

# Giant Negative T waves of Indeterminate Origin

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## SUMMARY

Giant negative T waves or massive T wave inversion are defined by the presence of negative T waves with an amplitude  $\geq 1$  mV in at least two consecutive electrocardiographic leads. A 66 year-old asymptomatic woman who underwent a routine medical examination was admitted in the CCU due to the presence of giant negative T waves in leads I, II, aVL, aVF and from V1 to V6, and a left anterior hemiblock. She had a history of type IV dyslipemia and mild hypertension. The ischemic etiology of T-wave inversion was ruled out. During follow-up, T waves progressively became less negative and 45 days later they normalized. The origin of T-wave inversion could not be established.

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## Key words >

Giant Negative T Waves - Left Anterior Hemiblock - Indeterminate Origin

## Abbreviations >

<b>RBBB</b>	Right bundle branch block	<b>T<sub>3</sub></b>	Triiodothyronine
<b>ECG</b>	Electrocardiogram	<b>T<sub>4</sub></b>	Thyroxine
<b>VPB</b>	Ventricular premature beat	<b>PE</b>	Pulmonary embolism
<b>LAH</b>	Left anterior hemiblock	<b>TSH</b>	Thyroid-stimulating hormone
<b>CMRI</b>	Cardiac Magnetic Resonance Imaging	<b>CCU</b>	Coronary care unit
<b>MRI</b>	Magnetic Resonance Imaging		

## CASE REPORT

We describe a 66-year old female patient with a history of dyslipemia and mild hypertension. An ECG performed in August 2000 showed a left anterior hemiblock, isolated ventricular premature beats and normal T waves. The exercise stress test was normal and a 24-hour Holter monitoring revealed the presence of frequent monomorphic ventricular premature beats with long coupling interval and right bundle-branch block (RBBB)-like and left anterior hemiblock (LAH)-like QRS pattern. The patient started treatment with atenolol 25 mg/day, amiodarone 200 mg/day, aspirin 100 mg/day and gemfibrozil 600 mg/day. She made medical visits twice a year; amiodarone was discontinued in August 2002 due to absence of arrhythmias in the last 24-hour Holter monitoring. On November 12, 2004, she underwent a routine medical examination and was admitted in the CCU due to the presence (Figure 1) of giant negative T waves in I, II, aVL, aVF and from V1 to V6, and a left anterior hemiblock, in sinus rhythm. She was hospitalized with a diagnosis of acute transmural ischemia. During hospitalization the ECG did not show significant abnormalities in the T wave and cardiac biomarkers were normal. Doppler echocardiography was also normal. On

November 13, 2004, stress Tc-99m sestamibi ECG-gated SPECT images showed absence of hypoperfused areas or wall motion anomalies. The patient did not complain of angina or anginal equivalents - dyspnea or heart failure. Basal negative T waves became positive during immediate exercise recovery (T-wave pseudonormalization); six minutes later they returned to the basal condition. Monomorphic ventricular premature beats (ITT 20500, 10 mets) were probably due to anomalies in ionic channels, to metabolic abnormalities or secondary to minimal anomalies in ventricular depolarization.

On December 7, cardiovascular magnetic resonance imaging with first-pass perfusion and late gadolinium enhancement was performed to detect ischemia and viability, with normal results (Figure 3).

It was unnecessary to perform a coronary angiography.

The patient was discharged and ECGs were recorded every week; T wave remained stable. On December 30, 2004 T waves were completely normal (Figure 2). In August 2005 a brain MRI was normal.

The patient is currently being followed-up; T wave inversion has not occurred again; however, she developed an intermittent complete bundle branch block that subsequently evolved to permanent block.

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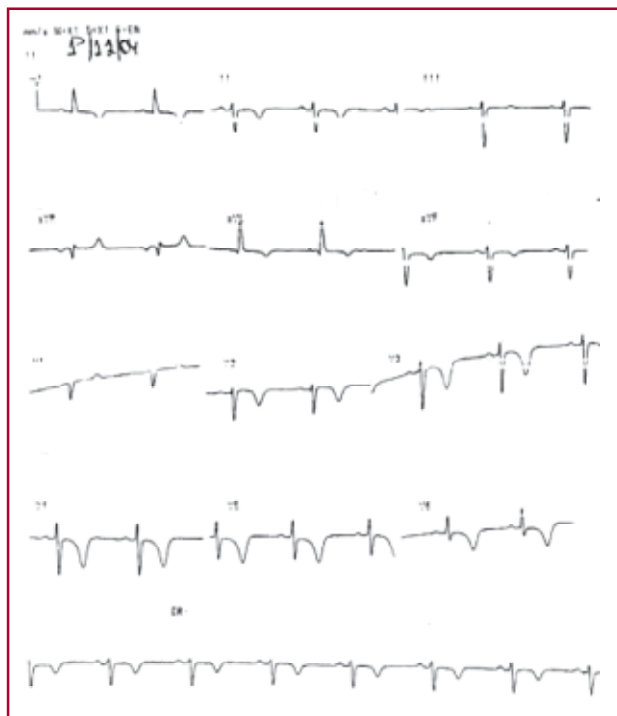
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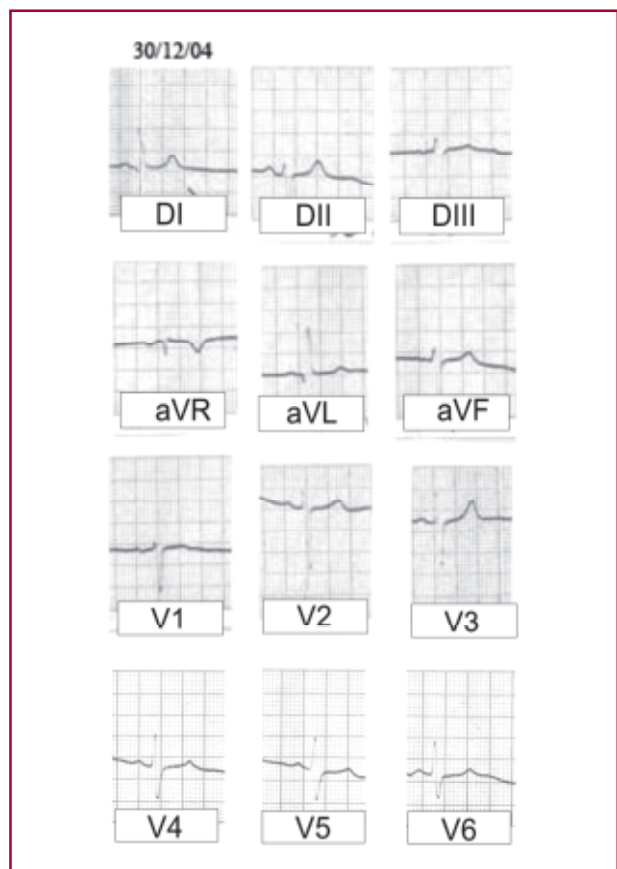
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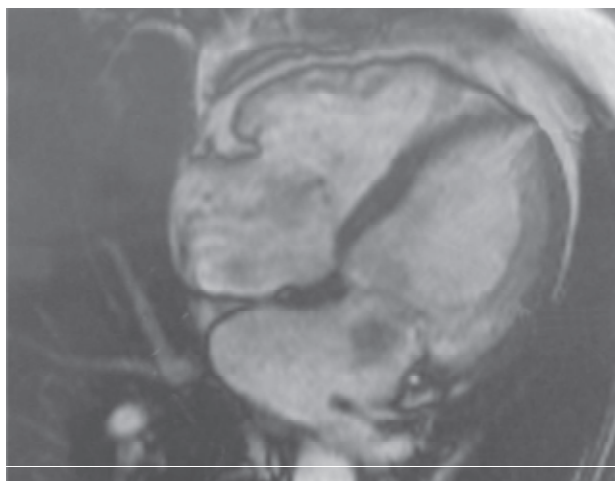
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**Fig. 1.** ECG. Sinus rhythm. LAH. Giant negative T waves in waves in I, II, aVL, aVF and from V1 to V6.



**Fig. 2.** ECG. Sinus rhythm. LAH. T wave has normalized.



**Fig. 3.** Normal cardiovascular magnetic resonance imaging.

## DISCUSSION

Giant negative T waves or massive T wave inversion are defined by the presence of negative T waves with an amplitude of  $\geq 1$  mV in at least two consecutive electrocardiographic leads, especially in the anterolateral wall. The following conditions have been associated with massive T wave inversion:

1. Acute pulmonary embolism with or without pulmonary hypertension.
2. Chronic recurrent pulmonary embolism with or without pulmonary hypertension.
3. Electroconvulsive therapy. (3)
4. Intracranial hypertension. (3)
5. Increased sympathetic tone. (8)
6. Hypertrophic cardiomyopathy.
7. Alcoholism. (4)
8. Disorders of fluid and electrolyte balance. (4)
9. Hypoparathyroidism. Hyperparathyroidism. (2)
10. Ischemic heart disease. (10)
11. Diffuse lacunar brain infarctions.
12. Electrotonic modulation of the T wave. (1, 10)
13. Sudden heart rate changes.
14. Increased meridional end-systolic stress.
15. B-cell lymphoma. (5)
16. General anesthesia. (6)
17. Complete AV block.
18. Guillain-Barre syndrome (7)
19. Maxillo-facial surgery. (8)
20. Subarachnoid hemorrhage. (9)
21. Therapy with sotalol to revert atrial fibrillation.
22. Tako-Tsubo syndrome. (11)
23. Apical left ventricular aneurysm and pseudoaneurysm. (11)

The above mentioned diagnoses were ruled out by means of the following tests:

- 1 and 2. Patient's clinical picture, chest X-ray and ventilation-perfusion scintigraphy excluded acute

and chronic PE with and without pulmonary hypertension.

3. The patient did not receive electroconvulsive therapy.
4. Intracranial hypertension was not observed in the MRI.
5. Absence of QT and QTc anomalies suggestive of sympathetic autonomic dysfunction.
6. Normal Doppler echocardiography and cardiovascular magnetic resonance imaging (CMRI) ruled out the diagnosis of apical hypertrophic cardiomyopathy.
7. Absence of history of alcohol intake.
- 8 and 9. Normal serum electrolytes,  $T_3$ ,  $T_4$  and TSH, serum and urinary calcium and phosphorus ruled out disorders of fluid and electrolyte balance and thyroid and parathyroid diseases.
10. The ischemic etiology was excluded due to normal perfusion images and CMRI with first-pass perfusion and late gadolinium enhancement.
11. There were no areas of lacunar infarctions in the brain MRI.
12. Electrotonic modulation of the T wave is based on the fact that T waves follow the direction of the vector of previous QRS complexes. Left bundle branch block, left or right ventricular pacing, RBBB, VPBs and VT and probable causes of T wave inversion. Left anterior hemiblock and VPBs with right bundle-branch block (RBBB)-like and left anterior hemiblock (LAH)-like QRS pattern were ruled out as caused of electrotonic modulation of the T wave with ECG and 24-hour Holter monitoring records. Right bundle branch block is also excluded as T wave vector does not follow the direction of QRS vector with this pattern.
13. Sudden heart rate changes were also ruled out due to low risk variability of heart rate in the 24-hour Holter monitoring.
14. Meridional end-systolic stress, assessed by Doppler echocardiography is derived from the formula:  $1.35 \times \text{left ventricular end-systolic dimension} \times \text{end-systolic pressure} / 4 \times \text{left ventricular end-systolic thickness} (1 + \text{left ventricular end-systolic thickness} / \text{left ventricular end-systolic dimension})$ . ESWS was not responsible for T wave inversion as normal values are  $40 - 120 \text{ g/cm}^2$ , and patient's value was  $70 \text{ g/cm}^2$ . The presence of inadequate left ventricular hypertrophy was excluded using the Ford's formula.
15. Absence of clinical picture or lab tests suggestive of B-cell lymphoma.
16. The patient did not undergo general anesthetics.
17. Absence of AV block in ECG and 24-hour Holter monitoring records.
18. Absence of clinical picture of Guillain-Barre syndrome.
19. The patient did not undergo cervical sympathectomy.

20. Absence of clinical picture or MRI of subarachnoid hemorrhage.

21. She was not under therapy with beta blockers or sotalol.

22. The presence of Tako-Tsubo syndrome with no apical and anterior dyskinesia but with persistent T wave changes cannot be excluded; however, although 30% of patients with this condition do not complain of chest pain, she had no history of physical or emotional stress. Normal biomarkers and persistent T wave inversion partially exclude this diagnosis.

## CONCLUSION

Although transient apical dyskinesia syndrome cannot be ruled out, this diagnosis is less likely due to the absence of symptoms and of history of physical or emotional stress.

The origin of T-wave inversion could not be established despite a profound assessment by clinical examination, lab tests, radiology, echocardiography, nuclear medicine imaging studies, and brain and cardiac magnetic resonance imaging.

## RESUMEN

Se define onda T negativa gigante o inversión masiva de la onda T a la aparición de ondas T negativas con una amplitud  $\geq 1 \text{ mV}$  en por lo menos dos derivaciones contiguas del ECG. Se presenta el caso de una paciente de 66 años con antecedentes de dislipidemia tipo IV e hipertensión arterial leve, a la que en buen estado de salud y totalmente asintomática en un examen de rutina se le detecta en el ECG de superficie un hemibloqueo anterior y ondas T negativas gigantes en las derivaciones I, II, aVL, aVF y de V1 a V6. La paciente fue internada en la UCI, donde se descartó que fueran de origen coronario. Durante el seguimiento, las ondas T se tornaron menos negativas hasta que al cabo de un tiempo (45 días aproximadamente) se normalizaron. A pesar de los estudios cardiológicos y no cardiológicos realizados, no se pudo establecer su origen.

**Palabras clave >** Ondas T negativas gigantes - Hemibloqueo anterior izquierdo - Origen indeterminado

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