# ECG

# WELLENS' SYNDROME ON ECG : OCCLUSION MI

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## OUTLINE

**Introduction**
Wellens' syndrome represents the reperfusion state of atuo-thrombolysed LAD circulation without having the impact of myonecrosis (Type A and B)

**Historical background : with Red Tails Story- a pre-infarction state**

**A diagnostic approach to Wellens' syndrome**

**Pathophysiology**

- Wellens' syndrome follows a temporary obstruction of the LAD coronary artery which is commonly caused by the rupture of an atherosclerotic plaque , with subsequent thromobolysis before complete myocardial infarct has set in.
- The exact mechanism of the ECG changes in Wellens' syndrome is still unknown but this is postulated that it is caused by **transmural ischemia-reperfusion mechanism leading to myocardial oedema.**
- There is a red alert because 75% of such cases may turn into acute anterior wall myocardial infarction due to reocclusion of proximal LAD coronary territory.

(A concept of Pseudo-Wellens' syndrome also mentioned)

**Understanding Wellens' syndrome on the basis of ventricular action potential**

**Diagnostic leads for T-wave changes in Wellens' syndrome**

**Differential diagnosis with inverted T wave in adult patients**

**Illustration of Wellens' syndrome by ECGs**

**Take Home message**

**References**

# Wellens' syndrome on ECG : Occclusion MI

**A Narrative Review**

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**Two phenomena** – protection and destruction have been chasing each other since time immemorial. Protection keeps us alive and luminous in life– upholding the Bio-tissues to run smoothly with hero-force. On the contrary , the destruction does the reverse and the hour of adversity sets in life. These are two sides of the same coin.

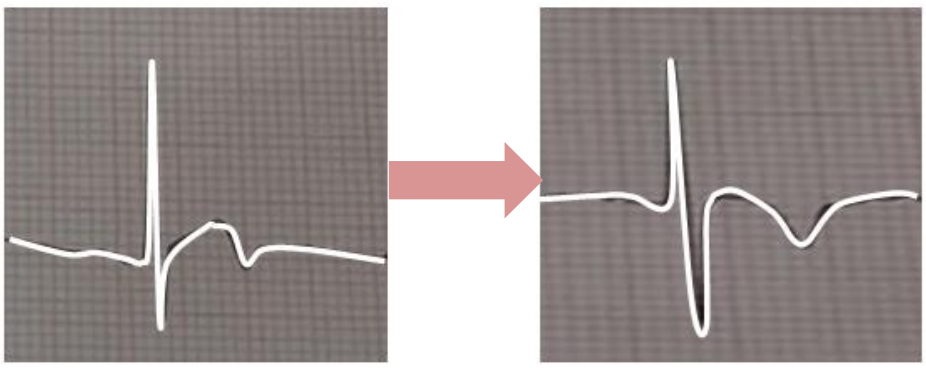
- ☐ **O heart thou is protected from the throatling hands of thrombosis in coronary circulation by the process of auto-thrombolysis with reperfusion – but might be ruined again by the gloomy blow with the resetting of thrombosis.**
- ☐ **Protection by auto-thrombolysis is the comedy and destruction by reocclusion is the tragedy.**

So is the basis of Wellens' syndrome – protection is chased by destruction. Pre-infarction state may be turned into true infarction state.

## 1. Introduction

Wellens' syndrome denotes an abnormal electrocardiography (ECG) pattern – biphasic or deeply inverted T-waves mainly on chest leads V2 and V3. To start with there is a critical occlusion of proximal LAD artery presenting as unstable angina but followed by auto-thrombolysis of so formed clot resulting in pain-free period with so typical T-waves changes. The artery may be reoccluded resulting in myocardial infarction.

**Nowadays this entity is kept under the heading of 'Occlusion MI'.**



**Biphasic T-wave (the initial positivity and terminal negativity)**

**Type A Wellens' Syndrome**

**Deeply inverted and symmetrical T-wave**

**Type B Wellens' Syndrome**

**Fig.** 1.1

This is worthwhile to mention here that the patients with this syndrome come to the clinicians in a pain-free state and with normal or slightly elevated cardiac markers with the recent history of preceding anginal pain in the background.

**This syndrome results from temporary obstruction of LAD coronary artery , usually caused by the rupture of an atherosclerotic plaque leading to LAD occlusion with subsequent auto-thrombolysis of the clot before complete myocardial infarction sets in. THIS IS CARDIO-PROTECTIVE PHENOMENON.**

**This is also to be noted here that 75% of the patients with such ECG findings would lapse into acute anterior wall myocardial infarction usually within a week. THIS IS THE CARDIO-DESTRUCTIVE PHENOMENON.**

**Protection is the comedy and destruction is the tragedy**. To prevent such a tragedy, intervention by urgent PCI (percutaneous corornay intervention) is needed.

## 2. Historical background with Red Tails story

A step towards the understanding of the history behind the evolution of Wellens' syndrome might enable us to deal more efficiently with the present problem.
Deep in evolution there are so many gallops of discoveries – red tails events in the history bringing a renewed knowledge over the surface which paves the pathway towards a better understanding of this syndrome.

**Dr. Zwaan , Wellens et al (1982) , a group of cardiologists from Netherlands** reported some notable findings , while observing their patients admitted for unstable angina.
Their findings are summarized as below :

- ☐ They described a subgroup of patients with an unstable angina (26/145 = 18%) having typical T-wave changes in precordial leads with negative cardiac enzymes but with poor outcome.
- ☐ All patients received relief of chest pain on conservative management but 8 of the first 9 patients developed anterior mycoardial infarction and three died – a poor prognostic outcome (The average time for infarction following such ECG changes was ranging from Day 1 to 23 ; with the average of 8.5 days).
- ☐ Some patients who underwent cardiac catheterization with coronary angiography were found to have greater than 90% stenosis in the proximal LAD.

**In a second prospective study initiated by de Zwaan et al** , 180 out of 1260 (14%) patients for unstable angina were detected to have typical T changes on ECG. All 180 patients underwent catheterization and all were found to have at least 50% blockade of LAD.
**In nutshell** as per their observation , there exists a subgroup of unstable anginal patients showing typical T-wave changes on ECG with poor outcome - such patients are usually turned to anterior myocardial infarction within a few days. **That's why , this ECG pattern with typical T changes are in particularly having high risk of developing an extensive myocardial infarction representing as Red Tails story.**

## 3. A diagnostic approach to Wellens' syndrome

It is obvious by the foregoing discussion that **Wellens' syndrome represents the reperfusion state of thrombolysed LAD circulation without having the impact of myonecrosis**. Therefore , the definition of this syndrome must include **the evidence of reperfusion-injury on ECG as T-wave changes without the evidence of myonecrosis.**

**Diagnostic criteria for Wellens' syndrome include :**

- **Reperfusion injury-related repolarization**
  Biphasic T-waves (with initial positivity and terminal negativity) or deeply inverted T-waves are commonly seen on leads V2 and V3. However , if the lesion is more proximal in the LAD , the T-wave changes would be more widely spread along the precordial leads.
  These T changes on ECG are present with a pain free state.

PLUS

- **Without the evidence of acute anterior wall myocardial infarction**
  such as
  - ECG without Q wave with isoelectric or minimally elevated ST segment - <1mm and with the absence of precordial poor R wave progression.
  - Normal or slightly elevated cardiac marker (in one prospective study, only 12% of such patients had elevated cardiac enzymes, and these elevations were less than twice the upper limit of normal).
- **Recent history of unstable angina coinciding with the previous temporary LAD occlusion.**

**It is to be noted that there is a lack of universal definition for a preserved precordial R-wave progression but common criteria include :**

- R-wave > 2-4 mm in V3 or V4
- R-wave in V4>V3 or V3>V2
- R-wave in V3 $\geq$ 3mm

There are two patterns of T-wave in Wellens' syndrome : namely **Type A** biphasic T-wave with initial positivity and terminal negativity (approximately in 25% of cases) and **Type B** with deeply and symmetrically inverted T-wave (approximately in 75% of cases). **Type A T-waves are usually evolved into Type B T-waves**. These T-wave abnormalities may be persisting for hours to weeks.

Wellen's syndrome is not always an acute process – can develop over days to weeks. Already mentioned that this ECG pattern develops when the patient is not experiencing the chest pain. The recurrence of chest pain indicates the onset of impending acute anterior wall myocardial infarction.

## 4. Pathophysiology

The pathophysiology of Wellens' syndrome can be summarized as follows :

- ❖ Wellens' syndrome follows a temporary obstruction of the proximal LAD artery which is commonly caused by the rupture of an atherosclerotic plaque , with subsequent thromobolysis before complete myocardial infarct has set in.
- ❖ The exact mechanism of the ECG changes in Wellens' syndrome is still unknown but this is postulated that it is caused by **transmural ischemia-reperfusion mechanism leading to myocardial oedema.**
- ❖ There is a red alert because 75% of such cases may turn into acute anterior wall myocardial infarction due to reocclusion of proximal LAD coronary territory.

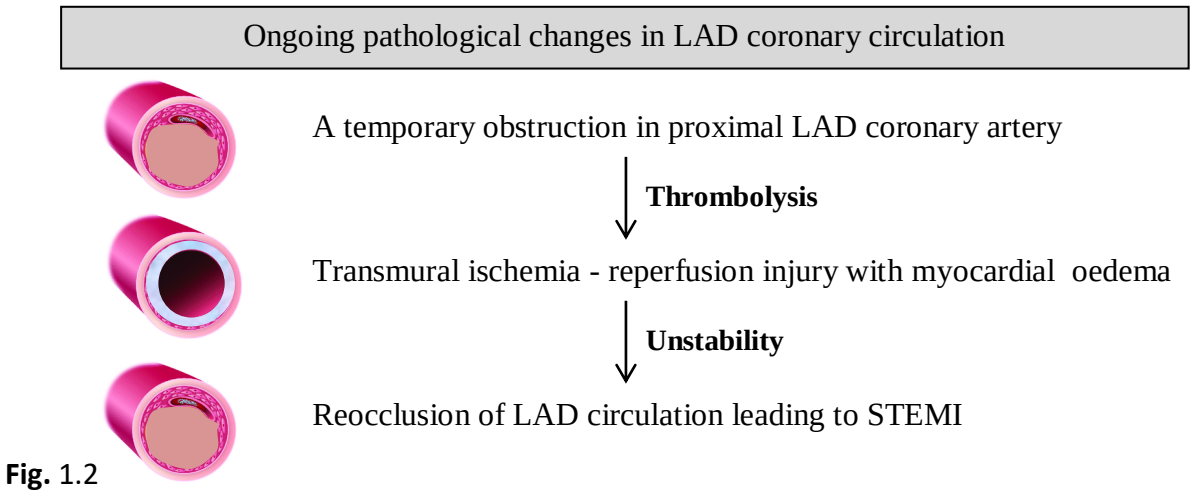



**Fig.** 1.2

**Evolution of ECG changes in the light of pathological scenario**

The following sequence of events is thought to be occuring in Wellens' syndrome :

| Pathological changes | ECG changes |
|---|---|
| ➢ Temporary obstruction in proximal LAD coronary artery | This phase may cause anginal chest pain. It may not be successfully captured on ECG – showing mild ST changes, or negative deflection of the T-wave in V2 and V3. |
| ➢ Reperfusion oedema ± pre-hospital aspirin. | There is resolution of the chest pain - ST changes improve with typical biphasic or inverted T-waves (If the artery remains open the biphasic T-wave would evolve to deeply inverted T-wave). |
| ➢ LAD can reocclude at any time | With the recurrence of anginal chest pain , the ST segment and T-wave pattern appear to normalize into hyperacute T-wave. So called **"Pseudo normalization"**. This is a sign of hyperacute STEMI. If the artery remains occluded , the patient can develop an evolving anterior STEMI with its characteristic features on ECG. |
| ➢ "Stuttering pattern" with intermittent reperfusion and reocclusion | As alternating ECGs patterns – Wellens' and Pseudo normalization. |

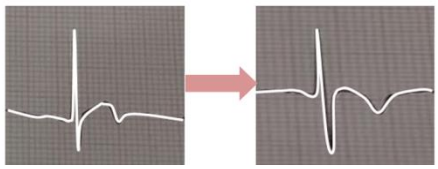
**NB** : These events as discussed are not limited to the anterior chest leads only – similar changes might also be seen in the inferior or lateral leads due to the occlusion of RCA or LCx in addition.

**Pseudo-Wellens' syndrome**

Coronary artery spasm in LAD may also be responsible for the genesis of Wellens' syndrome , defined as Pseudo-Wellens' syndrome – rarely reported in the literature , after the resolution of the spasm. Coronary flow is restored leading to reperfusion injury-related repolarization abnormalities depicted on ECG in precordial leads with typical biphasic or inverted T-waves, as discussed before.

The causative factor for 'Pseudo-Wellens' syndrome' may include the use of Cocaine / Marijuana and another rare cause is myocardial bridging in which the coronary artery tunnels under a bridge of myocardium resulting in LAD coronary artery stenosis. Additionally, the Wellens' pattern can be seen in Takotsubo cardiomyopathy, which is thought to be the result of myocardial oedema.

## 5. Understanding Wellens' syndrome on the basis of ventricular action potential



Biphasic T-wave ( initial positivity and terminal negativity)

Deeply inverted and symmetrical T-wave

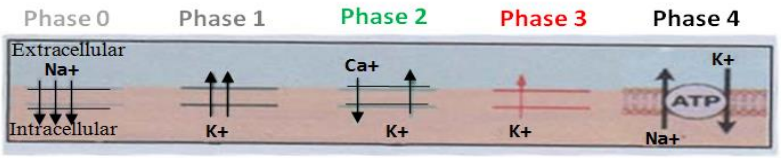


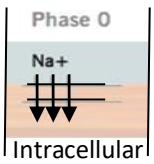

**Fig.** 1.3

Type A Wellen's syndrome  Type B Wellen's syndrome

The pathophysiologic background in Wellens' syndrome :

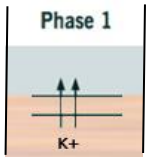
- A temporary obstruction of the LAD coronary artery , commonly caused by the rupture of an atherosclerosis plaque , the subsequent auto-thrombolysis before complete myocardial infarct sets in.
- The next is transmural ischemia – reperfusion injury leading to myocardial oedema.

**NB** : The description as noted below is in context with Wellens' syndrome (pain-free period).

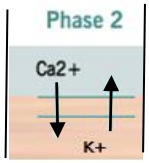



☐ **Phase 0** : This phase corresponds to depolarization reflected on ECG as QRS wave.

**Since there is reperfusion in the initial stage without myocardial infarction , the changes recorded are absence of pathological Q wave without poor R wave progression.**




☐ **Phase 1**: Normally the phase corresponds to the outward movement of $K^+$ channels for a transient period – inscribed by a J notch on the descending limb of the QRS complex. **No abnormality detected during this phase.**



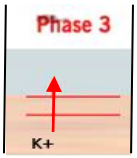

☐ **Phase 2** (plateau phase) : Normally during this phase , there is a mutual exchange of $Ca^{2+}$ and $K^{+}$ ions across the cardiac membrane travelling across a homogenous ventricular gradient with no current of injury resulting in isoelectric segment on ECG (Corresponding to ventricular contraction). This is inscribed as isoelectric ST segment on ECG.

Minimally elevated ST segment

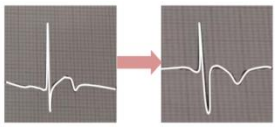
**Isoelectric or minimally elevated ST segment < 1mm**

**Reasoning** :

- Isoelectric ST segment is inscribed due to the preservation of myocardium without myonecrosis.
- Minimally elevated ST segment (<1mm) might represent the very minimal residual impact by the temporary occlusion of LAD artery.




☐ **Phase 3** : Normally this phase corresponds to repolarization being reflected on ECG in the form of upright T-wave.



Biphasic T-wave ( initial positivity and terminal negativity) Type A Wellen's syndrome

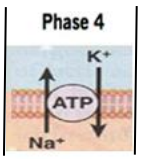
Deeply inverted and symmetrical T-wave

Type B Wellen's syndrome

Biphasic T-wave (with initial postivity and terminal negativity) may be followed by deeply inverted T-wave.

**Reasoning** :

- **Myocardial ischemia – reperfusion injury results in local myocardial oedema** which can change the direction of ongoing repolarization process. Due to the delay in passing the current through this oedematous myocardium – the current flows uniformly through the opposite direction i.e. away from the subepicardial zone. This produces symmetrical deep T-wave inversion.
- In the initial stage of partial oedematous myocardium the first half of the wave with initial positivity and the terminal part with negativity , as seen in Type A Wellens' syndrome.




☐ **Phase 4** : This phase is the resting phase brought about by '$Na^{+}K^{+}$ -ATPase' mechanism.

**No abnormality detected during this phase.**

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## 6. Diagnostic leads for T-waves changes in Wellens' syndrome

The diagnostic leads for T-waves of Wellens' syndrome are V2 and V3 , corresponding with a lesion in between the first (S1) and second (S2) septal branches of the LAD. However , if the lesion is more proximal in the LAD , the T-wave changes would be more widely spread along the precordial leads ranging from V1 to V6.

(The sequence of events related to Wellens' syndrome is not only limited to anterior chest leads entirely – similar changes might be observed also in the inferior or lateral leads due to the occlusion of RCA or LCx in addition).

The entire concept is depicted with the following sketch :

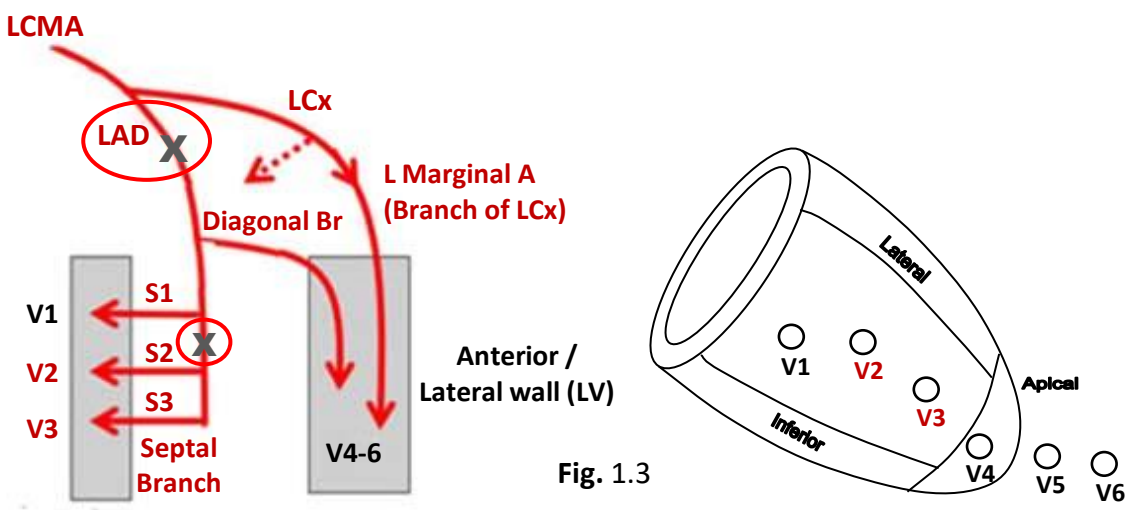



**Fig.** 1.3

## 7. Differential diagnosis with inverted T-wave in adult patients

Wellens' syndrome has a typical appearance on ECG. But at times it might be confused with other ECG entities , some of them are discussed below (a thorough history is a must to reveal the true pathological nature).

☐ **Primary T-wave inversion**

T-wave inversion , without concurrent changing in the orderly sequence of activation – as a result of global or segmental pathological processes that affects ventricular repolarization. Here T-waves are symmetrically inverted.

- **Persistent Juvenile T-wave pattern** : Normal variant , more commonly in young women , especially African , American women – inverted T-waves (biphasic T-waves in some cases) appear in leads V1-V3. The inverted T-waves are symmetrical less than 3 mm deep. This pattern continues into young adulthood , and therefore , the name 'Persistent Juvenile pattern'.
- **'Normal Variant' STE sometimes with biphasic T-wave** : Always seen in young male population , particularly African, American males , with very high QRS voltage with notching at J-point (fish-hook) with ST segment being concave upwards (as opposed to isoelectric or convex ST segment in Wellens' syndrome) . QT interval tends to be short.
- **Pulmonary embolism** : Inverted symmetrical T-waves in right-sided chest leads with other signs of right heart strain , such as right axis deviation , RBBB or the 'S1Q3T3' pattern and associated sinus tachycardia , etc.
- **CNS injury** (stroke , subarachnoid hemorrhage or intracereberal hemorrhage) T-wave inversion very similar to those seen in Wellens' syndrome but here also associated ST segment depression , QTc prolongation with associated U waves.

- **Acute coronary insufficiency** : Sometimes presented as deep symmetrical T-wave inversion with an isoelectric ST segment that is usually bowed upward , history is important in this context.
- **Digitalis effect** : T-wave abnormality is biphasic T-waves with initial negative deflection and positive terminal deflection (reverse the pattern seen in Wellens'), usually seen in leads with prominent R wave (V4-V6). Most commonly associated abnormality is a downsloping ST-segment with a characteristic scooped appearance , being continuous in the first part of T-wave.
- **Hypokalemia** : Biphasic T-wave initially negative with terminal positivity due to the merging of inverted T-wave with the positive U-wave with prolonged QTc.

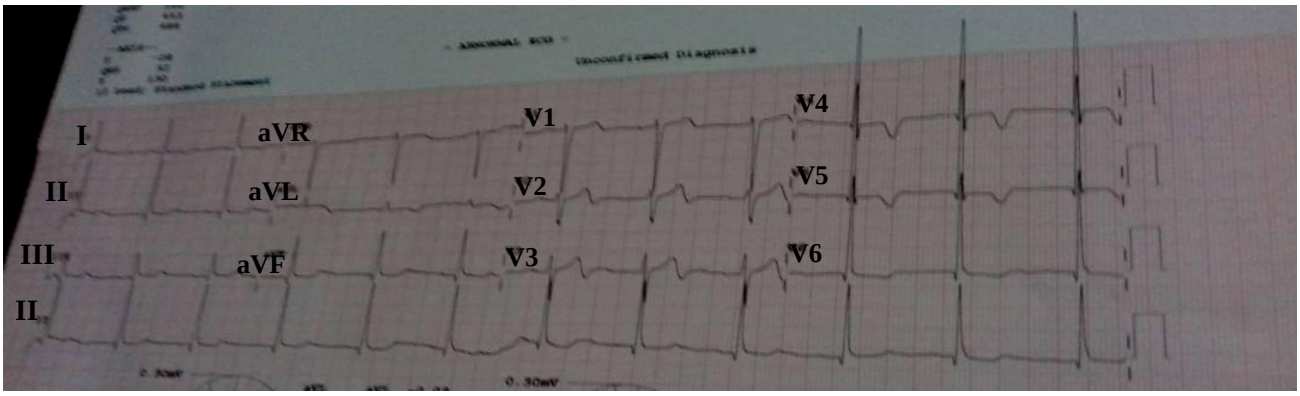
☐ **Secondary T-wave inversion**

T-wave changes result from aberrant ventricular activation. Here T-wave inversion is asymmetrical in nature (with a gradual initial downslope and abrupt return to the baseline).

Examples include bundle branch block , ventricular pre-excitation (WPW syndrome), uneven ventricular hypertrophy etc.

## 8. Illustration of Wellens' syndrome by ECGs

**ECG 1 : Type A Wellens' syndrome**




**Source** : : Prof. Dr. A.N. Rai , Former Prof. & Head Medicine and Principal ANMMCH , Gaya Bihar ; Chairman AIMS, Gaya

This ECG was recorded in a middle aged smoker male with a preceding history of chest pain with Trop I negative.

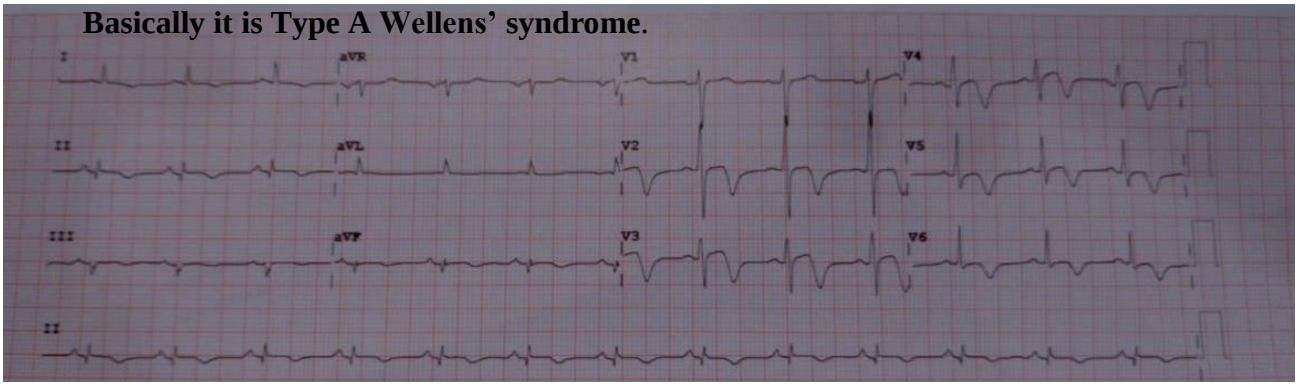
**Findings :**

- Biphasic T-wave with initial positivity and terminal negativity in leads V1-V3.
- Absence of precordial poor R wave progression.
- T-waves inversion in leads V4-V5 , also in lead aVL.

**Discussion :**

- These T-wave changes are more widely spread along the precordial leads. Therefore , the lesion is more proximal in the LAD.
- Since the lesion is admixture of both biphasic and negative T-wave in precordial leads, this indicates the lesion is progressing towards the next phase Type B.

## ECG 2 : Type B Wellens' syndrome



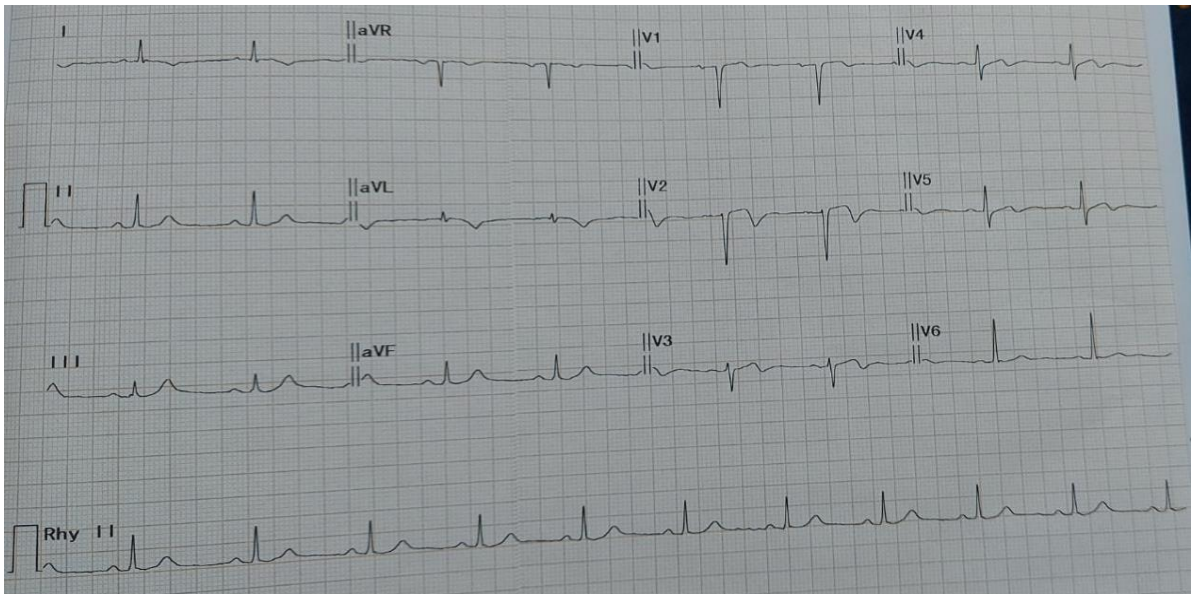

**Source** : : Prof. Dr. A.N. Rai , Former Prof. & Head Medicine and Principal ANMMCH , Gaya Bihar ; Chairman AIMS, Gaya

This ECG shows :

- Deeply inverted T-waves in leads V1-V6 with minimally elevated ST segment less than 1 mm.
- The absence of precordial poor R wave progression
- Troponin I : normal

**Discussion :**
**The findings are consistent with Type B Wellens' syndrome**.

## ECG 3




**Source :** Dr. SS Lakshmanan , Senior Consultant Physician and Cardiologist , Chennai

A male diabetic patient aged 60 years with anginal chest pain and trop I positive.

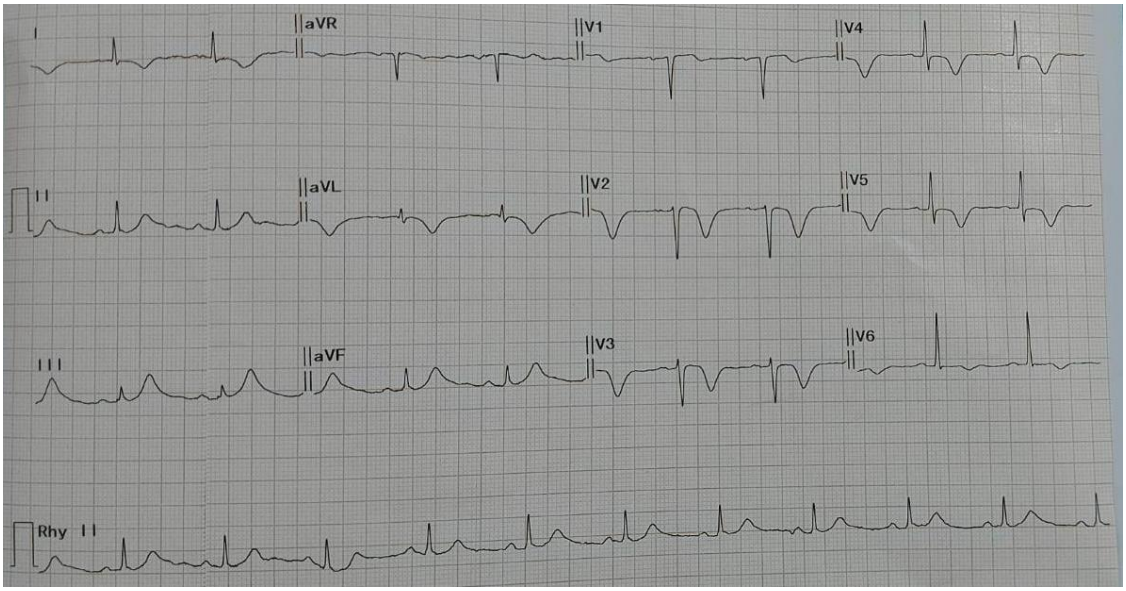
**Finding :**

- Biphasic T-wave with mild initial positivity and terminal negativity in leads V1-V5 with minimally elevated ST segment , lesion also extending towards lead I and aVL.

**Discussion :**

- These findings point towards the involvement of the proximal LAD coronary artery along with LCX as well.
- In one prospective study , 12% of patient with Wellen's pattern on ECG were found to have elevated cardiac enzymes – less than twice the upper limit of normal.

**Second ECG of the same patient :**




**Findings :**

- Deeply inverted T-waves through V1-6 , I and aVL as well with minimally elevated ST segment.
- Preservation of the precordial R wave progression , since R wave > 4 mm in lead V4 and R wave in V4 >V3.

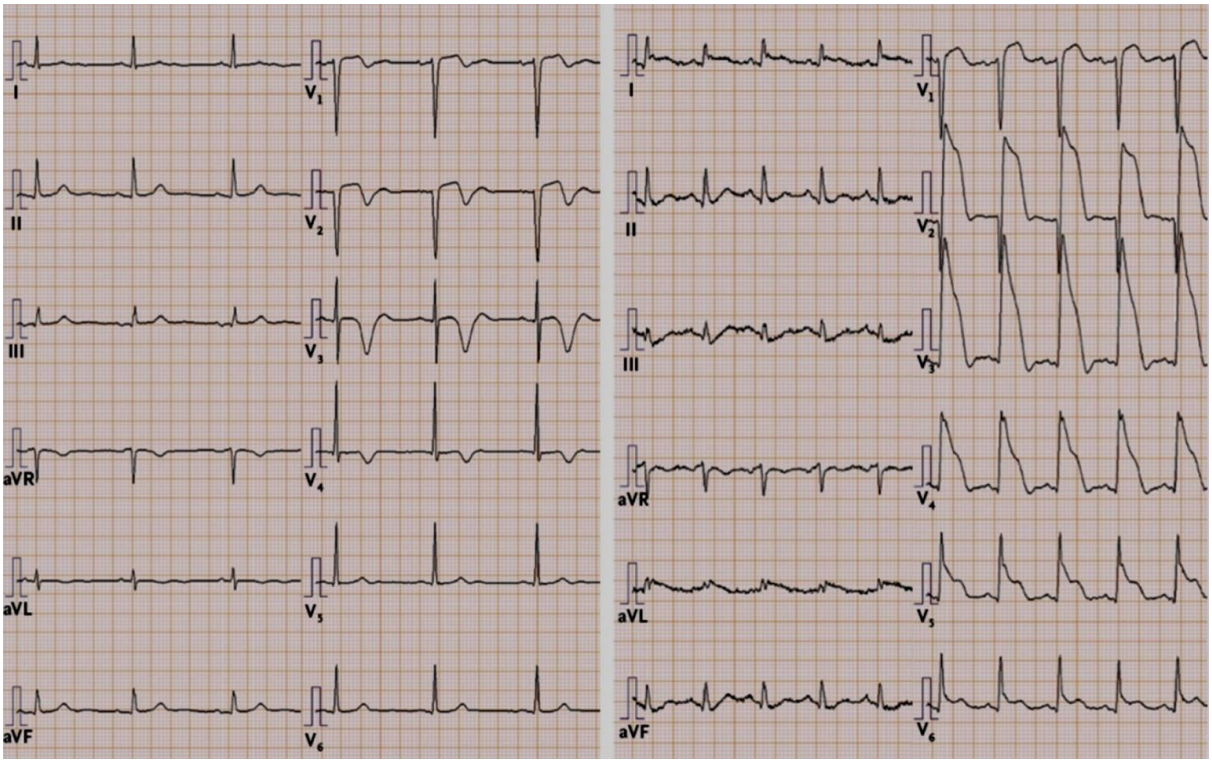
**It is to be noted that there is a lack of universal definition for a preserved precordial R-wave progression but common criteria include :**

- R-wave > 2-4 mm in V3 or V4
- R-wave in V4>V3 or V3>V2
- R-wave in V3 ≥ 3mm

**In conclusion this ECG is consistent with Type B Wellens' syndrome**.

**ECG 4 : Turning of Type B Wellens' syndrome into acute anterior ST elevation myocardial infarction**

There is a tendency to reocclude LAD branch of coronary circulation , the accurate reasoning of this reocclusion is still remaining in darkness. This fact should be emphasized here that undiagnosed cases might be missing the opportunity of intervention by PCI. This may result in MI catastrophe - illustrated by ECGs on the next page.




**Type B Wellens' syndrome** → **Acute ST elevation MI**

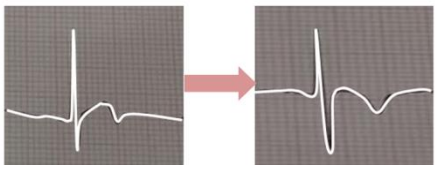
**Source :** Dr. N.K Singh , Director , Diabetes and Heart research centre , Dhanbad , Editor , www.cmeindia.in posted on CME INDIA.

MI with "Shark Fin" pattern – formed by the confluence of QRS and T wave as a result of extreme ST elevation.

## 9. Take Home message

- ☐ Wellens' syndrome represents the reperfusion state of atuo-thrombolysed proximal LAD circulation without having the impact of myonecrosis.
- ☐ This syndrome results from temporary obstruction of proximal LAD coronary artery , usually caused by the rupture of an atherosclerotic plaque leading to LAD occlusion with subsequent auto-thrombolysis of the clot before complete myocardial infarction sets in.

  75% of such patients would lapse into acute anterior wall myocardial infarction usually within a week.
- ☐ On ECG Wellens' syndrome denotes an abnormal electrocardiography (ECG) pattern – biphasic or deeply inverted T-waves mainly on chest leads V2 and V3



Biphasic T-wave ( initial positivity and terminal negativity)
Type A Wellen's syndrome

Deeply inverted and symmetrical T-wave
Type B Wellen's syndrome

If the lesion is more proximal in the LAD , the T-wave changes are widely spread along the precordial leads.

These T-changes on ECG are present with a pain free state.

**Fig.** 1.4

☐ Diagnostic criteria for Wellens' syndrome include :

- Reperfusion injury-related repolarization , as T-changes on ECG (discussed on the preceding page)

  PLUS

- Without the evidence of acute anterior wall myocardial infarction such as
  - ECG without Q wave with isoelectric or minimally elevated ST segment - <1mm and with the absence of precordial poor R wave progression.
  - Normal or slightly elevated cardiac marker (in one prospective study, only 12% of such patients had elevated cardiac enzymes, and these elevations were less than twice the upper limit of normal).
- Recent history of unstable angina coinciding with the previous temporary LAD occlusion.

☐ Pseudo-Wellens' syndrome , as with Coronary artery spasm in LAD , the use of Cocaine / Marijuana and another rare cause is myocardial bridging over the concerned proximal LAD , sometimes seen with Takotsubo cardiomyopathy, due to accompanying myocardial oedema.

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