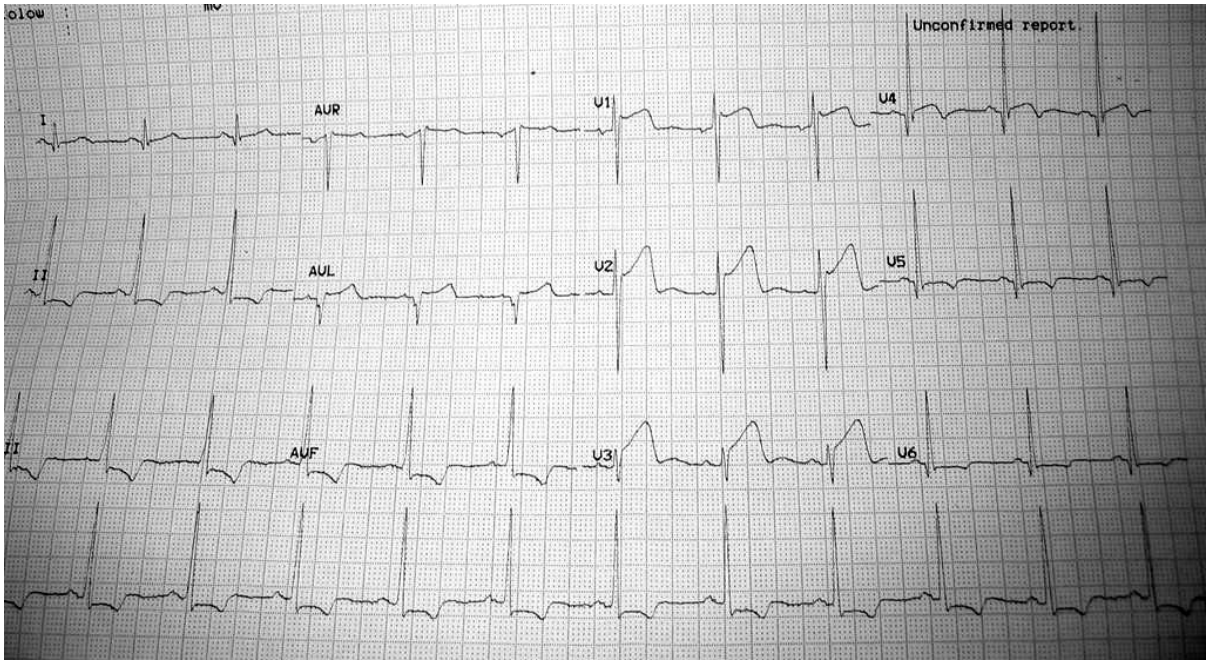
Dear all

My opinion ECG Diagnosis



1) Atypical Early Repolarization Variant (ERV). Why atypical? Because Early Repolarization Variant (ERV) the phenomenon is characterized by prominent J wave and ST-segment elevation upwardly concave predominantly ***in left to middle precordial leads***. It is considered present when at least two adjacent or anatomically contiguous leads on electrocardiogram show elevation of the ST segment, with values ≥2 mm in the precordial leads and/or ≥1mm in the limb leads. In this case, the ST segment elevation is located on right precordial leads (V1-V2). Brugada like type 2 ECG pattern. This pattern pseudo Brugada is observed in 8% of cases of ERV. "Clean" initial q wave from V4 to V6. Pseudo diastolic LV overloading.
2) Prominent U wave in V3 and V2 (U > P wave). The tallest positive U wave is usually observed in the area of leads V_2 to V_4.
3) Pseudo Left Ventricular Enlargement (LVE) pattern: Strain pattern of repolarization in left and inferior leads: ST segment depression followed by asymmetrical negative T waves. SAT - 65 degree in frontal plane and to front and rightward on HP. (rarely normal). Additionally QRS axis in frontal plane is near +80 degree (QRS complex in VL negative). This feature is against LVE. In LVE, SÂQRS may be deviated to the left as a consequence of levorotation of the heart in its longitudinal axis. Only in young people and children, LVE usually presents non-deviated SÂQRS. Additionally, there are not voltage or amplitude criteria of LVE: Sokolow Lyon index negative < 35mm(S of V1 + R of V5≥ 35 mm or 3.5 mV);

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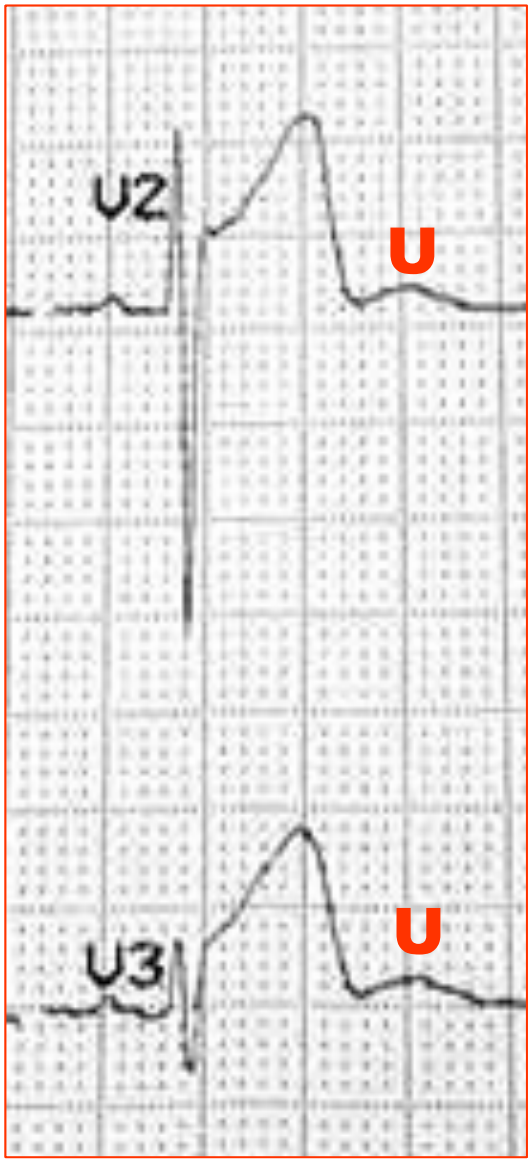


CAUSES OF PROMINENT U WAVES

1) **Bradycardia:** U wave voltage and U wave Constance is strongly rate dependent (inversely proportional). U wave is observed better during bradycardia. When HR is ≤ 65 bpm U waves are visible in 90% of cases. When HR is between 80 bpm & 95 bpm U waves are visible in 65% of cases. When HR is >95 bpm U waves are visible in 25% of cases. Figure. Bimodal T waves with hump-like morphology represent different levels of interruption of the descending slope of the T wave, called T2 instead of U wave. Bimodal or notched T waves may be distinguished from the T-U interval: the second apex of bimodal T wave (T2) is at a distance from the first one (T1) < 150 ms;

the T1-U interval is > 150 ms. The second apex of bimodal T wave (T2) is at a distance < 150 ms from the first module (T1): The T1-U interval is always > 150 ms[1]. The U wave increases its voltage or appears during slow rates or after pauses[2], as in the cases of long QT interval. In this case we observe a bifid T wave and a U wave only with slow HR.

2) Early repolarization variant (ERV): because bradycardia U waves are frequent, in ERV they are best observed in the V3 lead. U waves are frequent when sinus bradycardia is present.

3) Hypopotassemia or hypokalemia: Hypokalemia is associated with flattening of the T wave and the appearance of a prominent U wave. A pathological "U" wave as seen with hypokalemia is the consequence of electrical interaction among ventricular myocardial layers at AP phase 3 of which repolarization slows[3]. The main ECG features of hypokalemia are:

a) PR interval prolongation

b) Gradual ST segment depression ≥ 0.5 mm in II or from V1 to V3

c) Decrease of T wave amplitude (flat T wave)

d) Possible T wave inversion

e) Prominent U wave (U wave > 0.5 mm in II or > 1 mm in V3) secondary to prolongation of the recovery phase of the cardiac AP

f) Characteristic reversal in the relative voltage of the T and U waves

g) QTc interval prolongation

h) Tendency to TdP atypical tachycardia

i) Digitalis action enhancement. Hypokalemia potentiates the tachyarrhtymias produces by digitalis toxicity.

4) **Hypomagnesemia:** Prominent and alternant U wave with ventricular irritability was described in patiens qht low magnesium serum[4] (<1.8 mmol/L)

5) Hypocalcemia

6) Hypothermia

7) Class III antiarrhtymic drugs amiodarone, dofetilide, sotalol, Figure

8) Class IA quinidine, disopyramide and procainamide: Quinidine effect is characterized by discrete QRS prolongation only with an extreme quinidine effect, significative QT/QTc interval prolongation, depressed, widened, notched, and inverted T waves, prominent U waves and TdP tendency Figure

9) Digitalis effect or digitalis action: The earliest modification of digitalis effect on ECG or “digitalis action” are: PR interval prolongation, ST segment: shortening and superior convexity (“in spoon”) by shortening of phases 2 and 3 of AP, shortening QT and QTc intervals(main cause of acquired short QT interval), flattening with apiculate T wave of terminal portion in 10% of cases, possible symmetrical inversion of T wave (pseudo-ischemic T wave) and prominent U wave.

10) Phenothiazines The electrophysiological properties of phenothiazines are comparable to those of the Class Ia antiarrhythmic quinidine. Numerous ECG aberrations may be induced by these agents, including changes in the morphology of the T wave, prolongation of the QT interval, and accentuation of the U wave. Even with standard clinical dosages (100-400 mg/day), thioridazine causes minimal prolongation of the QT interval, reduction of T wave amplitude, and prominent U waves in nearly 50 percent of patients.

11) Forced inspiration

12) Post-exercise

13) Mitral valve prolapse

14) LVE

15) Alterations of the central nervous system that course with endocranial hypertension: In patients with cerebrovascular accident T and U waves are augmented consequently the T/U ratio is not altered. Any effect of retina

16) Cardiomyopathies: e.i. Hypertrophic cardiomyopathy with solitary hypertrophy of papillary muscle[5]

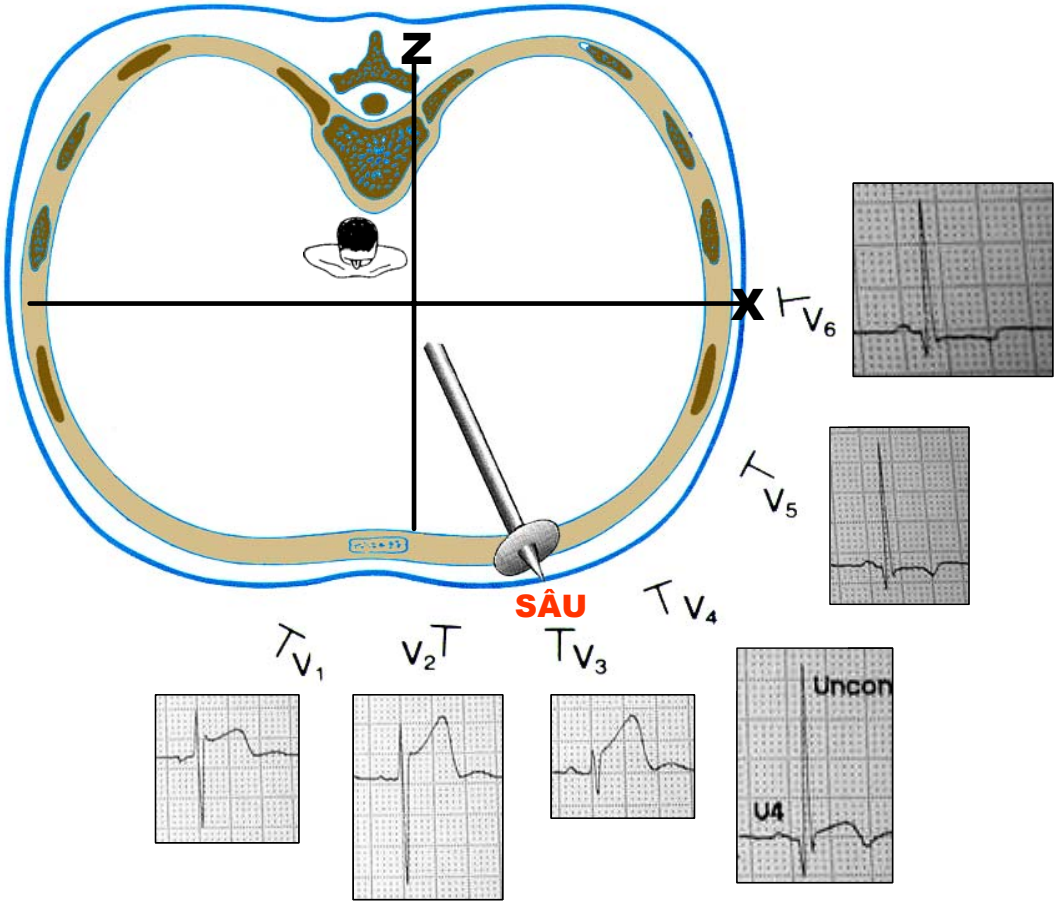
17) Acquired Complete AV block: Torsades de Pointes during acquired complete atrioventricular block is rare. The predictors of ventricular arrhythmias during acquired complete atrioventricular block are presence of prolonged QTc/JTc intervals, pathologic U wave and T-U complex, prolonged Tpeak-Tend interval, and LQT2-like QT morphology[6]

18) Congenital long QT syndrome: augmentation and greater degree of merging of the T and U waves and QTc interval prolongation are changes alert about the possibility of congenital long QT syndrome, specifically genotype 2 or 1[7;]. T or U wave alternant in association with long QTU and TdP is uncommon and its mechanism(s) is unknown. 1) TU alternans may be due to 2:1 propagation of an EAD or to alternation of the recovery kinetics of a repolarization current; (2) The constant occurrence of EAD in relation to phase 0 in spite of alternation of plateau duration suggests an ionic mechanism synchronized to depolarization; (3) Tachycardia dependent TdP in clinical and experimental examples of long QTU seems to be characteristically associated with TU alternant. Dispersion of repolarization may underlie the increased ventricular electrical instability in these cases[8]. Andersen-Tawil syndrome (ATS) is a channelopathy affecting inward rectifier potassium I(K1) with QT prolongation, large U waves, and frequent ventricular tachycardia (VT) [9].

19) Left circumflex-related myocardial infarction ECG criteria of strictly posterior myocardial infarction with the left circumflex coronary artery as an infarct-related coronary artery apply at less than 6 hours or at 24 hours from the onset of the symptoms. 1) ST depression ≥ 0.1 mV in two consecutive chest leads; 2) prominent positive U wave ≥ 0.1 mV in leads V2 or V3; 3) T/U ratio in leads V2 or V3 ≤ 4. Considering two of the

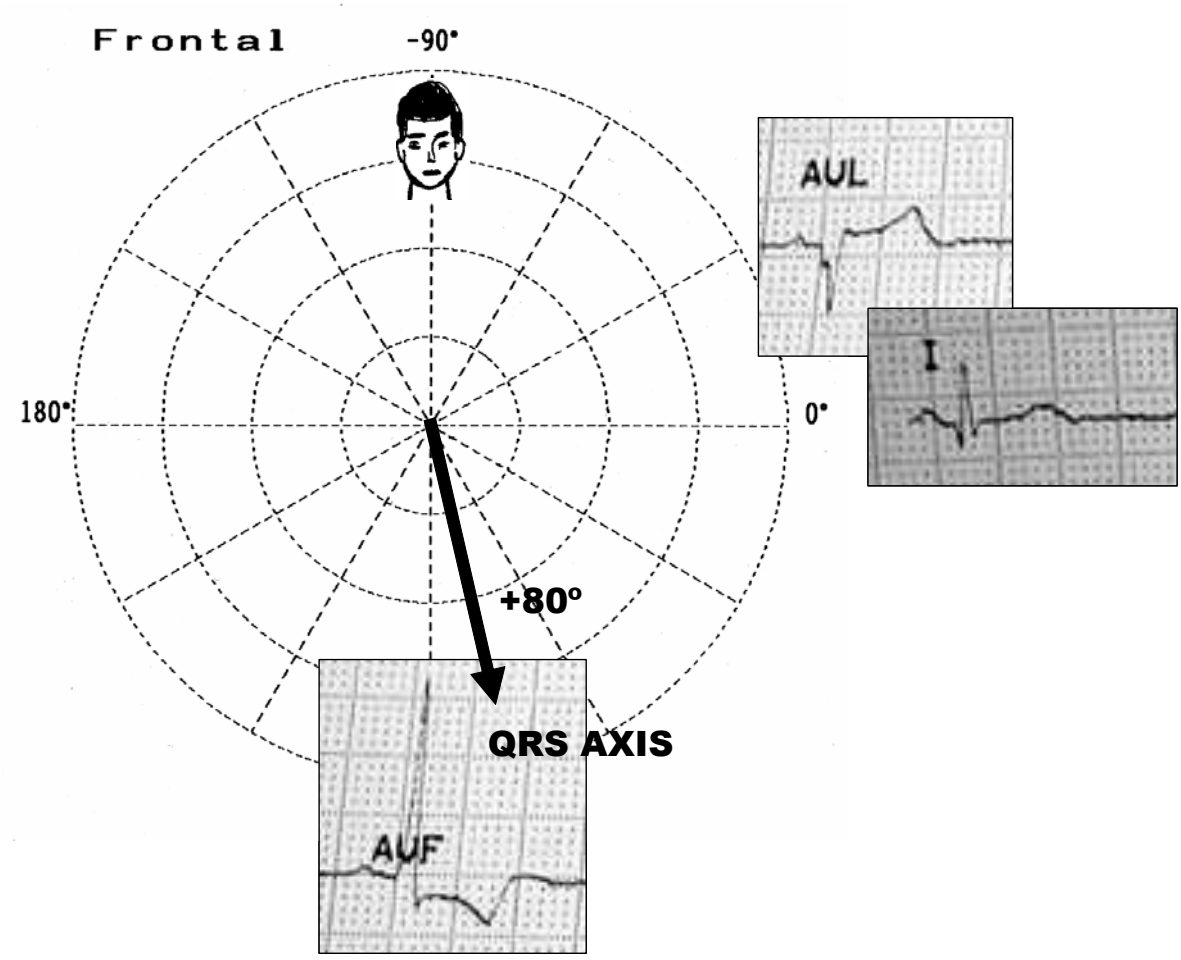
above criteria as positive, the sensitivity was 71.9%, the specificity 97.0%, and the diagnostic accuracy 88.8%[10].

NEGATIVE SOKOLOW-LYON INDEX (1949)1



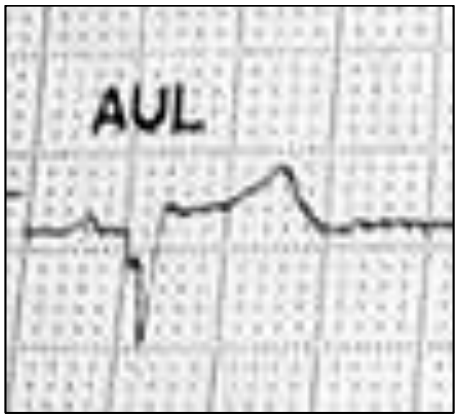
S wave of V1 + R of V5 ≥ 35 mm or 3.5 mV in adults older than 30, > 40 mm or 4.0 mV between 20 and 30 years (Sokolow-Rapaport), > 60 mm between 16 and 20 years and > than 65 mm between 11 and 16 years. Sensitivity: 25%. Specificity: 95%.

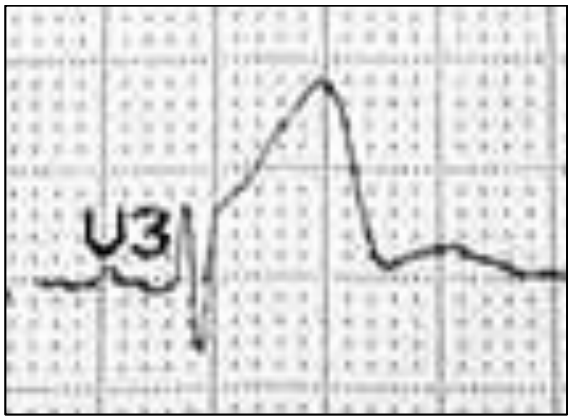
In this case, SV1= 11mm and RV5= 21mm. Sokolow-Lyon index= 32mm = NEGATIVE VOLTAGE CRITERIA for LVE.



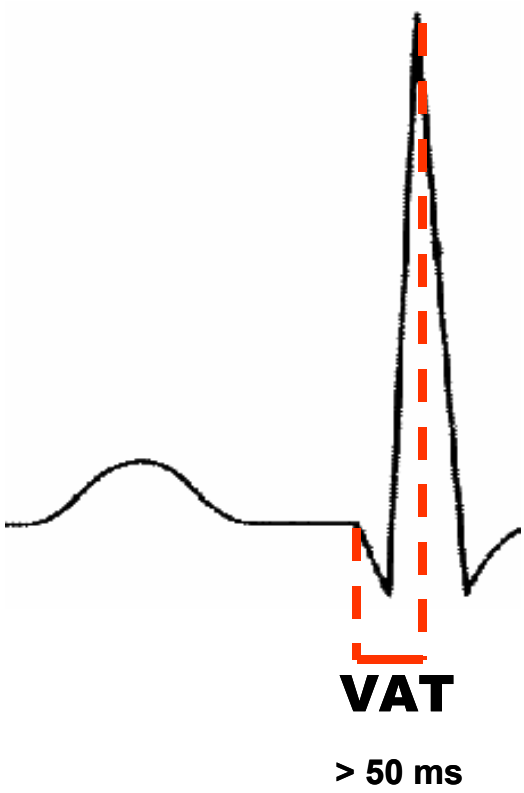
The QRS axis in frontal plane is near +80 degree (QRS complex in VL negative). This feature is against LVE.

Cornell index negative (CI = R of aVL + S of V3 > than 28 mm in men),

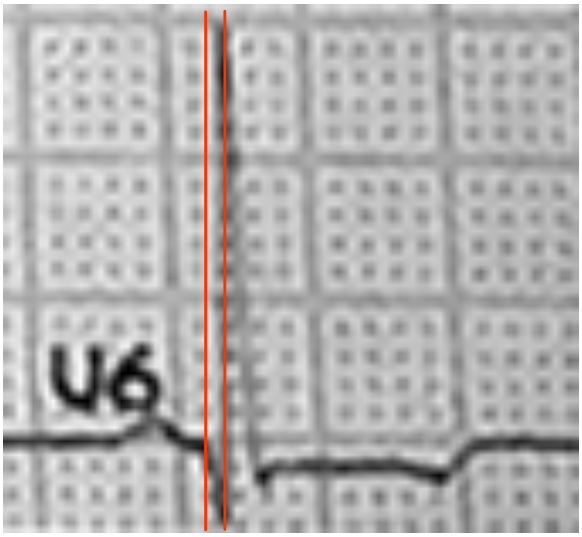




The Ventricular Activation Time or “R peak time” is normal in this case. This criteria based on the discrete increase in QRS complex duration at the expense of a delay in the time of appearance of R wave apex: “R peak time” in the leads that are opposite to the LV, initial time of intrinsic deflection or ventricular activation time (VAT);



In our case Ventricular Activation Time is: <40ms.



Finally, ROMHILT SCORE SYSTEM FOR LVE has only 3 point. (5 or more points: certain LVE; 4 points: probable LVE.)

The authors attribute values from 1 to 3 points to the different existing criteria, 5 or more points: certain LVE; 4 points: probable LVE.

ECG finding	**Scoring**	**Our Case**
Voltage criteria R or S wave in limb leads ≥20 mm S wave in V1 or V2 ≥30 mm R wave in V5 or V6 ≥30 mm	3 points	0 point
ST-T abnormalities Without digitalis With digitalis	3 points 1 point	3 points
Left atrial abnormality Negative area under P wave in Lead V1 ≥ 1 mm2 (1 box)	3 points	0 point
Left axis deviation	2 points	0 point
QRS duration > 90 ms	1 point	0 point
Intrinsicoid deflection V5, V6 ≥50ms	1point	0 point

Total= 3 points. Has not criteria.

Finally, PERUGIA SCORE SYSTEM is negative.

- The Perugia score (Verdecchia P, et al. Prognostic value of a new electrocardiographic method for diagnosis of left ventricular hypertrophy in essential hypertension. J Am Coll Cardiol. 1998; 31:383-390)carried the highest population-attributable risk for cardiovascular morbidity and mortality compared with classic methods for detection of LVH. Traditional interpretation of standard electrocardiography maintains an important role for cardiovascular risk stratification in essential hypertension. ECG-LVH. Perugia Score Positivity of one or more of the following criteria:
  - SV3+ RaVL >2.4mV (men) or >2.0mV (women);
  - Left ventricular strain pattern;
  - Romhil-Estes score of five or more points.

- The diagnostic criteria of the Perugia Score, the newest scoring system for electrocardiogram-diagnosed LVH. However, the Perugia score has a low sensitivity. They showed that the prevalence of LVH in the hypertensive population is highest using the Perugia score, followed by the Sokolow-Lyon Voltage criteria.

CONCLUSION

This patient has not Left Ventricular Enlargement ECG criteria.

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All the best
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