TOTAL THYROIDECTOMIZED YOUNG WOMAM, WITHOUT REPLACEMENT THERAPHY

MULHER JOVEM COM TIREOIDECTOMIA TOTAL SEM TERAPIA SUBSTITUTIVA

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Estimado Prof. Andrés e amigos do foro.

Paciente do sexo feminino, 36 anos, tireoidectomia total há 8 anos por neoplasia invasiva. Deu entrada na Unidade Coronária com quadro de fraqueza muscular progressiva, adinamia e estado de torpor acompanhado de anasarca.

A família informa que há vários meses abandonou por conta prórpia a terapia de substituição hormonal (hormônio tiroidiano).

Posteriormente mandarei mais detalhes.

Analisando somente os antecedentes e o ECG qual é o diagnóstico clínico-elétrocardiográfico?

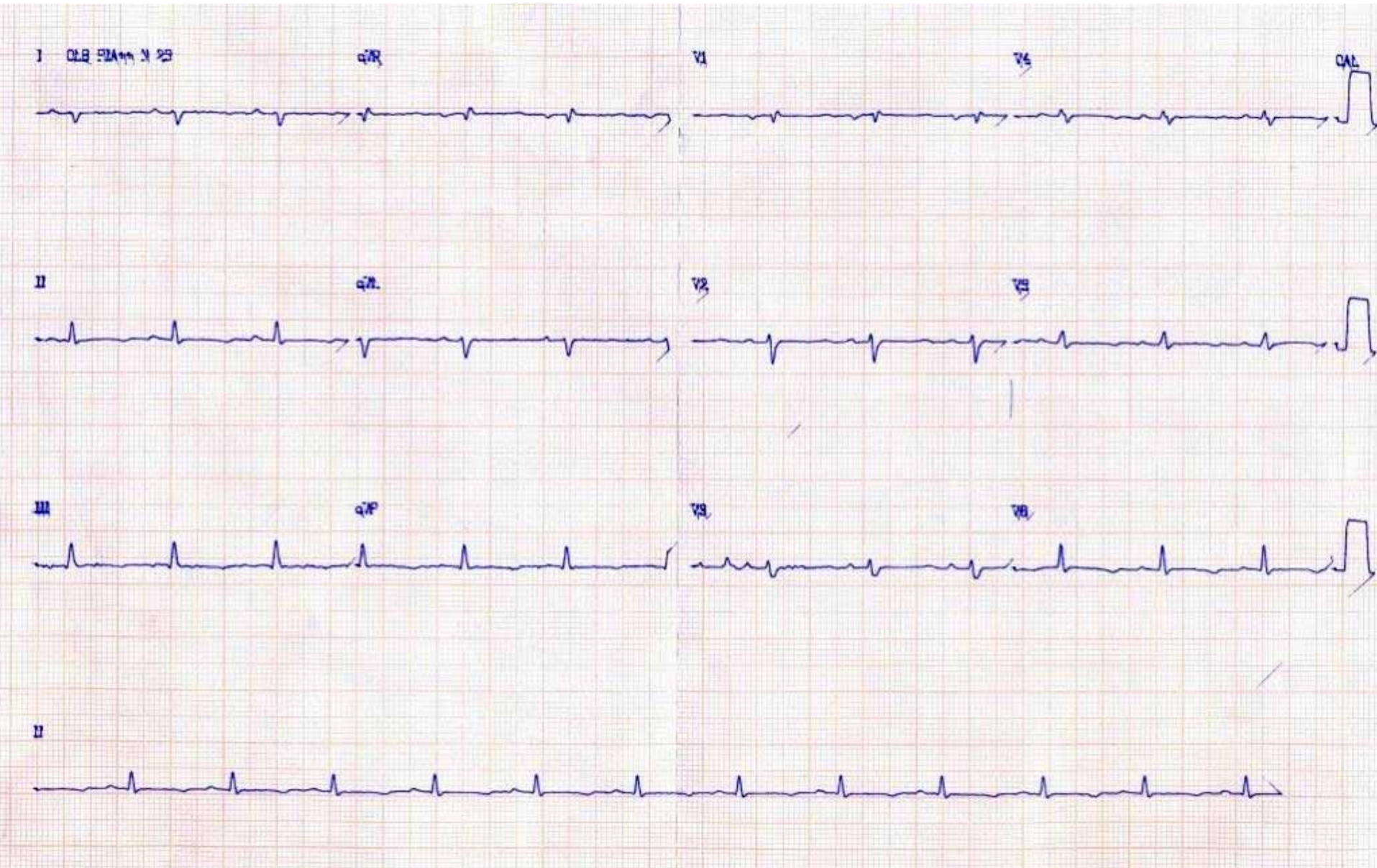
Dear Prof.. Andres and forum's friends:

Female, 36 yo, with previous antecedent of total thyroidectomy consequence of invasive cancer.

She was admitted to the our Coronary Care Unit with a progressive condition manifested by muscle weakness, malaise and torpor accompanied by anasarca.
The family reports that she stopped the substitutive thyroid hormone therapy several months ago.

Later I will send more details.

Question: analyzing only the ECG and the antecedents which is the clinical and electrocardiographic diagnosis?


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

COLLEAGUES OPINIONS

Ritmo sinusal, complejos QRS de bajo voltaje por infiltración miocárdica mixedematosa, intervalo QT muy prolongado, bloqueo fascicular posterior más isquemia subepicárdica inferolateral.
Como esta el perfil tiroideo?
Dr. Jairo Armando Rodriguez Fernandez: jairocordis@hotmail.com

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Sinus rhythm, low voltage of QRS complexes consequence of myxedema myocardial infiltration, very long QT interval, posterior fascicular block subepicardial inferolateral ischemia.
How is the thyroid profile?

Ritmo sinusal, QT prolongado, complejos QRS de bajo voltaje por derrame pericárdico secundario al hipotiroidismo. Habria que evaluar muy bien la hemodinamia (seguramente esta comprometida), reponer rapidamente el suplemento hormonal, y seguir muy de cerca la evolución,
muy bonito caso,
Saludos hotdog

Sinus rhythm, prolonged QT interval, low QRS complexes voltage secondary to hypothyroidism pericardial effusion. Would have to assess very well the hemodynamics (surely is committed). Quickly replace the hormone supplement, and follow closely the evolution.
Nice case
Greetings Hotdog

Major ECG findings consist of:
Sinus rhythm 75/min
Low QRS complexes
Right axis deviation
Prolonged QT interval
Diffuse T wave changes
These findings are compatible with severe hypothyroidism + pericardial effusion
I am expected marked ECG improvement after thyroid hormone therapy.

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Principales conclusiones del ECG consisten en:
Ritmo sinusal
Frecuencia cardiaca 75/lmin
Baja voltage de los complejos QRS
Desviación del eje eléctrico del QRS para la derecha
Intervalo QT prolongado
Difusos cambios en las ondas T
Estos hallazgos son compatibles con hipotiroidismo severo + derrame pericárdico
Espero mejoría del ECG después del tratamiento con hormona tiroidea.

Andres,

We all remember selected patients who were either very complicated or had an uncommon disease and we were the first physician to make the diagnosis. I had two such patients during my internship (back when the dinosaurs where still controlling the world). While I won't mention her name which I still remember to this day, some 40+ years later, she had severe myxedema with the coarse skin, facial puffiness, deep husky voice, delayed deep tendon reflexes and she was nearly comatose when her family first brought her to the hospital. She did not have a prior thyroidectomy and as a lowly intern in 1968, I made the diagnosis. She was in the hospital for almost a month at that time as we slowly up-titrated her Armour desiccated thyroid hormone replacement. As I recall her ECG, her heart rate was much slower in the range of 40 bpm, she had very low voltage on her ECG and diffuse flattened T waves. I don't recall whether or not she had a long QT but she did have a prolonged PR interval (long QT was too subtle for me at the time). My patient's QRS was slightly widened as is this case, I measure the QRS duration on the present case at about 100 ms. On this ECG, the axis is + 120 degrees and the q wave in V1 and lack of a small q wave in V5-6 raises the issue of a septal myocardial infarction but this may all resolve with thyroid replacement therapy.

My diagnosis is hypothyroid and possibly even myxedema but the history is a give away. I look forward to the answer and worry that I missed something by jumping on the obvious.

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Andres,

Todos nos lembramos de pacientes selecionados que foram muito complicados ou que tiveram uma doença rara e fomos o primeiro médico a fazer o diagnóstico. Eu tive dois desses pacientes durante meu estágio (quando os dinossauros ainda controlavam o mundo). Não menciono o seu nome,mais eu ainda me lembro até hoje, cerca de 40 anos atrás.

Ela apresentava mixedema grave com a pele grossa, inchaço facial, voz rouca profunda, reflexos tendinosos profundos lentos e estava toporosa, quase em coma quando a sua família decidiu levá-la para o hospital. Ela não tinha história de tireoidectomia prévia.

Eu um humilde estagiário nos idos anos de 1968, fiz o diagnóstico. Ela estava no hospital havia quase um mês.

Lembro que seu ECG,apresentava uma freqüência cardíaca foi muito mais lenta na faixa de 40 bpm, tinha a voltagem do QRS muito baixa e ondas T difusamente achatadas.

Não me lembro se tinha ou não um intervalo QT longo, mas ela tinha um prolongamento do intervalo PR (o diagnóstico de QT longo era sutil demais para mim na época).

A duração do QRS era levemente alargado como é este caso, (a medição do QRS neste caso é cerca de 100 ms.)

Nesta paciente o ECG, mostra um eixo em + 120 graus e onda q em V1 alem de que falta de uma pequena onda q em V5-6 aumenta a suspeita de um infarto do miocárdio septal, mas tudo isto pode ser resolver com terapia de reposição hormonal.

Meu diagnóstico: é de hipotireoidismo e, possivelmente, mixedema mesmo.

Aguardo a resposta e me disculpe se falei sobre o o óbvio.

Discusión del caso del Dr Raimundo Barbosa Barros

Evidentemente este es un caso de hipotiroidismo clinicamente y electrocardiograficamente clásico sin diagnóstico diferencial. No obstante, existe una asociación en este ECG que llama la atención: la relación entre el eje frontal de la onda P y del QRS esta muy abiertos: P – 40º y QRS + 120°. Esta asociación electrocardiográfica hace sospechar la existencia de una dilatación del ventriculo derecho.

Porque es importante este cuadro electrocardiografico? Porque una mujer, portadora de hipotiroidismo severo, puede presentar microembolias pulmonares, que inducen hipertensión pulmonar y dilatación ventricular derecha sin hipertrofia ventricular.

El sintoma mas frecuente de este cuadro es disnea sin causa aparente

Un fraternal abrazo

Samuel Sclarovsky

Discussion of Dr Raimundo´s case

Obviously this is a case of classic hypothyroidism without clinical and electrocardiographic differential diagnosis. However, there is an association in this ECG that is striking: the relationship between the frontal plane axis P wave and the QRS axis. It is very wide : P - 40 º and QRS + 120 °. This partnership makes electrocardiographic suspicion of a dilated right ventricle.

Why this electrocardiographic picture is important? Because a woman carries severe hypothyroidism, pulmonary microemboli may occur, which induce pulmonary hypertension and right ventricular dilatation without ventricular hypertrophy.

The most common symptom of this condition is unexplained dyspnea.

A fraternal hug

Samuel

Caros colegas, gostaria de emitir minha opinião nesse caso que achei muito interessante. Interpreto o ECG como ritmo sinusal, disturbio de condução do ramo direito, baixa voltagem, intervalo QT longo e desvio do eixo QRS para direita.

Em casos de tireoidectomia não é infrequente o desenvolvimento de hipopatatireoidismo por lesão da paratireóide durante o procedimento, portanto a hipocalcemia poderia justificar o intervalo QT longo.

A baixa voltagem pode ser devido a derrame pericárdico, pois as serosites são comuns nos casos de mixedema.

Gostaria de saber a opinião dos senhores quanto ao desvio do eixo QRS para direita.

Abraço a todos

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Dear colleagues, I would give my own opinion in this case which I found very interesting. I interpret the ECG as sinus rhythm, incomplete right branch, low QRS voltage, Prolonged QT interval and right axis deviation.

In cases thyroidectomy is not infrequent development of hipopatatireoidismo by parathyroid injury during the procedure, so hypocalcaemia could explain the long interval QT interval.

The low QRS voltage may be due to pericardial effusion because the serositis is common in cases of myxedema.

I would like to know others opinion about the right QRS axis deviation

Hug to everyone

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OUR FINAL DIAGNOSIS AND THEORETICAL CONSIDERATIONS

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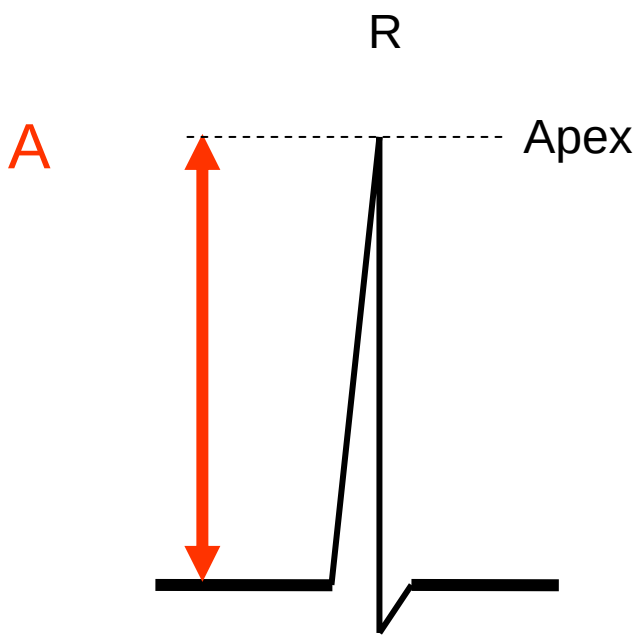
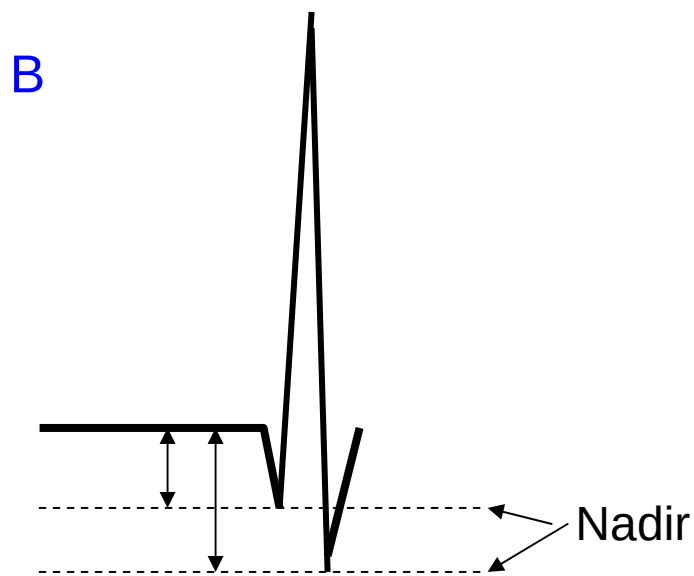
ECG diagnosis

Rhythm: sinus.; **Heart rate:** 79bpm.; **P** wave. **P** axis +30º **; PR interval:** 180ms ; **QRS duration:** 100ms.; QRS axis +125º(aVR minimally positive). Right axis deviation. the presence of pericardial effusion is associated with an increase in pulmonary artery pressure and RVH

QRS voltage: Low QRS voltage in both planes frontal (PF) and horizontal (HP): no wave exceeds 5 mm (1 large square or 5 small squares, vertically) in the leads of the FP and no wave exceeds 10 mm in HP. The voltage of the R wave should be measured from the superior border of the baseline to the apex of the R wave (A) and the voltage of Q and of S should be measured from the inferior border of the baseline to the nadir of the wave (B)





Low QRS voltage is associated with lower colloid osmotic pressure in patients with hypothyroidism[1]. Attenuation of ECG voltage in patients with anasarca correlates with weight gain, and it can be attributed to a shunting of the cardiac potentials due to the low resistance of the anasarca fluid[2].

Are causes of low QRS voltage: Obesity, **anasarca**, **pleural, pericardial, or pleuro-pericardial effusion**, left pneumothorax, **hypothermia,** cardiomyoesclerosis, extensive infarctions, cardiomyopathies, hemochromatosis, **myxedema,** caquexia, heart failure, mitral stenosis, and emphysema.

Madias and Narayan[3] describe the case of a senior man with an acute anterior myocardial infarction complicated by anoxic encephalopathy, respiratory, and acute renal failure, who developed gradually marked reduction in the QRS complexes of his ECG in the process of gaining 44 pounds, due to anasarca. Such ECG pattern has been associated with marked peripheral edema in the context of critical illness of varying etiologies. This patient did not have a pericardial effusion, and his gradually increasing weight values correlated strongly with the corresponding sums of the amplitudes of the QRS complexes of the 12-lead ECGs. The mechanism of this ECG phenomenon is attributed to a reduction of the overall transfer impedance of the conducting volume surrounding the heart, which leads to an attenuation of the potentials recorded on the body surface, due to a shunting effect of the edematous subcutaneous connective tissues. This reduction of the composite transfer impedance of the conducting medium is being mediated by the low resistivity of the water of the retained anasarca fluid.

1. **Madias JE, Bazaz R, Agarwal H, Win M, Medepalli L. Anasarca-mediated attenuation of the amplitude of electrocardiogram complexes: a description of a heretofore unrecognized phenomenon. J Am Coll Cardiol. 2001 Sep;38:756-764.**
2. **Yamanaka S, Kumon Y, Matsumura Y, et al. Link between pericardial effusion and attenuation of QRS voltage in patients with hypothyroidism. Cardiology. 2010;116:32-36.**
3. **Madias JE, Narayan V. Diminution of QRS complexes caused by anasarca after an acute myocardial infarction: a case report and a discussion of the plausible underlying pathophysiological mechanisms. J Electrocardiol. 2003 Jan;36:59-66.**

V1 lead: triphasic rsr´ patter suggest incomplete RBBB (QRSd 100ms). Anasarca leads to apparent amelioration of the features of bundle branch block (BBB) or intraventricular conduction delay (IVCD) , with subsequent return to the baseline complete BBB or IVCD after loss of fluid overload. Consequently, accurate characterization of a patient's BBB or IVCD is interfered with by the presence of anasarca. The pathophysiologic mechanism of this phenomenon has been traced to decreases of voltage across the entire ECG curve due to attenuation of the electrical impedance of the body volume conductor due to accumulation of the anasarca fluid3;. Eventually is observed apparent conversion of complete to incomplete bundle branch block due to peripheral edema[1].

QT interval: QT= 520ms. HR 79bpm. RR: 0.80 seconds, QT for women mean value 364ms, Lower limit = 313ms, Upper limit = 401. Prolonged QT interval. Madias[2] evaluate the hypothesis that changes in QTc in patients with anasarca and after hemodialysis are at least partially apparent, due to changing edematous states, and not totally due to altered electrophysiology. The author measured the QTc in patients with anasarca on admission, at peak weight (N = 28), and at their subsequent lowest weight (N = 12), in 28 control patients without change in weight during hospitalization, and in one patient before and after 26 hemodialysis sessions.

In the patients with anasarca, the QTc was 451 +/- 36 ms on admission and dropped to 423 +/- 46 ms at peak weight. QTc was 421 +/- 44 ms at peak weight and raised to 434 +/- 30 at subsequent lowest weight. In the controls, QTc on admission and at discharge were 435 +/- 34 and 428 +/- 23 ms, correspondingly. QTc increased from 472 +/- 18 ms before to 489 +/- 36 ms after hemodialys. Alterations in QTc in anasarca, or hemodialysis suggest that the changes in the QTc may be partially only apparent, and due to the ECG machine-based measurement of the attenuated/augmented QRST complexes resulting from fluid shifts.

1. **Madias JE. Apparent conversion of complete to incomplete bundle branch block due to peripheral edema J Electrocardiol. 2005 Oct;38:415-416.**
2. **Madias JE. QTc interval in patients with changing edematous states: implications on interpreting repeat QTc interval measurements in patients with anasarca of varying etiology and those undergoing hemodialysis. Pacing Clin Electrophysiol. 2005 Jan;28:54-61.**

In this case we observe a QT interval prolongation with very long ST segment and a late T wave occurrence (similar with congenital LQT3 variant). This phenomena is characteristic of hypocalcaemia, frequent in patients with antecedent of previous total tyroidectomy. Additionally, hypoparathyroidism is associated with chronic hypocalcaemia which can have a variety of manifestations including reversible dysfunction of both ventricles presenting as a dilated cardiomyopathy or right dilated cardiomyopathy(global)[1] . The regulation of serum and bone calcium is ensured by parathormone (PTH), D vitamin and calcitonin, through a complex mechanism of loop and feedback, which acts on the bones, kidneys and intestines. Calcium is also affected by the level of serum phosphorus and magnesium.

CAUSES OF HYPOCALCEMIA

1) **HYPOALBUMINEMIA:** it maintains normal ion calcium.
2) **HIPOPARATHYROIDISM: by accidental surgical exeresis of parathyroid glands during the course of total thyroidectomy** by neo-idiopathic hypoparathyroidism: sporadic or familial. These symptoms develop with low serum PTH.
3) **PSEUDO-HYPOPARATHYROIDISM:** by bone and renal resistance to PTH hormone action. These symptoms develop with high serum PTH.
4) **HYPOMAGNESEMIA:** it suppresses PTH secretion and it causes bone resistance to the action by the hormone.
5) **DEFICIT OF VITAMIN D:** nutritional, poor enteric absorption, by liver diseases, by drugs that degrade vitamin D (phenobarbital, alcohol, diphenylhydantoin);
6) **NEPHROTIC SYNDROME:** because it causes hypoalbuminemia and serum reduction of 25 OH D3 transporter of vitamin D;

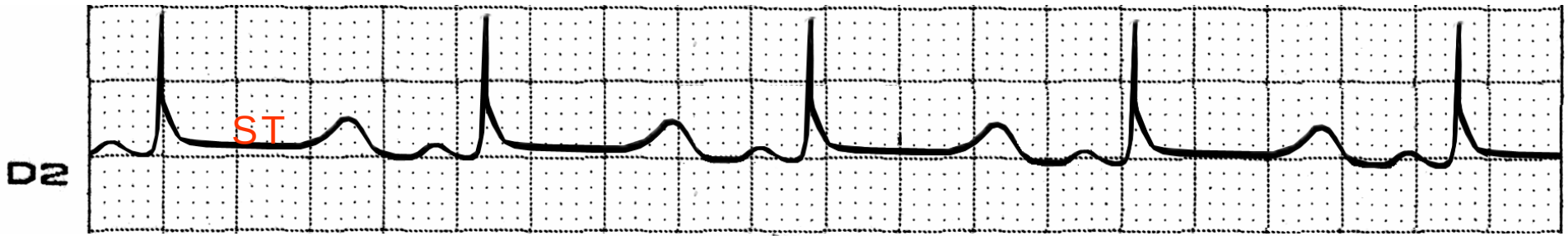
1. **Jariwala PV, Sudarshan B, Aditya MS, et al. Hypoparathyroidism--a cause of reversible dilated cardiomyopathy. J Assoc Physicians India. 2010 Aug;58:500-502**.

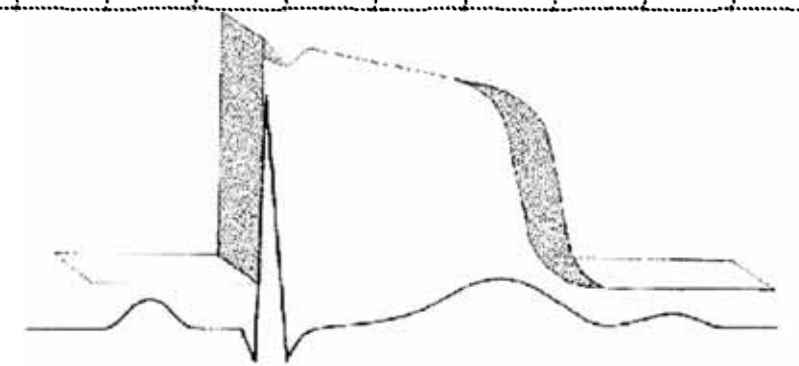
7) VITAMIN D-DEPENDENT RICKETS;
8) RENAL INSUFFICIENCY;
9) HYPERPHOSPHATEMIA;
10) "HUNGRY BONE" SYNDROME: osteoblastic metastasis;
11) CHRONIC PANCREATITIS (it decreases vitamin D absorption);
12) PARTIAL GASTRECTOMY (it decreases vitamin D absorption);
13) CHRON'S DISEASE (it decreases vitamin D absorption).

THE ECG IN HYPOCALCEMIA

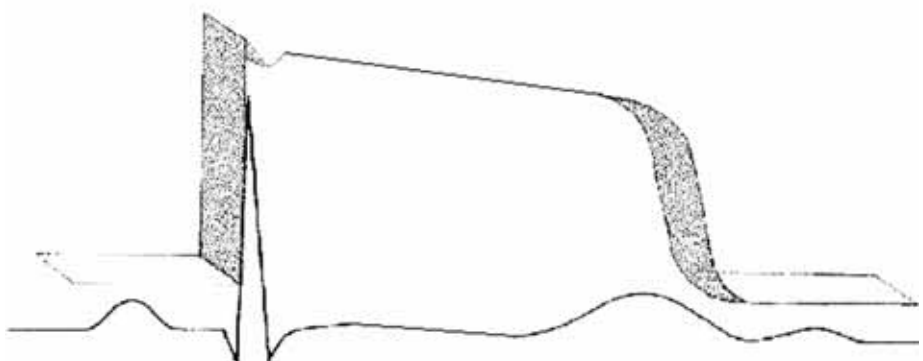
1. Usually ECG manifestations appear in ECG when ion calcium levels reach values below $\leq$ 7 mg/dl.
2. Major ECG manifestation: QT interval prolongation at the expense of the increase in ST segment duration, with no modification of T wave. The phenomenon is observed in hypocalcemia and in hypothermia.
3. The degree of ST segment prolongation is proportional to severity and velocity of hypocalcemia installation.
4. Prolongation of Q-oTc interval: interval that extends from Q wave onset up to T wave onset, corrected by heart rate.
5. T wave: it may be apiculate and with late inversion. These facts are more evident in the right precordial leads.
6. Hypocalcemia does not affect P wave, PR interval and U wave, and it may shorten QRS complex duration; however, this shortening is hardly acknowledged.
7. There are references to wide and diphasic U waves.
8. It causes decrease in sensitivity with digitalis.
9. It produces decrease of myocardial contractility (negative inotropic), and it may lead to CHF, hypotension and angina.

10 Characteristically, hypocalcemia is not associated with arrhythmias; however, ventricular arrhythmias such as TdP, induced form of VF, may be recorded.
11, It may be confused with chronic coronary insufficiency, where occasionally ST segment is rectified, and QT interval prolongation.
It must be differentiated from hypopotasemia, which prolongs the QT interval, affecting the T wave and causes prominent U wave.





NORMAL



HYPOCALCEMIA

HYPOTHYROIDISM OVERVIEW

Hypothyroidism is a condition characterized by abnormally low thyroid hormone production. There are many disorders that result in hypothyroidism. These disorders may directly or indirectly involve the thyroid gland. Because thyroid hormone affects growth, development, and many cellular processes, inadequate thyroid hormone has widespread consequences for the body.

Hypothyroidism is a very common condition. It is estimated that 3% to 5% of the population has some form of hypothyroidism. The condition is more common in women than in men, and its incidence increases with age.

Are common causes of hypothyroidism in adults:

Hashimoto's thyroiditis

Lymphocytic thyroiditis (which may occur after hyperthyroidism)

Thyroid destruction (from radioactive iodine or surgery)

Pituitary or hypothalamic disease

Medications

Severe iodine deficiency.

THYROID DESTRUCTION SECONDARY TO RADIOACTIVE IODINE OR SURGERY

Patients who have been treated for a hyperthyroid condition (such as Graves' disease) and received radioactive iodine may be left with little or no functioning thyroid tissue after treatment. The likelihood of this depends on a number of factors including the dose of iodine given, along with the size and the activity of the thyroid gland. If there is no significant activity of the thyroid gland six months after the radioactive iodine treatment, it is usually assumed that the thyroid will no longer function adequately. The result is hypothyroidism.

Similarly, total removal of the thyroid gland during surgery will be followed by hypothyroidism as the present case.

The symptoms of hypothyroidism are often subtle. They are not specific (which means they can mimic the symptoms of many other conditions) and are often attributed to aging. Patients with mild hypothyroidism may have no signs or symptoms. The symptoms generally become more obvious as the condition worsens and the majority of these complaints are related to a metabolic slowing of the body. Common symptoms are: Fatigue, depression, modest weight gain, cold intolerance, excessive sleepiness, dry, coarse hair, constipation, dry skin, muscle cramps, increased cholesterol levels, decreased concentration, vague aches and pains swelling of the legs.

Early manifestations: muscle hypotonia, fatigue, cold intolerance, increased sensitivity to cold, constipation, constipation depression muscle cramps and joint pain Goiter, Thin, brittle fingernails, paleness, decreased sweating, dry, itchy skin weight gain and water retention, bradycardia (low heart rate – fewer than sixty beats per minute)

Late Slow speech and a hoarse, breaking voice – deepening of the voice can also be noticed, caused by Reinke's edema. dry puffy skin, especially on the face, thinning of the outer third of the eyebrows (sign of Hertoghe), abnormal menstrual cycles, low basal body temperature.

As the disease becomes more severe, there may be puffiness around the eyes, a slowing of the heart rate, a drop in body temperature, and heart failure. In its most profound form, severe hypothyroidism may lead to a life-threatening coma (myxedema coma or torpor as the present case). In a severely hypothyroid individual, a myxedema coma tends to be triggered by severe illness, surgery, stress, or traumatic injury. This condition requires hospitalization and immediate treatment with thyroid hormones given by injection.

Properly diagnosed, hypothyroidism can be easily and completely treated with thyroid hormone replacement. On the other hand, untreated hypothyroidism can lead to an enlarged heart (cardiomyopathy), worsening heart failure, and an accumulation of fluid around the lungs (pleural effusion).

UNCOMMON

Impaired memory, impaired cognitive function (brain fog) and inattentiveness, Diminished cardiac output and decreased contractility , reactive (or post-prandial) hypoglycemia, sluggish reflexes, hair loss, Anemia caused by impaired hemoglobin synthesis (decreased EPO levels), impaired intestinal iron and folate absorption or B12 deficiency from pernicious anemia, difficulty swallowing, shortness of breath with a shallow and slow respiratory pattern, Increased need for sleep, Irritability and mood instability, yellowing of the skin due to impaired conversion of beta-carotene to vitamin A, Impaired renal function with decreased glomerular filtration rate, Elevated serum cholesterol, acute psychosis (myxedema madness) (a rare presentation of hypothyroidism), due to impairment of testicular testosterone synthesis, decreased sense of taste and smell (anosmia), puffy face, hands and feet (late, less common symptoms), gynecomastia, deafness.

THE CARDIOVASCULAR RISK

The cardiovascular risk in patients with hypothyroidism is related to an increased risk of functional cardiovascular abnormalities and to an increased risk of atherosclerosis. The pattern of cardiovascular abnormalities is similar in subclinical and overt hypothyroidism, suggesting that a lesser degree of thyroid hormone deficiency may also affect the cardiovascular system. Hypothyroid patients, even those with subclinical hypothyroidism, have impaired endothelial function, normal/depressed systolic function, left ventricular diastolic dysfunction at rest, and systolic and diastolic dysfunction on effort, which may result in poor physical exercise capacity. There is also a tendency to increase diastolic blood pressure as a result of increased systemic vascular resistance[1].

1. **Biondi B, Klein I. Hypothyroidism as a risk factor for cardiovascular disease. Endocrine. 2004 Jun;24(1):1-13**.

All these abnormalities regress with L-T4 replacement therapy. An increased risk for atherosclerosis is supported by autopsy and epidemiological studies in patients with thyroid hormone deficiency. The "traditional" risk factors are hypertension in conjunction with an atherogenic lipid profile; the latter is more often observed in patients with TSH >10 mU/L.
More recently, C-reactive protein, homocysteine, increased arterial stiffness, endothelial dysfunction, and altered coagulation parameters have been recognized as risk factors for atherosclerosis in patients with thyroid hormone deficiency. This constellation of reversible cardiovascular abnormalities in patient with TSH levels <10 mU/L indicate that the benefits of treatment of mild thyroid failure with appropriate doses of L-thyroxine outweigh the risk.

ANASARCA

Anasarca, also known as "extreme generalized edema" is a medical condition characterized by widespread swelling of the skin due to effusion of fluid into the extracellular space.
Some potential underlying medical conditions behind the fluid build up which leads to the swelling in anasarca may include: congestive heart failure, eclampsia / pre-eclampsia, liver failure (cirrhosis of the liver: alcoholic liver disease (ALD), chronic hepatitis B or C, autoimmune hepatitis, and Non-alcoholic steatohepatitis (NASH).) or renal failure/disease, severe malnutrition/protein deficiency, hookworm (ancylostomiasis), systemic amyloidosis, severe hypothyroidism, POEMS syndrome (Polyneuropathy, organomegaly, endocrinopathy, monoclonal gammopathy, and skin changes), the administration of exogenous intravenous fluid, certain plant-derived anticancer chemotherapeutic agents, such as docetaxel, cause anasarca through a poorly understood systemic capillary leak syndrome or Clarkson syndrome[1] a rare clinical condition characterized by generalized edema associated to monoclonal gammopathy. Finally, anasarca can be caused by idiopathic, i.e. primitive lymphedema.

1. **Clarkson B, Thompson D, Horwith M, Luckey A. Am J Med. 1960;29:193-216**

Prezado Dr. Andrés, fantástica a análise eletrocardiográfica do Prof. Samuel. Só faltou descrever a hipocalcemia(QT aumentado às custas do segmento ST)
Exames laboratoriais:
Cálcio iônico:0,39(N=1,12-1,30) Cálcio não iônico: 3,3; Sódio:113; K:3,8; TSH:32,0 T4:18,9
Ecocardiograma:VE:53/45 AE:35 FE: 32%. Aumento importante de câmaras direitas com disfunção sistólica do VD + Insuficiência tricúspide importante. Derrame pericárdico discreto
Após 1 semana de reposição hormonal,corticoterapia e correção eletrolítica a paciente apresentou sensível melhora clínica.
Posteriormente mando a evolução do ECG
Um abraço
Raimundo Barbosa

Dear Dr. Andrés, fantastic electrocardiographic analysis of Prof. Samuel.
He only missed describe hypocalcaemia (QT interval prolongation consequence of ST segment and late T wave)
Laboratory: Ionic $Ca^{2+:}$ 0.39 (N = 1.12 to 1.30) Non ionic Calcium: 3.3; Na^{+}: 113; K: 3.8; TSH:32.0; T4: 18.9
Echocardiography: LV: 53/45 LA: 35 EF: 32%. Significant increase of right chambers with systolic dysfunction of RV and severe tricuspid insufficiency. Minimal pericardial effusion.
After one week of hormonal reposition therapy, corticoidetheraphy, and electrolytic correction the patient had a good evolution