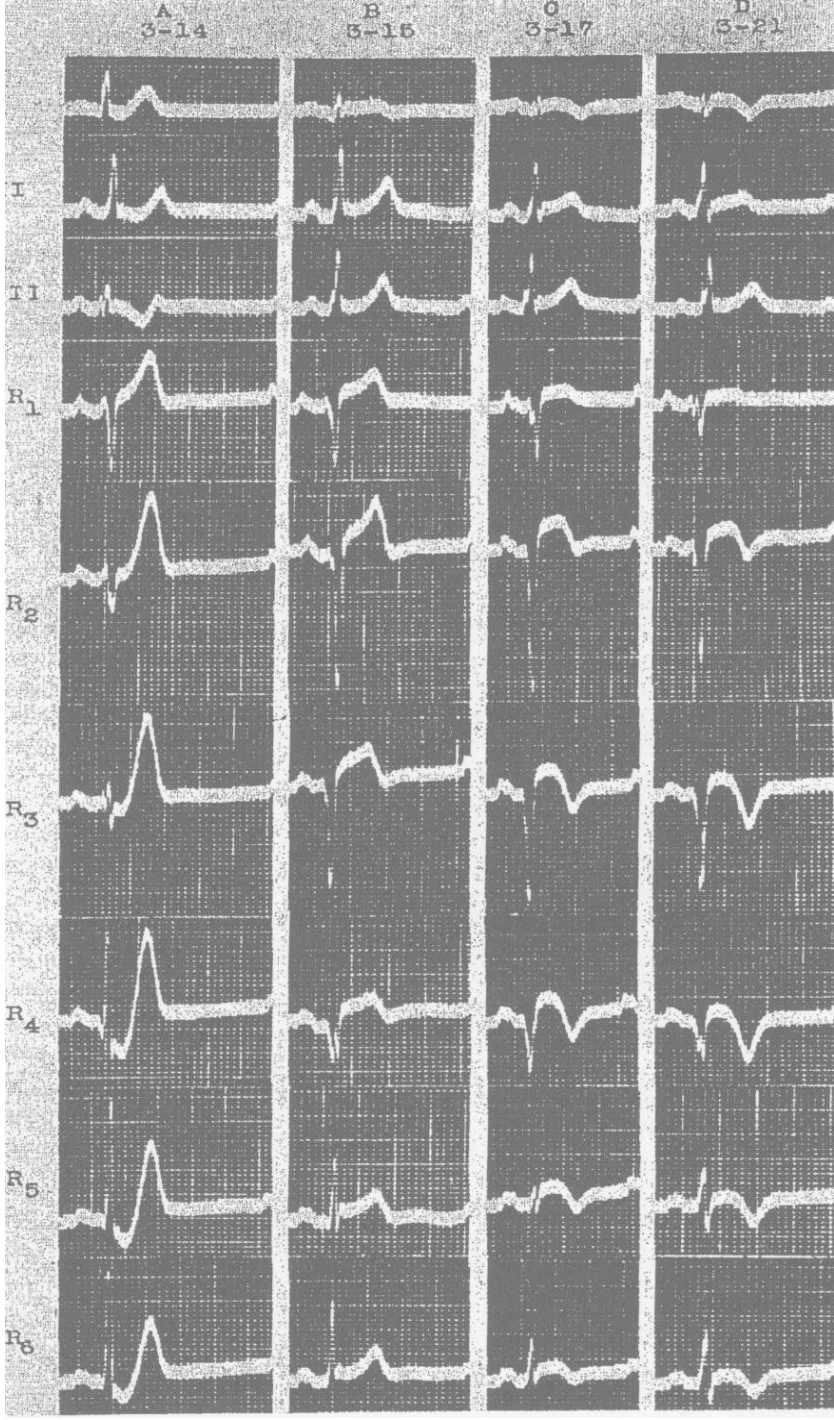
Adail Paixão Almeida M.D. Vitória da Conquista Bahia Brazil

Dear Maestro Andrés: I agree with Raimundo and Adail. ST depression with prominent positive T waves in the precordial leads has been described as regional subendocardial ischemia by Master Samuel Sclarovsky. He noted that the patients with this ECG pattern have subtotal LAD occlusion or total occlusion of a side branch, typically the diagonal branch. In the publications by de Winter (NEJM 2008) and Verouden (Heart 2009), upsloping ST depression with prominent positive T waves were associated with LAD occlusion. In our primary PCI network we treat these patients as STEMI and take them to emergent PCI. Interestingly, Dressler and Roesler already in 1947 were discussing the issue of high T waves (Am Heart J 1947). They presented one case (attached LAD1) with (downsloping) ST depression with prominent T waves but without QRS changes. Later on, Q waves and inverted T waves developed and the authors diagnosed anteroseptal infarction. In another case (LAD2) they presented a patient with prominent precordial T waves without significant ST or QRS changes. Later on inverted T waves developed – also this case was classified as anteroseptal infarction. According to Master Samuel, prominent precordial T waves without significant ST elevation represents **Grade 1 of ischemia**, indicating well protected myocardium with slow development of Q waves in LAD occlusion. See next slide the ancient ECGs

Best regards

Kjell Nikus M.D.Ph.D.
Tampere
Tampere University Hospital, Tampere Finland
Finland


3 hrs.
A
13 hrs.
B
5 days
C
5 weeks
D
5½ months
E
I
II
III
CF2
CF4


A
3-14
B
3-15
C
3-17
D
3-21
I
II
R1
R2
R3
R4
R5
R6

I believe This patient has a proximal LAD occlusion. The ECG shows upsloping ST depression in precordial leads with peaked T waves.
This pattern was described by de Winter and colleagues (de Winter T waves)
The patient should undergo angioplasty and stent placement.
Very nice case!
Thank you again for sharing such wonderful tracings.

Mario

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Estimados foristas, Caso del Dr. A Pérez Riera

1. Diagnóstico electrocardiográfico: La explicación más lógica para el patrón de onda T alta y picuda y moderada depresión del ST es que hay un retraso en la repolarización en el área subendocárdica con un cambio en la forma del potencial de acción transmembrana (ascenso lento y de larga duración). La suma de este pequeño cambio con el potencial de acción transmembrana del subepicárdico explica la depresión del punto J y la onda T alta del trazado electrocardiográfico. La presencia de ondas T altas y simétricas asociadas a depresión del punto J pueden persistir durante horas, a menudo debido a un tratamiento antiagregante/antitrombótico agresivo que impide la evolución a ST elevado. Es frecuente la presencia de circulación colateral o precondicionamiento isquémico

2. Arteria culpable: Suboclusión crítica de la DA

3. Abordaje: cateterismo emergente. Lo habitual es que la arteria finalmente se ocluye y acaba presentando un ST elevado que se detecta con la realización de muchos ECG seriados, porque la elevación de ST puede no aparecer hasta el último momento.

Miquel Fiol -Sala

Dear forum participants, About the Case of Dr. Andrés R. Pérez-Riera

1. **ECG diagnosis**: The most logical explanation for the high T wave pattern and pointed to moderate depression of ST is that there is a delay in repolarization in the subendocardial area with a change in the form of the action potential transmembrane (slow ascent and long duration). The sum of this small change in transmembrane potential subepicardial action explains the J point depression and high T wave electrocardiographic tracing. The presence of high and symmetrical T waves associated with J point depression can persist for hours, often due to antiplatelet / antithrombotic treatment that prevents aggressive evolution to high ST. Often the presence of collateral circulation or ischemic preconditioning.
2. **Culprit artery**: severe subocclusion of the LAD.
3. **Approach:** Emerging catheterization. Typically, the artery is occluded and just finally presenting a high ST is detected with the realization of many serial ECG because of ST elevation may not appear until the last moment. Miquel Fiol –Sala M.D.Ph.D.

Análisis del ECG del Profesor Andres Ricardo Perez Riera

Esta es una obstruccion subita de una arteria secundaria. Las obtrucciones subitas y unicas de las arterias secundarias tienen un patrón tipico: Una o 2 derivaciones consecutivas con ST-T elevado 2) la derivacion proxima al ST elevado , el ST es isoelectrico 3) las siguientes derivaciones precordiales tienen ST deprimido con T muy apiculada y alta sugeriendo que esta arteria irriga el subendocardio.

Porque el centro de la isquemia esta en V1(septo alto derecho) y no hay intervencion de las derivaciones de los miembros. Mi primer diagnóstico es obstrucción subita de la arteria primera marginal derecha la cual tiene que ser larga porque penetra profundamente en el septo izquierdo. En algunos casos muy raros, obedeceria a obstrucción de la arteria del cono derecho. Como diagnóstico diferencial podria pensarse en obstrucción de una diagonal primera, pero esta compromete la base cardiaca , ocasionando elevación del ST-T en aVL y depression en DII,DIII. Además, la elevación del ST siempre es mayor en V2 que en V1.

No veo otra posibilidad diagnostic. Un fraternal abrazo

Samuel sclarovsky

ECG analysis of Professor Andres Ricardo Perez Riera

This is a sudden blockage of a secondary artery. Sudden obstructions in secondary arteries have a typical pattern: One or two contiguous leads with ST-SE 2) the lead next to the obstruction has isoelectric ST. 3) the following precordial leads have ST depressed followed by very apiculate and high T waves suggesting that this artery supplies the subendocardium.

Because the center of ischemia was added to V1 (high right septum) and no intervention of the limb leads.

My first diagnosis is sudden obstruction of the right marginal artery which first have to be long because it penetrates deep into the left septum.

In some rare cases, obey artery blockage of the right cone.

As differential diagnosis could think of obstruction of a first diagonal, but this compromises the cardiac base, causing ST-T elevation in aVL and depression in DII, DIII. In addition, ST elevation in V2 is always greater than V1.

I see no other possibility diagnosed

A fraternal hug

The de Winter ECG pattern: a specific pattern equivalent of an anterior STEMI, but presents like an NSTEMI

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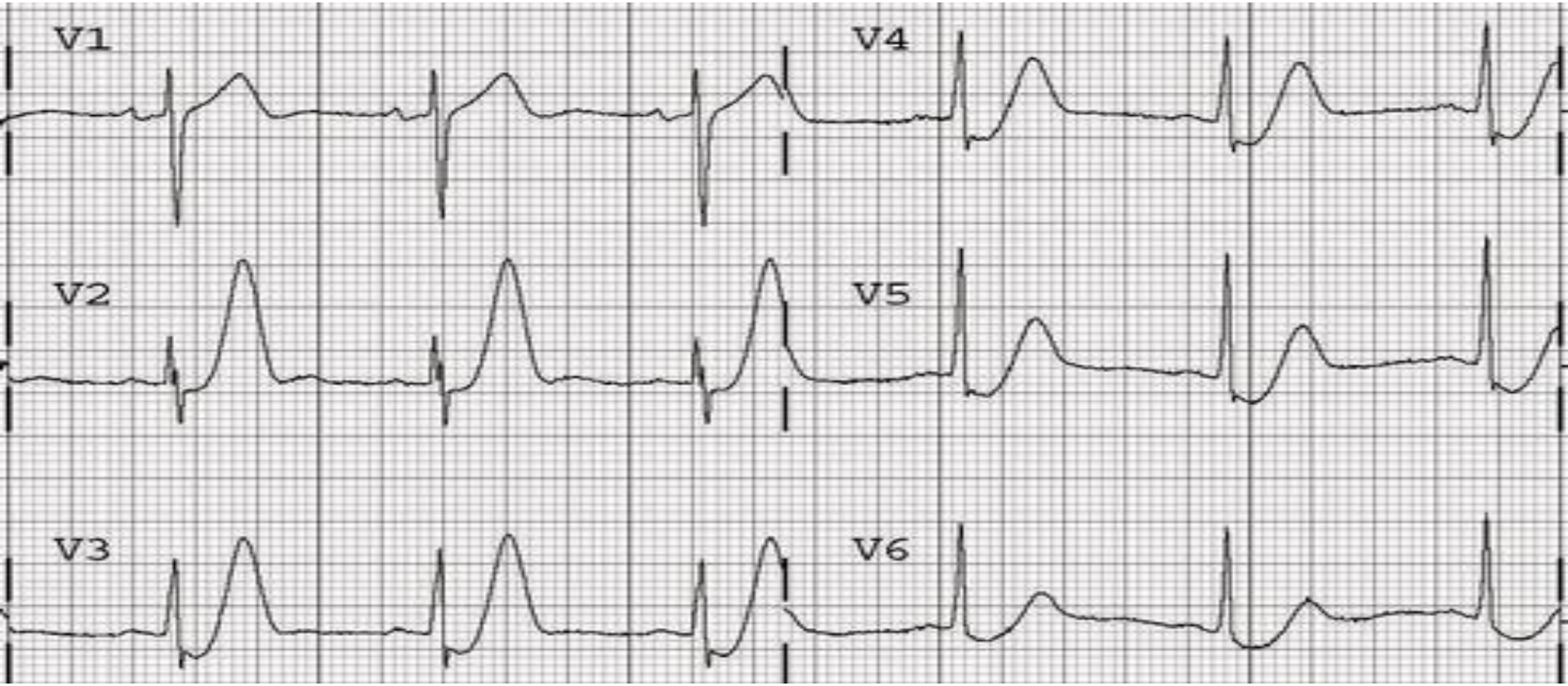
The de Winter ECG pattern ST/T wave complexes about 2% of acute LAD occlusions (contrast with subacute of Wellens syndrome)



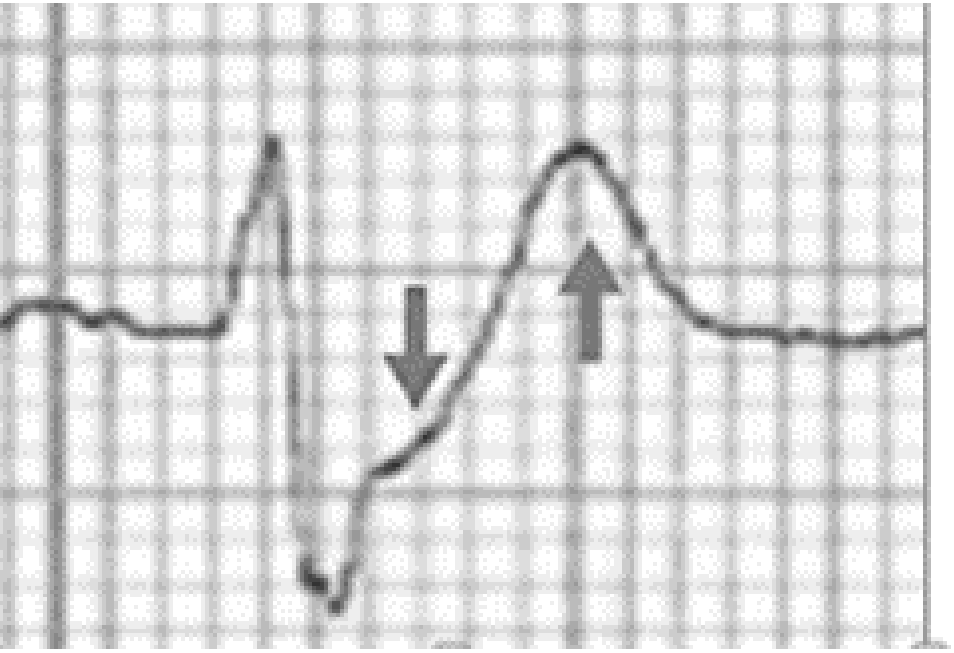
Upsloping 1-3 mm ST segment depression concave upwards at the J-point in the precordial leads followed by peaked prominent, symmetric anterior T(V2-V6) waves, (so-called hyperacute T waves), with the ascending limb of the T wave commencing below the isoelectric baseline. Additionally, sometimes sublte ST segment elevation (≥0.5mm-1mm) in aVR. Lead aVR shows slight ST-segment elevation in most cases = It is the so-called the De Winter ECG pattern. This is a sign of acute LAD occlusion and should be treated as a STEMI equivalent. The de Winter pattern is seen in ≈ 2% of acute LAD occlusions and is under-recognized by clinicians. There is also some high lateral involvement, with subtle ST elevation in aVL plus reciprocal change in III + aVF. This is consistent with LAD occlusion occurring proximal to the first diagonal.

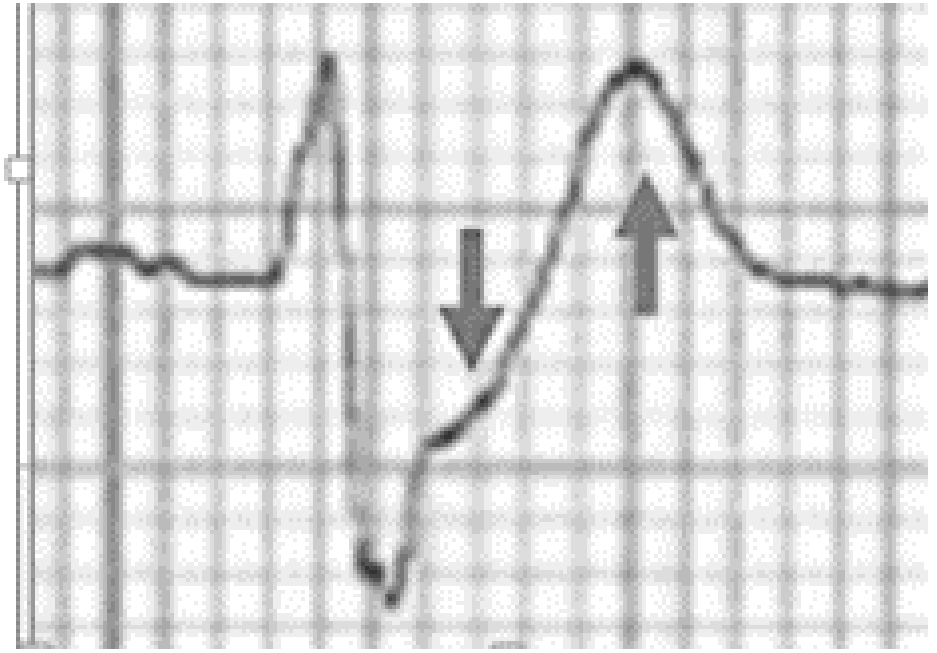
“Normal” STEMI morphology may precede or follow the de Winter pattern. Winter report suggested that the ECG did not change or evolve until the culprit artery had been opened. Since then, cases have been reported where the deWinter pattern evolved from, or evolved to a “classic” anterior STEMI. Normal” STEMI morphology may precede or follow the deWinter pattern. The de Winter ECG pattern was first reported in a 2008 case series by de Winter, Wilde and Welles, and, who observed this ECG pattern in 30 / 1532 patients with acute LAD occlusions (2% of cases).

Verounden et al. (**Verouden 2009**) found a de Winter ECG pattern in 35 / 1890 patients requiring PCI to the LAD (2% of cases). Patients with the de Winter ECG pattern were younger, more likely to be male and with a higher incidence of hypercholesterolaemia compared to patients with a classic STEMI pattern.

There is now growing evidence to suggest that the de Winter ECG pattern is highly predictive of acute LAD occlusion.

Some authors have proposed that the de Winter pattern should be considered a “STEMI equivalent”, and that patients with chest pain and this ECG pattern should receive emergent reperfusion therapy with PCI or thrombolysis.(**Yan 2007**).



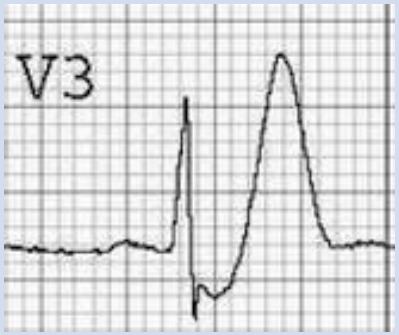
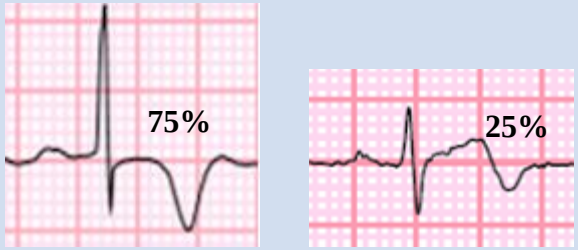


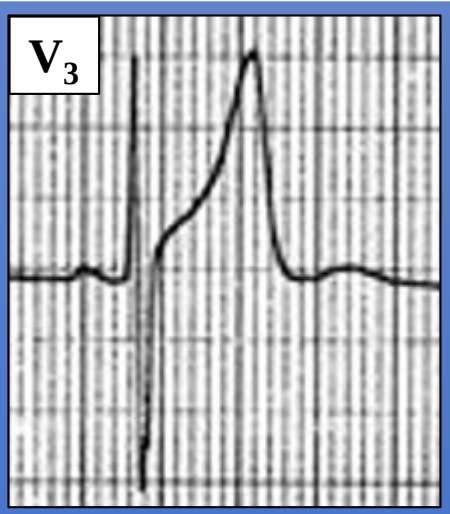
Anterior myocardial infarction (MI) from acute left anterior descending (LAD) coronary artery occlusion is usually characterized by ST-segment elevations (STE) indicating the need for emergent restoration of coronary flow. Rarely, acute anterior MI may present with ST-segment depressions (STD) in precordial leads, called deWinter T-wave ECG pattern. Recognition of this unique ECG pattern is important for appropriate triage.

deWinter's pattern differs from the deep T-wave inversion of Wellen's sign seen in critical LAD stenosis and impending infarction. Recognizing this, the patient was then taken for emergent coronary angiography. Acute LAD occlusion was confirmed and she underwent percutaneous coronary intervention with a drug eluting stent (see next slide).

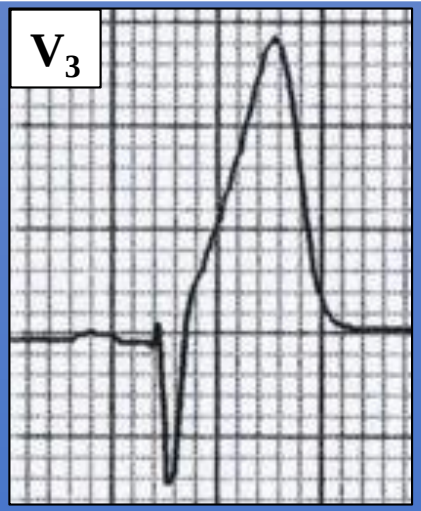
Unfamiliarity with this ECG pattern may lead to delayed coronary intervention causing morbidity and mortality as in our patient who suffered VF during delayed restoration of coronary flow. Awareness of the rare but highly characteristic deWinter ECG pattern, though not included in current guidelines for STEMI, is essential to avoid overlooking anterior MI patients who present with this ECG pattern rather than ECG patterns typically associated with acute LAD occlusion.

de Winter pattern versus Wellens syndrome

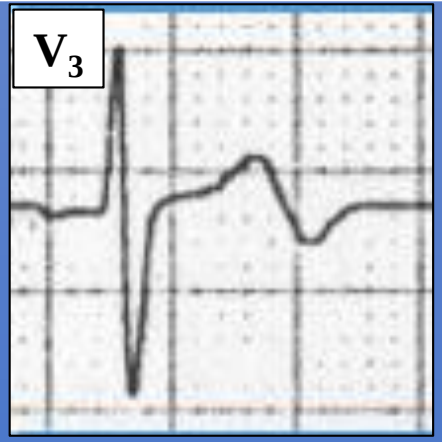
	de Winter pattern	**Wellens syndrome or** LAD coronary T-wave syndrome
ECG changes and pain relationship	The ECG changes occur concomitant with the pain (during episodes of pain) (Acute)	The ECG changes usually occur during a pain-free periods when other evidence of ischemic or unstable angina may be absent. (Subacute)
ST segment	Upsloping 1-3 mm ST segment depression concave upwards at the J-point in the precordial leads (V2-V6).	Little or no significant ST-segment elevation (≤1 mm) concave to the top or straight**.**
T wave shape/polarity	Peaked prominent, symmetric anterior T(V2-V6) waves, (so-called hyperacute T waves), with the ascending limb of the T wave commencing below the isoelectric baseline. 	Persistently symmetrical, deep negative and broad-based T-waves (Type A) or plus-minus T waves with inversion of the terminal portion (Type B). The anterior terminal Inversion of T waves is called Wellens' Warning. Type A Type B 
Precordial R-wave progression	Frequent loss on precordial leads	Normal or prominent (**Riera 2008**)



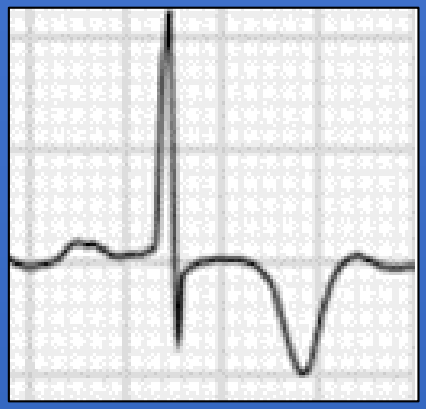
Normal variant asymmetrical T-wave



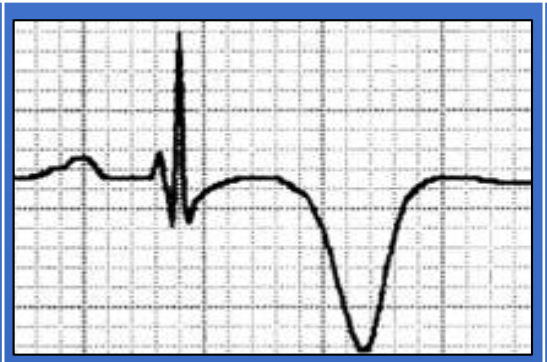
Hyperacute T waves due to anterior STEMI



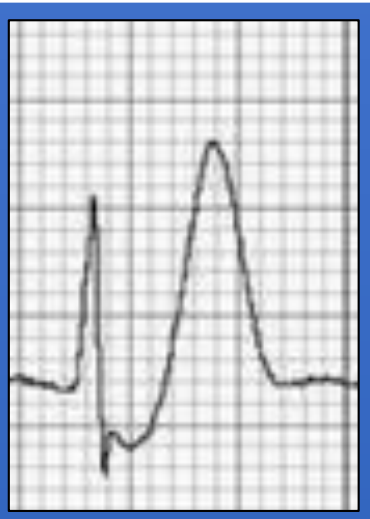
Biphasic T waves due to ischemia, plus-minus T wave or Type A or 2 Wellens syndrome



Wellens' Type B or 1



Ischemic negative, wide base, symmetrical, acute nadir T wave in "seagull wings".

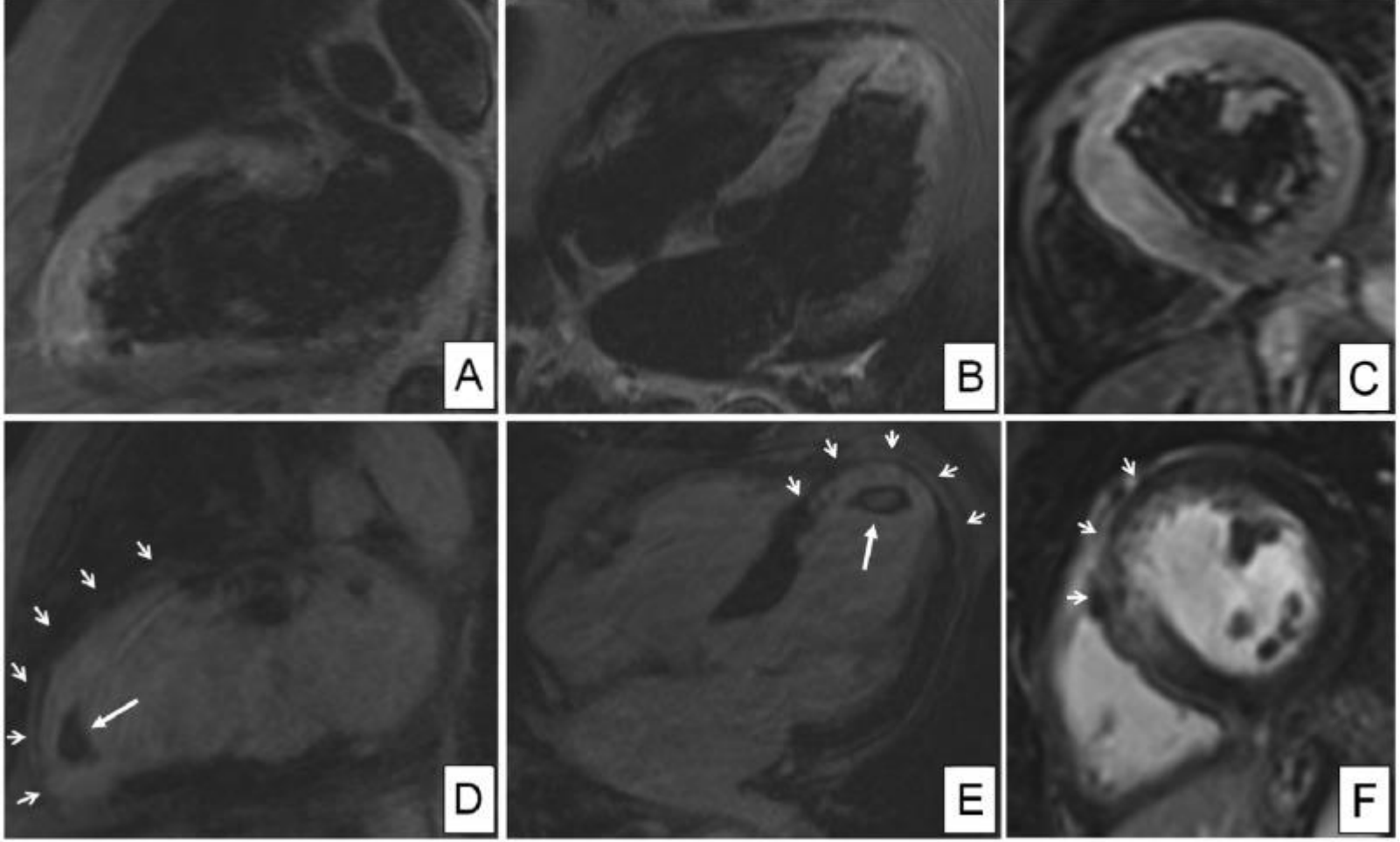


De Winter's T Waves - a STEMI equivalent

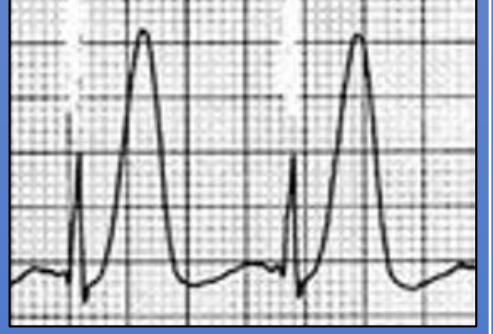
Mechanism of electrocardiogaphic changes

An anatomical variant of the Purkinje fibers, with endocardial conduction delay.

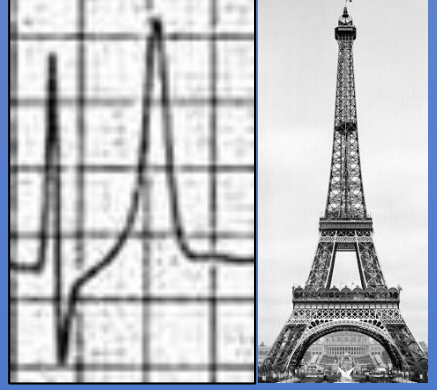
The absence of ST elevation may be related to the lack of activation of sarcolemmal ATP sensitive potassium (K_{ATP} or "sarcK_{ATP}") channels by ischemic ATP depletion, as has been shown in K_{ATP} knockout animal models of acute ischemia. K_{ATP} channels are found in the sarcolemmal membrane; however some may also be found on subcellular membranes. These latter classes of K_{ATP} channels can be classified as being either sarcolemmal ("sarcK_{ATP}"),mitochondrial ("mitoK_{ATP}"), or nuclear ("nucK_{ATP}").
Ischemia activates ATP-sensitive K' channels through intracellular metabolic alterations such as an increase in ADP, and/or a decrease in pH and ATP. In experiments using microelectrodes, the activation of ATP-sensitive K^+ channels has been shown to shorten the action potential duration.-However, the effects of ATP-sensitive K^+ channels on the configuration of ECGs in the in situ beating heart remained unelucidated.
In fact, it is likely that the activation of the K_{ATP} channel during ischemia elicits the accumulation of extracellular K^+,(**Wilde 1990**) which elevates the ST segment via decreasing the resting membrane potential. Therefore, the inhibitory effect of glibenclamide on ST segment elevation after coronary occlusion might be attributed not only to the diminution of the shortening of action potential duration but also to the reduction of K^+ efflux from the myocytes. Peaked and tall T waves represent a hyperacute ECG manifestation of coronary artery occlusion which usually evolves into ST-segment elevation. Using cardiac magnetic resonance (CMR) the myocardial tissue changes underlying an atypical ECG pattern of presentation of LAD occlusion consisting of persistent hyperacute T waves and mild ST-segment depression. This ECG pattern is often associated with the presence of **collateral circulation**, which may modulate myocyte action potential changes in response to ischemia and prevent the appearance of ST-segment elevation. However, CMR findings resembled those of typical anterior myocardial infarction with nearly transmural necrosis in the large myocardial area supplied by LAD. Accordingly, persistent hyperacute T waves should be regarded as an equivalent to ST-segment elevation and immediate reperfusion therapy should be considered.(**Zorzi 2012**)



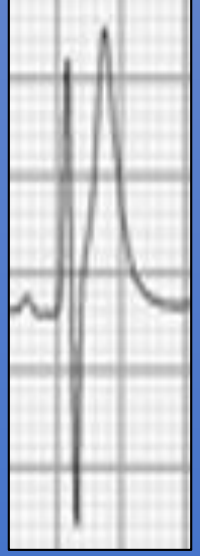
Cardiac magnetic resonance findings. (Top) Increased signal intensity on T2-weighted sequences in the 4-chamber (a), 2-chamber (b) and mid short axis (c) views consistent with transmural myocardial edema of the LV anterior wall, ventricular septum, and apex (area at risk = 37%). (Bottom) T1-weighted inversion recovery post-contrast sequences in the same views (d–f) showing non-transmural late-gadolinium enhancement suggestive of myocardial necrosis (infarct size = 32%) sparing the epicardial layers of the anterior wall and apical segments (arrowheads). Salvaged myocardium was 5%. An apical thrombosis is also evident (arrow).



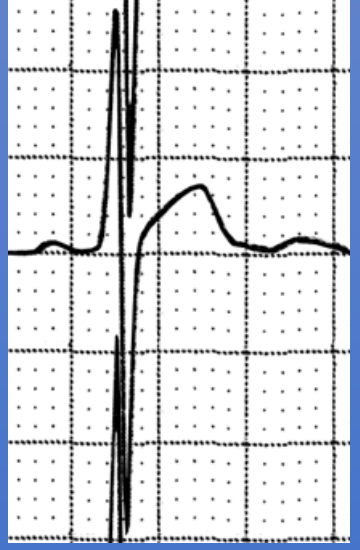
Hyperacute ischemic T-wave: symmetric but not narrow, not pointed, and not tented



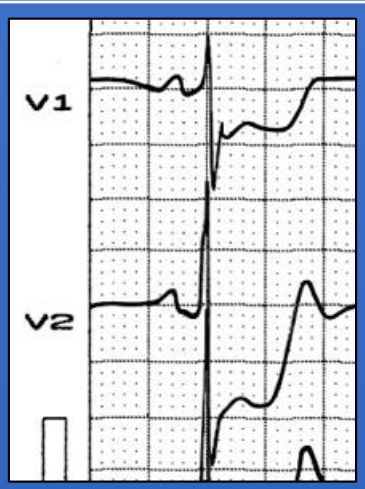
Hyperkalemic T-wave: symmetric, narrow, pointed, and tented T-wave (Chew 2005). Shortened QT interval, ST-segment depression, Eiffel tower T-wave



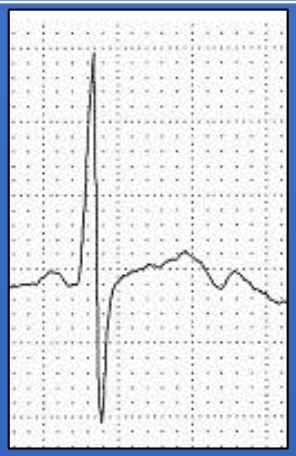
Congenital SQTS



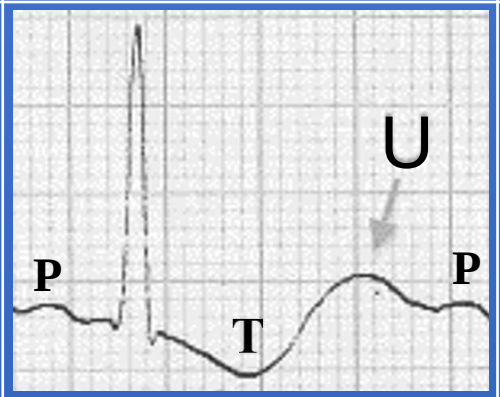
Hypercalcemia: absenc of ST segment



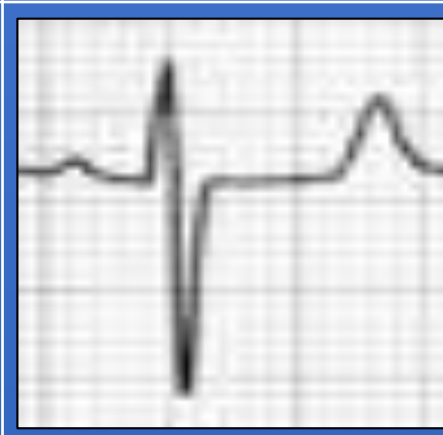
Reciprocal or mirror image AMI ancient dorsal (actual inferior basal)



Camel hump' T waves T-waves with double peak in severe hypokalaemia

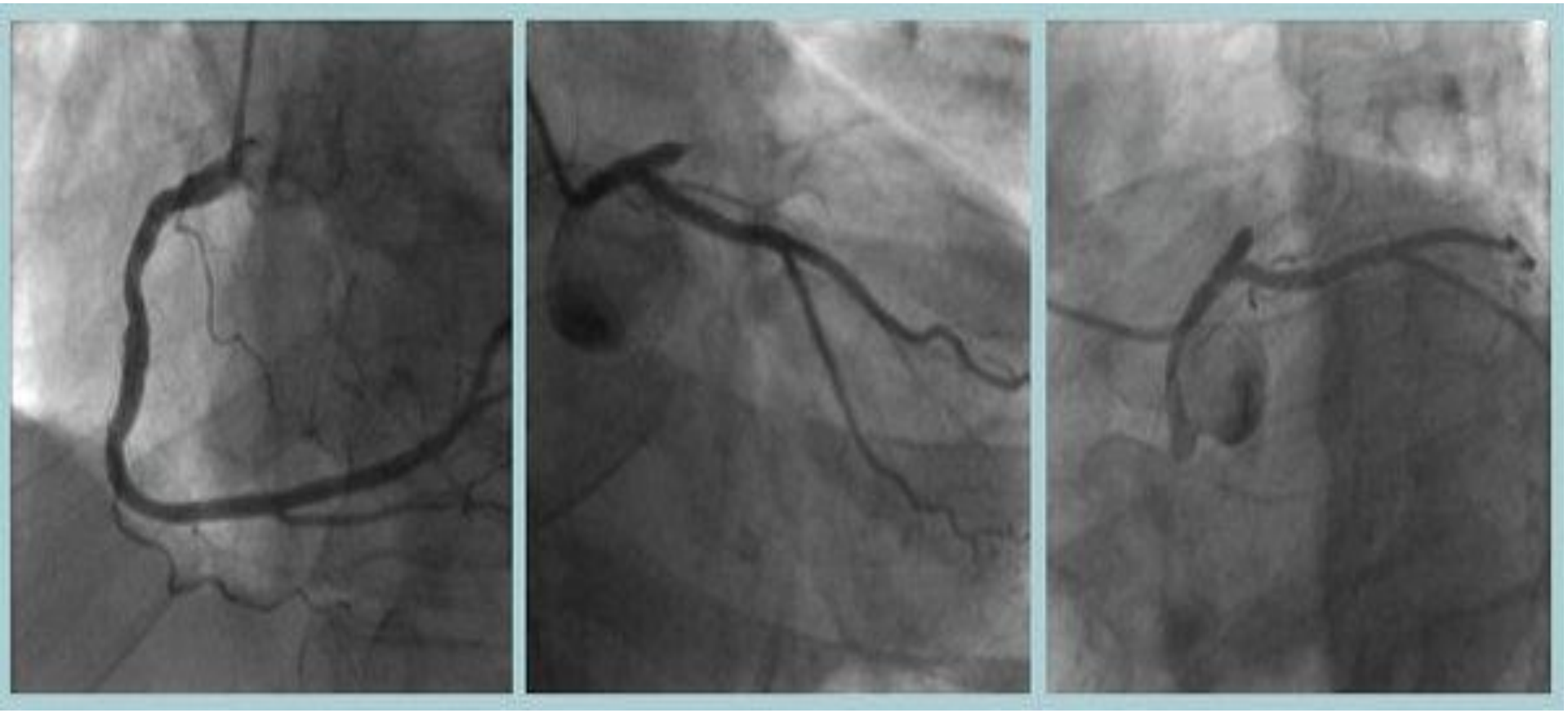


ST depression, inverted T-wave, prominent U-wave, QT prolongation: hypokalaemia



Hipocalcemia + hyperkalemia = uremi ST prolongation and peaked T-wave

Coronary angiogram showing proximal LAD obstruction with thrombus



In 1947 William Dressler and Hugo Roesler studied 27 instances of MI, in which the first ECG was taken as early as one and 15 minutes, and not later than 12 hours, after the onset of symptoms. Follow-up tracings were obtained in all but two of these cases. In 13 cases serial records, taken daily or at intervals of several hours, were available for study. In 25 cases the earliest ECG signs of MI were **high T waves**, the majority of which became inverted in the healing stage of MI. In most of the cases the high T waves were no longer present 24 hours after the attack. In 5 instances where the T waves persisted for several days, early mortality was as high as 60%, as compared with 14% in the cases where the high T waves underwent regression within 24 hours. The high T waves are thought to be part of the injury pattern. They were accompanied by STSE in 21 cases. In 5 instances elevation of ST was either absent or within normal range and only in **one instance huge upright T waves were associated with marked depression of ST.** In the majority of the cases high T waves preceded the development of significant changes in the QRS complex. The latter usually appeared within 24 hours after the onset of symptoms, when the high T waves were already in the regressive stage. In one instance characteristic changes in QRS did not appear until 6 days after the attack, and in 2 instances they failed to develop at all. In 5 cases high T waves were not associated with abnormal elevation of ST, nor with significant changes in QRS. Thus, they represented the leading diagnostic sign in the early stage of MI.

Thirty-two consecutive patients who initially had horizontal or downward-sloping ST segment depression confined to the precordial leads were studied by Sclarovsky et al (**Sclarovsky 1998**). Patients were divided into two groups: **Group A** included 21 patients with horizontal or downward-sloping ST depression with peaked positive T waves; **Group B** comprised 11 patients with peaked negative T waves and downward or horizontal ST depression. The incidence of AMI) was similar (group A 38.1% vs group B 36.4%; p greater than 0.05). In-hospital mortality was much more significant in group B. Coronary arteriography was performed in 31 patients. Of the 10 patients in group B who were catheterized, seven (70%) had left main occlusion. Of the 21 patients in group A, none had a significant left main lesion ($p = 0.001$), although eight (38.1%) had single-vessel disease ($p = 0.05$). Thus the ECG pattern of horizontal or downward-sloping ST depression passing into a peaked negative T wave identifies a subgroup of high-risk patients in whom the prognosis is poor once AMI occurs. Early catheterization is recommended when this ischemic pattern is apparent on the ECG.

Management

- Rapid recognition of pattern
- Cath lab activation
- Education of consultants may be necessary due to unfamiliarity

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