UNUSUAL TACHYARRHYTMIA
Bidirectional Ventricular Tachycardia: BVT

Case of Dr. Raimundo Barbosa de Barros Fortaleza - Ceará Brazil
Commentaries Andrés Ricardo Pérez Riera MD Sao Paulo Brazil
Prezado Dr. Andrés

Paciente do sexo feminino, 47 anos, portadora de cardiomiopatia dilatada, em uso de digoxina, carvedilol, captopril e furosemida. Deu entrada na sala de emergência com queixas de palpitações, náuseas, vômitos e piora do quadro dispnéico. Qual o diagnóstico e a terapia adequada? Um abraço

Raimundo Barbosa Barros

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Appreciated Dr. Andrés

Feminine 47 years old, carrier idiopathic dilated cardiomyopathy. In regular use of digoxin, carvedilol and furosemide in therapeutic dose. Gave entry in our emergency room with complain of palpitations, nauseas vomiting and worsening dispneic picture. Which the diagnosis and the appropriate therapy? A hug

Raimundo Barbosa Barros
Note the alternating QRS axes on frontal plane from right axis deviation followed by left axis deviation (alternating aberrant conduction by left anterior fascicle and left posterior fascicle). Additionally, irregular alternating right and left bundle branch block-type on precordial leads is observed. This pattern could occur in the setting of a supratherapeutic serum level of digoxin as a consequence of acute renal failure.

Conclusion: Type II bidirectional tachycardia: BVT Type II the intervals among the QRS complexes are shorts or longs. The intervals among equals QRS complexes are constants, and the intervals among different morphological QRS complexes are not equals.

1911: Levy and Lewis\(^1\) suggested from experiments in cats that two independently firing ventricular pacemakers were operating.

1922: First description\(^2\) Schwensen concluded that this rhythm was ventricular in origin.

1923: Felderbaum\(^3\) proposed that the focus might be in the atrioventricular node or main His.

1925: Luten D\(^4\) and Sir Thomas Lewis\(^5\) are favorable to single ventricular focus origin conducted with alternant aberration by bundle branch block.

1926: First Gallavardin\(^6\) suggest a single ventricular focus origin and a double circle movement with eight morphology to explain the alternating QRS complexes

1928: Palmer and White\(^7\)

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2. Schwensen E: Ventricular tachycardia as a result of the administration of digitalis Heart 1922; 9: 199.
1939: Sherf and Kisch\(^1\) postulated a classification in tree types: I, II and III.

- **Type I** – rare – has equal intervals among the QRS complexes.
- **Type II** the intervals among the QRS complexes are shorts or longs. The intervals among equals QRS complexes are constants, and the intervals among different morphological QRS complexes are not equals.
- **Type III:** The morphology of QRS complexes are different and the intervals are irregularly variables.

1947: Zimdahl and Kramer\(^2\) postulated the origin in two focuses: supraventricular and ventricular because at least one QRS pattern complex could be abolished with the carotid massage.

1961: Agustin Castellanos Jr\(^3\), and later Sherf and Bonemann\(^4\) (1967) also suggest the single ventricular origin conducted with alternant aberration by bundle branch block.

1969: Mauricio Rosebaugh Marcelo Elizari and Lazzari\(^5\) studied 13 patients and concluded that bidirectional tachycardia is supraventricular with a functional triphasicular block: right bundle branch and alternant aberrant conduction in the two division of the left bundle branch. Additionally, these authors observed that the was perfectly rhythm regular and vagal stimulus responsive.

1973 (April): Michael Rosen\textsuperscript{1} demonstrated experimentally in dogs with microelectrodes techniques the effects of ouabain in toxic concentration on phase 4 of canine Purkinje fibers transmembrane potentials: Triggered Activity in phase 4 (delayed after depolarizations or DAD)\textsuperscript{1}.

1973 (June) Stafford I Cohen et al\textsuperscript{2}. demonstrated in human heart using ECG and His bundle potential the left ventricular origin and enhanced phase 4 despolarization on the divisions of the left bundle branch.

1975: First description associated with Catecholaminergic Polymorphic Ventricular Tachycardia (CPVT) and isoproterenol infusion\textsuperscript{3}.

1976: first effective publication related purified digoxin-specific Fab fragments are capable of rapid reversal of advanced digoxin toxicity\textsuperscript{4}.

1992 (May): first description of BVT induced by herbal aconite poisoning\textsuperscript{5}.

\textsuperscript{3} Reid DS, Tynan M, Braidwood L, Fitzgerald GR. Bidirectional tachycardia in a child: a study using His bundle electrography. \textit{Br Heart J.} 1975;37:339-344
2004: first description of BVT associated with Andersen -Tawil – Syndrome\(^1\).

2006: first description of BVT associated with fatty infiltration of the anterior right ventricular free wall\(^2\).

2007: first description of BVT treated successfully with flecainide in Andersen-Tawil-Syndrome\(^3\).

2009: first description of BVT associated with acute myocardial infarction\(^4\).

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BVT POSSIBLES CAUSES

I) Acquired
1. Digitalis toxicity consequence of supra-therapeutic serum level of digoxin or digitalis poisoning
2. Digoxin-cyclosporine interaction in transplantation heart
3. Hypokalemia
4. Herbal aconitine poisoning
5. Isuprel infusion
6. Arrhytmogenic Right Ventricular Dysplasia (ARVD)
7. Hypertrophic Cardiomyopathy (HCM)
8. Dilated Cardiomyopathy (DCM)
9. Congenital heart disease
10. Myocarditis
11. Myocardial infarction/myocardial ischemia
12. Fatty infiltration of right ventricular free wall

II) Congenital familial-hereditary with structurally normal heart or channelopathies
1. Andersen-Tawil Syndrome hyperkalemic periodic paralysis
2. Catecholaminergic Polymorphic Ventricular Tachycardia (CPVT)
   • Autosomal dominant RYR2
   • Autosomal recessive CASQ2 gene mutation
FACTORS THAT PREDISPOSE DIGITALIS INTOXICATION AND INCREASE SENSITIVITY TO DIGITALIS

Outline of main factors that predispose digital intoxication occurrence.

- Hypokalemia < K⁺: diuretics, vomiting, acute gastroenterocolitis, hemodialysis, corticotherapy
- Hypomagnesemia: Mg²⁺
- Hypercalcemia > Ca²⁺
- Amyloidosis
- Advanced age
- Pulmonary diseases: Low saturation of O₂
- Drugs that reduce clearance of Digoxin: quinidine, verapamil, diltiazem, amiodarone, propafenone, espinolactone, erythromycin, triamterene
- Severity and degree of substitution of contractile myocardial cell: degree of fibrosis and/or necrosis
- Hypothyroidism
- Sympathetic hypertone
- Digoxin intoxication
MANIFESTATIONS THAT SUGGEST DIGITALIS INTOXICATION

- **MAIN CARDIAC ARRHYTHMIAS**

- Sinus bradycardia.
- Sinus arrest.
- Blocks: of SA outflow, Ex.: Wenckebach.
- AV block of all degrees with predominance of first degree (conduction slowing and prolongation of refractory period in AV node), of outflow.
- AV dissociation: dominant suppression of pacemaker with passive escape of a low junctional focus or inappropriate acceleration of subsidiary pacemaker or more rarely, dissociation within the AV node proper.
- Atrial tachycardia with variable AV conduction: the most common one and almost pathognomonic.
MANIFESTATIONS THAT SUGGEST DIGITALIS INTOXICATION

- **MAIN CARDIAC ARRHYTHMIAS**
  - Sudden appearance of atrial tachycardia during digitalis therapy in patient with AF.
  - Accelerated junctional rhythm or nonparoxysmal junctional tachycardia with frequent isorhythmic dissociation.
  - Isolated or bigeminal extrasystoles.
  - Fascicular VT.
  - AIVR or accelerated idioventricular rhythm.
  - **Bidirectional (BVT) or bifascicular VT.**
  - Ventricular flutter.
  - Slow VF.
MANIFESTATIONS THAT SUGGEST DIGITALIS INTOXICATION

➤ EXTRACARDIAC

• GASTROENTERIC: anorexia, nausea, vomiting, diarrhea and abdominal pain.

• NEUROPSYCHIATRIC: headache, confusion, fatigue, dizziness, restlessness, delirium, lability of humor, sleepiness, psychosis, mental confusion, memory lapses, pseudo dementia.

• VISUAL: dyschromatopsia more common for red, green, and yellow.

Extracardiac manifestations of digitalis intoxication.
Delayed Afterdepolarizations (DADs) trigged activity: they are oscillations of the membrane potential that occur after having completed phase 3 of AP or in phase 4. When they reach the limit, trigger a new AP. They are observed in high rates (tachycardia-dependent). Their mechanism is caused by the opening of the INS. channel, sensitive to increased intracellular Ca\(^{2+}\) concentration. It is a stimulus for repetitive release of even more Ca\(^{2+}\) form sarcoplasmic reticulum. The causes of DADs include digitalis, catecholamine, hypercalcemia and rapid pacing. Arrhythmias caused by DADs are PVCs, tachycardia related to digitalis excess, some catecholamine-dependent atrial and ventricular tachycardias.
ECG CHARACTERIZATION OF BVT

1. Rapid rhythm in which QRS morphology alternated so that the direction of the complexes is predominantly upward in one beat and downward in the next
2. Heart rate: between 140 to 180bpm
3. QRS duration: may be equal to, less than or greater than 120ms
4. Right Bundle Branch Block pattern is seen in V₁ lead.
5. In frontal plane is observed a QRS complex axis between -60° to -80° and + 120°.

ECG CHARACTERISTIC OF BVT SECONDARY TO DIGITALIS INTOXICATION

1) Heart rate between 140 and 180bpm
2) QRS duration <140ms
3) RBBB pattern
4) QRS axis in frontal plane from right (+ 120°) to left (-60°) alternatively

EPS: The absence of His bundle activity preceding the earliest onset of ventricular activation strongly favored a ventricular origin.
The earliest modification of digitalis effect on ECG or “digitalis action” are:

1) Prolonged PR interval.
2) ST segment: shortening and superior convexity (“in spoon”) by shortening of phases 2 and 3 of action potential (AP);
3) QT and QTc intervals shortening: main cause of acquired short QT;
4) T wave flattening with apiculate form of terminal portion in 10% of cases. Possible symmetrical inversion of T wave (pseudo-ischemic T wave);
5) Prominent U wave.
VARIABLE HIGH-DEGREE AV BLOCK WITH 7:1 AND 5:1 CONDUCTION RATE BY ACUTE INTOXICATION BY DIGOXIN IN SUICIDE ATTEMPT

Example of high degree AV block, variable with conduction rate 7:1 and 5:1 by acute intoxication by digoxin in a patient who ingested 1 box of digoxin as a suicidal attempt.
ATRIAL TACHYCARDIA
WITH 2:1 BLOCK

P WAVES SEPARATED BY ISOELECTRIC LINE

P-P INTERVAL
214 bpm

P-P interval rate: usually between 100 and 180 bpm.
1) Automatic form: 150 bpm to 250 bpm
2) By microreentry: between 130 and 150 bpm.

Example of atrial tachycardia with 2:1 block, considered typical arrhythmia of digitalis intoxication.
AF AND BIGEMINAL VENTRICULAR EXTRASYSTOLES

MONOFOCAL BIGEMINAL VENTRICULAR EXTRASYSTOLES

SMALL AND RELATIVELY NORMAL CONDUCTED BEATS

AF with slow and irregular ventricular response rate. Advanced 2nd degree AV block: typical of digitalis intoxication.

Example of atrial tachycardia with 2:1 block, considered atypical arrhythmia of digitalis intoxication.
SA block by digitalis intoxication with bigeminal rhythm.
Toxicity from the digitalis family of cardiac glycoside medications remains common. Successful treatment depends on early recognition; however, the diagnosis of potentially life-threatening toxicity remains difficult because the clinical presentation is often nonspecific and subtle. The hallmark of cardiac toxicity is increased automaticity coupled with concomitant conduction delay. Though no single dysrhythmia is always present, certain aberrations such as frequent premature ventricular beats, bradydysrhythmias, paroxysmal atrial tachycardia with block, junctional tachycardia, and bidirectional ventricular tachycardia are common.

Treatment depends on the clinical condition rather than serum drug level. Management varies from temporary withdrawal of the medication to administration of digoxin-specific-antibody Fab fragments (Fab) purified from immunoglobulin G produced in sheep for life-threatening cardiovascular compromise. The dose of Fab fragments is equal to the amount of digoxin or digitoxin in the patient's body as estimated from medical histories or determinations of serum digoxin or digitoxin concentrations. First-line therapy with Fab fragments in patients with digitalis poisoning was associated with a low mortality rate. The most common side effect is rapid development of hypokalemia and exacerbation of congestive heart failure. Purified digoxin-specific Fab fragments are capable of rapid reversal of advanced digoxin toxicity. MgSO4 is an useful therapeutic tool for the treatment of various ventricular tachycardias. Given at low rate (50 mg/min) and continued 2 hours after disappearance of arrhythmia, and infusion repetition at the same rate for 60-90 min twice daily for the next 3-4 days. Lignocaine can be useful in the treatment of digitalis-induced bidirectional tachycardia. The drug is superior to other class I or class IV agents.

The rootstocks of aconitum plants have been commonly employed in traditional Chinese herbal recipes for "cardiotonic" actions and for relieving "rheumatism". Bidirectional tachycardia can be induced by aconites present in a Chinese herbal decoction consume. The tachycardia show marked susceptibility to vagotonic maneuvers, cholinesterase inhibition, and adenosine triphosphate. The incessant nature of the tachycardia, rapid recurrence after transient suppression, and failure to respond to direct current cardioversion suggested an automatic tachycardia mechanism consistent with known data on the cellular electrophysiological mechanism of aconitine-mediated arrhythmogenesis.

A fascicular or ventricular myocardial origin of the tachycardia with alternating activation patterns, or dual foci with alternate discharge, appeared most plausible. Multiple pitfalls could occur during the processing of these herbs that might have predisposed to aconite poisoning. The need for strict control and surveillance of herbal substances with low margins of safety is highlighted.

According to the fact that aconitine acts as a agonist of Na-channel receptor, antiarrhythmic agents which belong to Vaughan-Williams' classification I might be the first choice for the therapy of aconitine induced arrhythmias.

BVT of CPVT

1. Children or young people
2. Familial history of syncope or sudden death in 30%
3. Mechanisms of arrhythmias associated to impaired Ca\(^{2+}\) regulation homeostasis\(^1\)
4. Unexplained syncope. Unmasking false epilepsy
5. Absence of structural heart disease
6. Autosomal dominant mutations in the cardiac ryanodine receptor (RyR2) or recessive Mutation on calsequestrin 2 (CASQ2) have been reported\(^2\).
7. Normal QT interval\(^3\).
8. Unexplained syncopal episodes, or sudden death\(^4\) even when the resting ECG is normal.
9. Stress- or emotion-induced syncope related to PVT or BVT.
10. Induced by any form of increasing adrenergic stimulation (adrenergic-dependence) could be induced by increasing the heart rate by atrial pacing or isoprenaline administration.
11. His bundle electrography show that the arrhythmia is ventricular in origin.
12. Therapy: appropriate ß-blocker associated with ICD

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Femenine, white 20 years old. Repetitive stress- or emotion-induced syncope's episodes. Autosomal dominant mutations in the cardiac ryanodine receptor (RyR2). ECG QRS axis from $-60^\circ$ to $+120^\circ$.beat to beat. QRS pattern with RBBB and LBBB patterns in alternating fashion.
BVT of Andersen-Tawil-Syndrome (ATS)

1. It is a potassium channelopathy with autosomal-dominant inheritance
2. Mutation of KCNJ2 gene which encodes the alpha-subunit of the strong inward-rectifier Ba^{2+}-sensitive potassium channel I_{k1} coding for Kir 2.1 protein with important roles in maintaining membrane potential and during the terminal phase of cardiac action potential repolarization (phase 3 and 4).
3. Includes a clinical triad: periodic paralysis (episodic muscle weakness), cardiac arrhythmia with syncope or sudden cardiac death\(^1\) and usually mild but diagnostically useful dysmorphic facial/skeletal features.
4. Cardiac manifestations are variable and may include a long QT syndrome (LQT7), PVCs, complex ventricular ectopy, and polymorphic or BVT.
5. Potential neuropsychiatric system involvement.
6. Treatment with a combination of spironolactone, amiloride, and potassium supplements\(^2\). The class IC antiarrhythmia drug flecainide has been shown to depress ventricular ectopy and improve exercise capacity in patients with ATS\(^3;4\). Imipramine therapy had a positive effect on arrhythmia\(^5\).

Colleagues Commentaries
This could be a case of bidirectional VT typical arrhythmia of CMPVT and can be associated with digitalis intoxication maybe during hypokalemia during furosemide therapy. Last time we described a fulminated myocarditis in Kardiol. Pol. in female with EF % of about 15% during digoxin therapy (Fulminant myocarditis complicated with cardiogenic shock and various tachyarrhythmias)


Best wishes from Poland
Piotr Kukla, MD, PhD

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Dear Prof. Riera and All

This is a case of bidirectional ventricular tachycardia mostly due to Digoxin toxicity.

Immediate therapy with LIDOCAINE [others prefer PHENYTOIN] and evaluation of K level. FAB, if available, is the drug of choice for malignant arrhytmias induced by digoxina toxicity.

Digoxin toxicity is precipitated by hypokalemia and the toxicity by itself can cause hyperkalemia.

Raed Abu Sham'a, M.D
Andres, I will allow you to circulate this to the group with your usual elegant additional explanations. With the patient on digoxin and having a significant underlying cardiomyopathy, this is a bidirectional ventricular tachycardia consistent with digitalis intoxication. At least, this is my first diagnosis. There is an excellent example published recently in the Journal of the American College of Cardiology (Images in Cardiology) by Drs. Richter and Brugada, JACC 2009; 54: issue 13. I would request a serum digoxin level and also consider starting the patient on Digibind, if available, even before the serum digoxin level returns. Towards the end of the anterior precordial lead recordings, there are two other morphology PVCs as a couplet. With the multifocal PVCs in this setting, I believe that this is digitalis toxicity until proven otherwise and the patient is at a particularly high risk of degenerating to ventricular fibrillation. If this was a young patient without identified structural heart disease, particularly if associated with exercise, consider CPVT. There are also reported cases in the absence of digitalis associated with a severe underlying cardiomyopathy. This can also be a supraventricular tachycardia with alternating rate-dependent bundle branch block. I have attached some brief power point presentations that can be shared with the group. ECG Case Study 0029 shows non-conducted APBs and both RBBB and LBBB functional (Ashman’s) aberrant conduction. Case 0028 is various manifestations of Ashman’s Aberration and Case 0080 is classic Tachycardia (Phase 3) dependent BBB. If there was a baseline 12 lead ECG and one of the repetitive complexes was consistent with the patient’s intrinsic sinus or supraventricular conducted QRS complex, then this could be ventricular bigeminism with a PVC alternating with a conducted QRS. It would help if I could identify P waves but with the very brief rhythm strips associated with each lead of the 12 lead tracing and the alternating morphology of the QRS complex, I am unable to identify a distinctive P wave or repetitive distortion in the ST-T wave to identify AV dissociation or even confirm that one of the QRS complexes is sinus and the other is a PVC.
Another cause of “electrical alternans” is a pericardial effusion with tamponade but this is usually seen best in one or two leads with minor variation in the other leads and the clinical history is likely to be different.

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Andres,
Eu solicitaria uma dosagem sérica de digoxina e também consideraria começar com Digibind, se disponível, mesmo antes de ter o resultado da digoxinemia. Nas derivações precordiais existem duas morfologias. Com a presença de extrasístoles polifocais o paciente encontrase-se em um risco alto de degenerar para fibrilação ventricular.
Se fosse um paciente jovem sem doença estrutural, particularmente se associado com exercício, consideraria o diagnóstico de taquicardia ventricular polimórfica catecolaminérgica. Existem casos reportado na ausência de digital associada a severa cardiomiopatia subjacente.
Este evento poderia ser uma taquicardia supraventricular com bloqueio de rama alternante dependente da freqüência (Ashman). Eu ajuntei algumas apresentações que poderão ser compartilhadas com o grupo condução aberrante.
Outra causa de “alternância elétrica” é o derrame pericárdico com taponamento mas isto é normalmente visto melhor em uma ou dois derivações com mínima variação em outras derivações e a história clínica seria diferente.

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Andres:

The patient has severe digitalis toxicity compounded by low K+.

Rx:
Discontinue all medications, start an IV and give intravenous K+ immediately, but cautiously, (no more than 40 mEq/hour), initiate cardiac monitoring with continuous professional observation. Prayer.

EKG Dx:
Patient has a full constellation of Digitalis toxicity effects. Supraventricular tachycardia approaching 300/minute...very dangerous in this severely compromised patient. Bidirectional tachycardia, at least one source being ventricular and the other probably Junctional. The supraventricular origin shows Left Axis Deviation. Occasional PAC's and multifocal ventricular extrasystole's. AV node only minimally blocked, SA node probably overdrive suppressed by the SV tachycardia. Note: great ST elevation in extrasystole's of ventricular origin typical of Marriotts' ventricular extrasystole "snapshot effect"; the signs of ischemia most likely due to the high ventricular rate effect (i.e., relative ischemia due to pulse rate up to 300/minute) outstripping the metabolic demands on non-infarcted (somewhat normal) but severely stressed myocardium.

Dale Dubin M.D.
Dear Andres,
In principle it is a bidirectional TV, and using digoxine, the possible cause would be the digitalic poisoning. The question would be if the expanded cardiomyopathy is of well-known origin and the arrhythmia is originating of its same cardiopathy associated to the digitálica poisoning. If it were suspended to the digitalis and the arrhythmia it continued would be possible to be thought about the syndrome of Andersen-Tawil (QTL) that does not have expansion Generally I SAW. We had a case that the TVB generated a taquimiocardiopatía that reverted when flecainida dealing itself with. To this we published it case in JCE (Flecainide Suppresses Ventricular Bidirectional Tachycardia and Reverses Tachycardia-Induced Cardiomyopathy in Andersen-Tawil Syndrome Journal of Cardiovascular Electrophysiology. Volume 19, Issue 1, Date: January 2008, Pages: 95-97). This it is the small contribution that I can do. Greetings.

Estimado Andrés,
En principio es una TV bidireccional, e ingiriendo digoxina, la posible causa sería la intoxicación digitálica. La cuestión sería si la miocardiopatía dilatada es de origen conocido y la arritmia es proveniente de su misma cardiopatía asociada a la intoxicación digitálica. Si se suspendiera la digital y la arritmia continuara se podría pensar en el síndrome de Andersen-Tawil (QTL) que Generalmente no tiene dilatación VI. Nosotros tuvimos un caso que la TVB generó una taquimiocardiopatía que revirtió al tratarse con flecainida. Este caso lo publicamos en JCE (Flecainide Suppresses Bidirectional Ventricular Tachycardia and Reverses Tachycardia-Induced Cardiomyopathy in Andersen-Tawil Syndrome Journal of Cardiovascular Electrophysiology. Volume 19, Issue 1, Date: January 2008, Pages: 95-97).
Este es el pequeño aporte que puedo hacer. Saludos.
Oscar Pellizón MD.
Las veces que he visto este tipo de taquicardias es en libros, como ejemplo de arritmias provocadas por intoxicación digitálica (taquicardia bidireccional) lo cual condice con el cuadro de nauseas y vómitos, si tengo como medir digoxemia sería el primer estudio a hacer, evaluación iónica y función renal, suspensión de digoxina y si tengo disponible, anticuerpos antidigoxina. Si la arritmia le está generando fallo hemodinamico, se puede probar con lidocaina o con difenilhidantoina (fenitoina).

Ignacio Retamal
Córdoba

Need to see previous ECG. Is this atrial fibrillation?
Somewhat atypical bidirectional VT, usually caused by Digital.
Blood dig level?

Boris Surawicz

Necessário ver ECG prévio. Estava em fibrilação atrial?
Um tanto Taquicardia ventricular bidireccional, usualmente ocasionada por digital
Como estava o nível sérico?

Boris Surawicz.
Dear Andres.

In my two cents this is a beautiful demonstration of digitalis toxicity on ECG: bidirectional ventricular tachycardia alternating from regions nearby the anterior and posterior fascicles of left ventricle.

Many thanks to you and to Dr. Raimundo Barbosa Barros for sharing this very interesting ECG!

Li Zhang M.D.

Querido Andrés.

Na minha experiencia isto é uma bonita demonstração de toxicidade digitálica no ECG: A taquicardia ventricular bidirecional alternando a condução pelo fascículo anterior e posterior do ramo esquerdo.

Muito obrigado para você e para o Dr. Raimundo Barbosa Barros por compartilhar este muito interessante ECG!
Suspender la digital, administrar sales de potasio, dosar digoxinemia. Sólo si no cede el cuadro con estas medidas administraría amiodarona.

Saludos.
Luciano Pereira  Ciudad del Este República del Paraguay

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Suspender a digital, administrar sais de potássio, fazer a dosagem de digoxinemia. Apenas se não reverter o quadro com estas medidas administrar amiodarona. 

Saudações
Luciano Pereira Cidade del Este República Oriental do Paraguai.

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Suspend the digital, manage potassium salts, make the seric digoxin dosage. Just if do not revert the picture with these measured then to administer amiodarone.

Greetings
Luciano Pereira. del Este City Paraguay

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