ELDERLY HYPERTENSIVE WOMAN WITH SEVERE UREMIA AND ST-ELEVATION ACUTE CORONARY SYNDROME

IDOSA HIPERTENSA EM UREMIA E QUADRO DE SINDROME CORONARIANA AGUDA COM ELEVAÇÃO DO SEGMENTO ST

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M.A.R., 70 anos, hipertensa de longa data. Antecedente de nefrectomia esquerda.
Há 20 dias vinha com dispnéia progressiva aos esforços. Admitida na sala de emergência há 4 dias por quadro de franca insuficiência respiratória.
Antecedentes pessoais: Hipertensa de longa data, nefrectomizada do rim esquerdo por pielonefrite crônica. Em uso regular de IECA.
EF: PA: 160/80 Laboratório na admissão (26/09/2009): pH: 7,04 Saturação de oxigênio arterial (pO2: 86% ; Intubação orotraqual/ventilação mecânica IOT/VM PO2: 126 PCO2: 14; K: 7,0mEq/L; HCO3: 3,8 Glicemia: 134 Ureia: 475; Creatinina: 18,9; Cálcio: 0,47; Hemoglobina: 6,4; Tp: 0,13; CK-MB: 14,1U/L
DIAGNÓSTICO: IRA por acidose metabólica. IRC agudizada. Hipercalemia. Hipocalcemia

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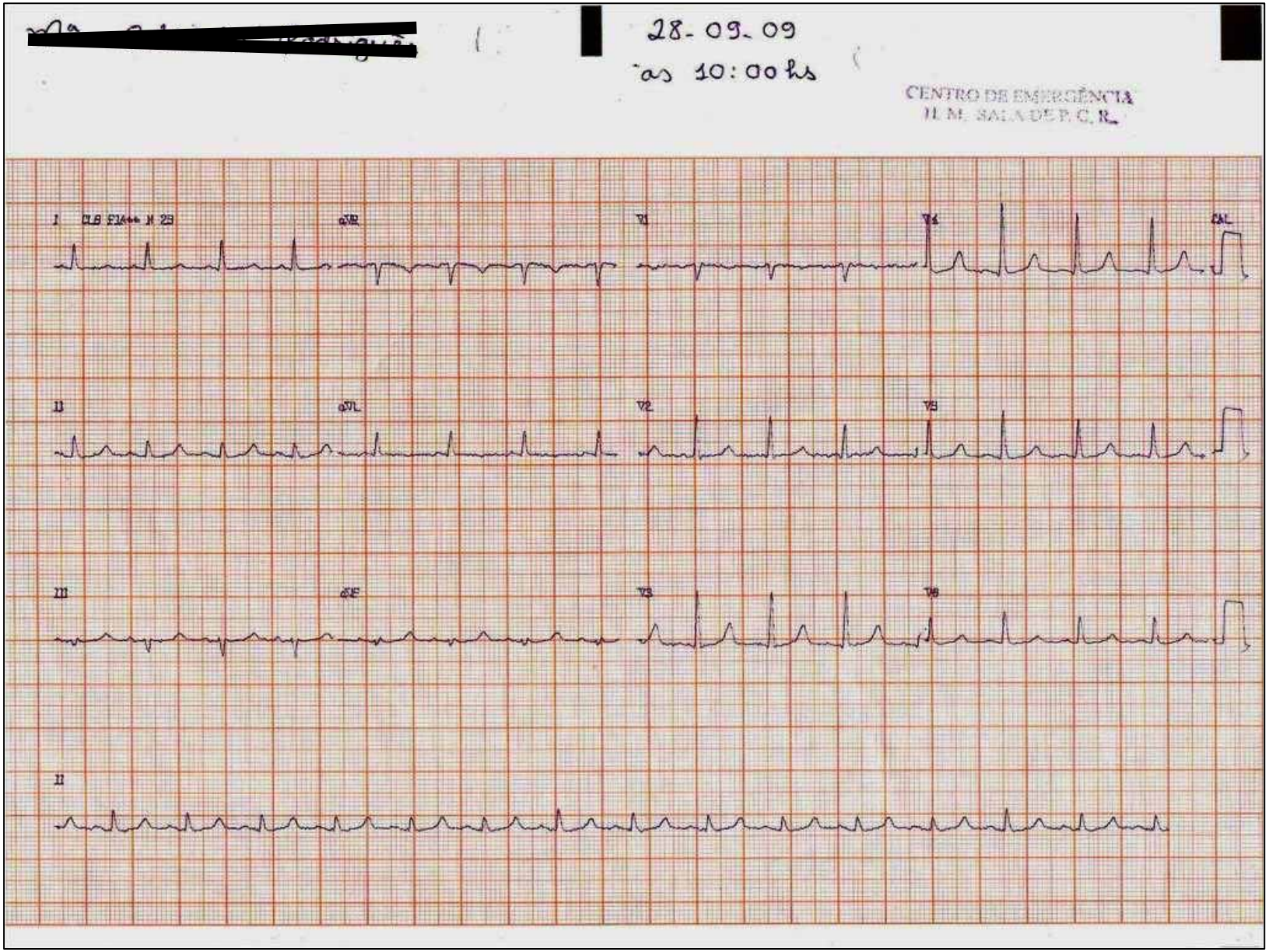
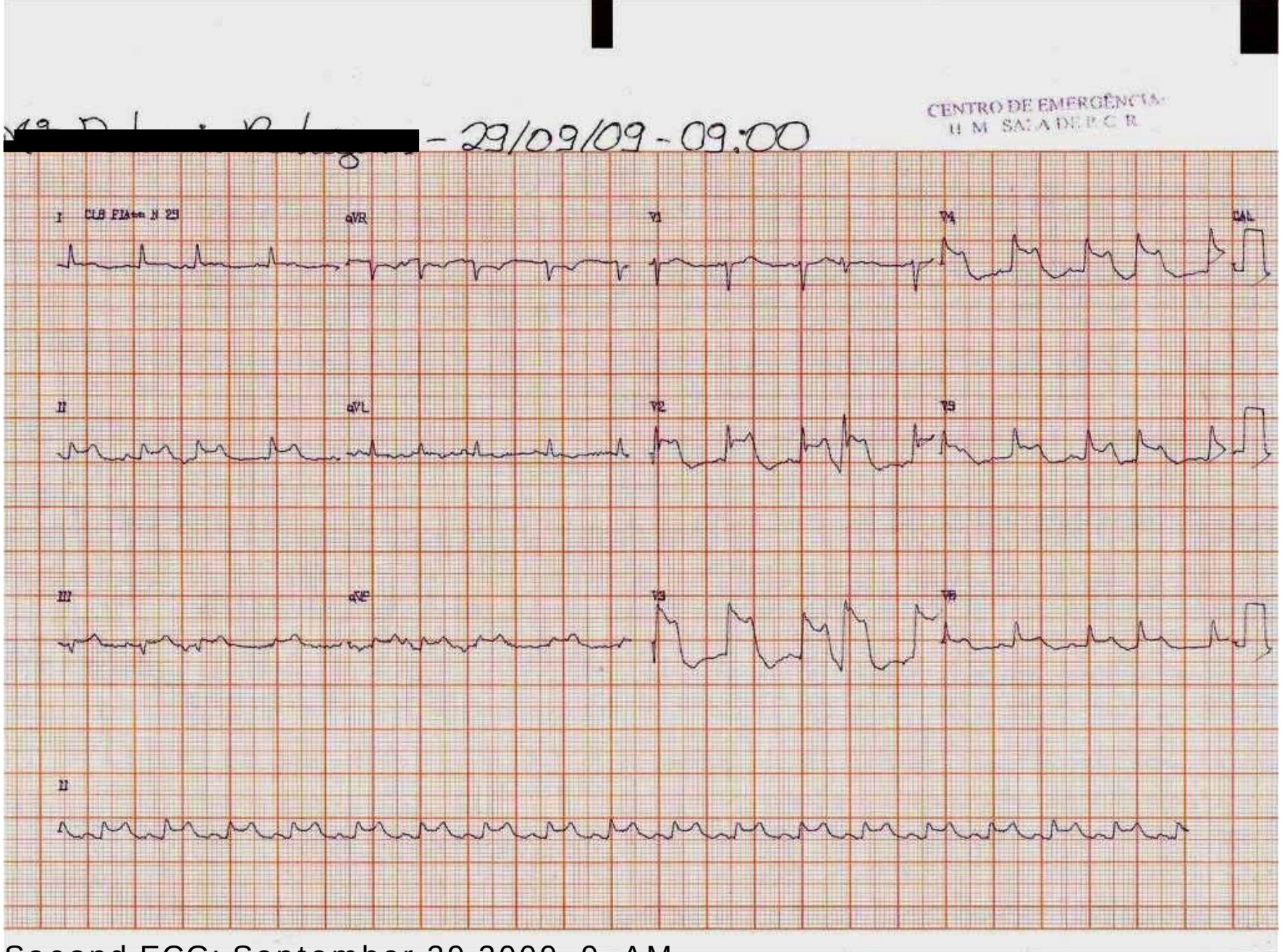
CORONARY CARE UNIT CASE REPORT

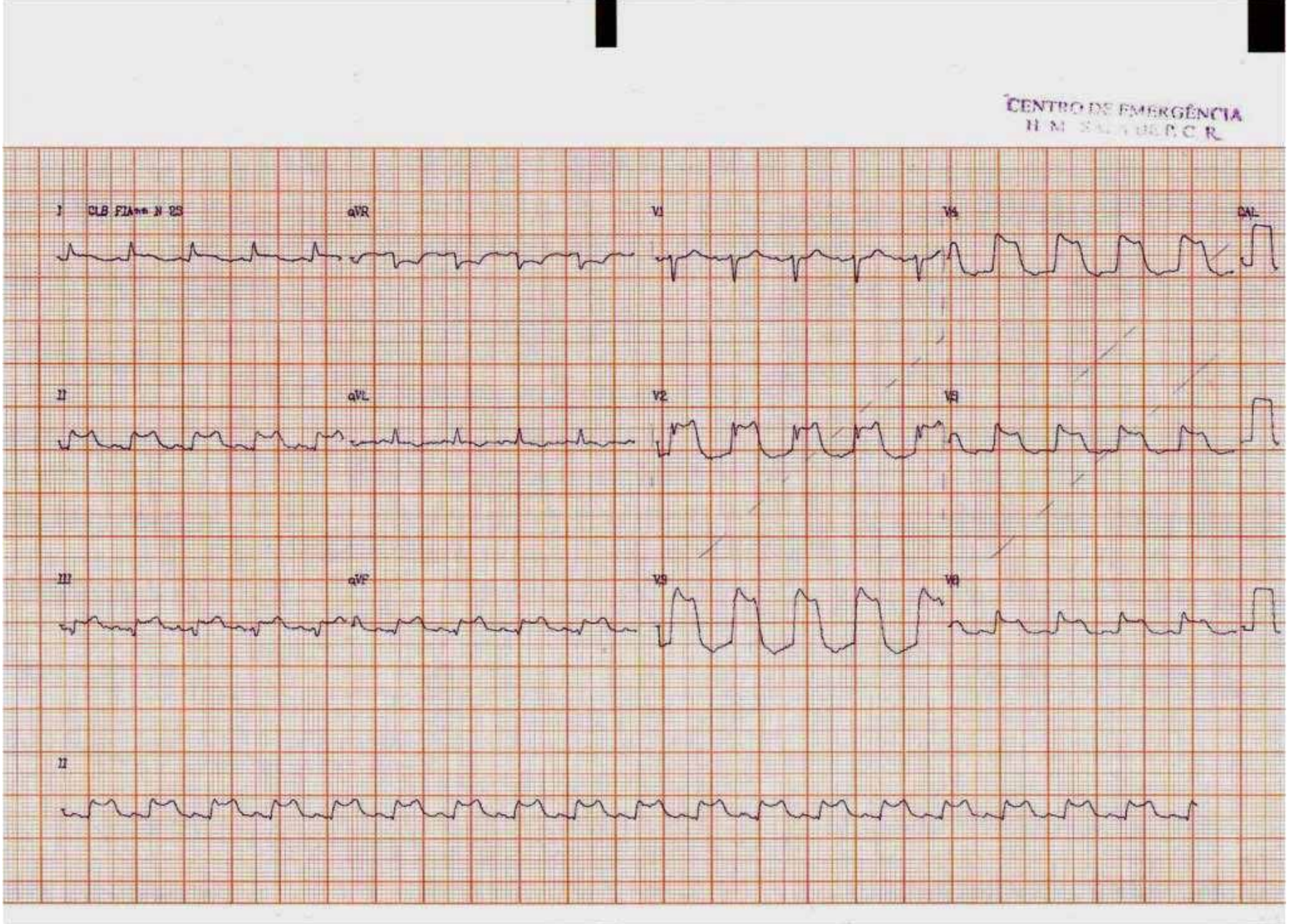
M.A.R., 70, had longstanding high blood pressure. Personal of longstanding history of left nephrectomized left kidney consequence of chronic pyelonephritis a long time ago. There had been 20 days with progressive exertional dyspnea. Admitted to the emergency room for 4 days a framework for respiratory insufficiency.
In regular use of ACE inhibitors.
PE: BP: 160/80 Laboratory at admission 9.26 pH: 7.04; arterial oxygen saturation (pO2: 86% OTI / VM; PO2: 126 PCO2: 14, K: 7.0 mEq / L, HCO3: 3.8; Glucose: 134; Blood Urea Nitrogen (BUN): 475mg/dL; serum creatinine level : 18.9mg/dL. ; Calcium: 0.47 mmol/l**(normal *total calcium* of 2.2-2.6 mmol/L (9-10.5 mg/dL) and a normal *ionized calcium* of 1.1-1.4 mmol/L (4.5-5.6 mg/dL).), Hemoglobin: 6.4mmol/L;** Troponin **Tp = 0.13; CK-MB: 14.1U/L**
DIAGNOSTICS: Acute kidney injury by metabolic acidosis- acute-on-chronic renal failure (AoCRF) –uremia - hyperkalemia- hypocalcemia.



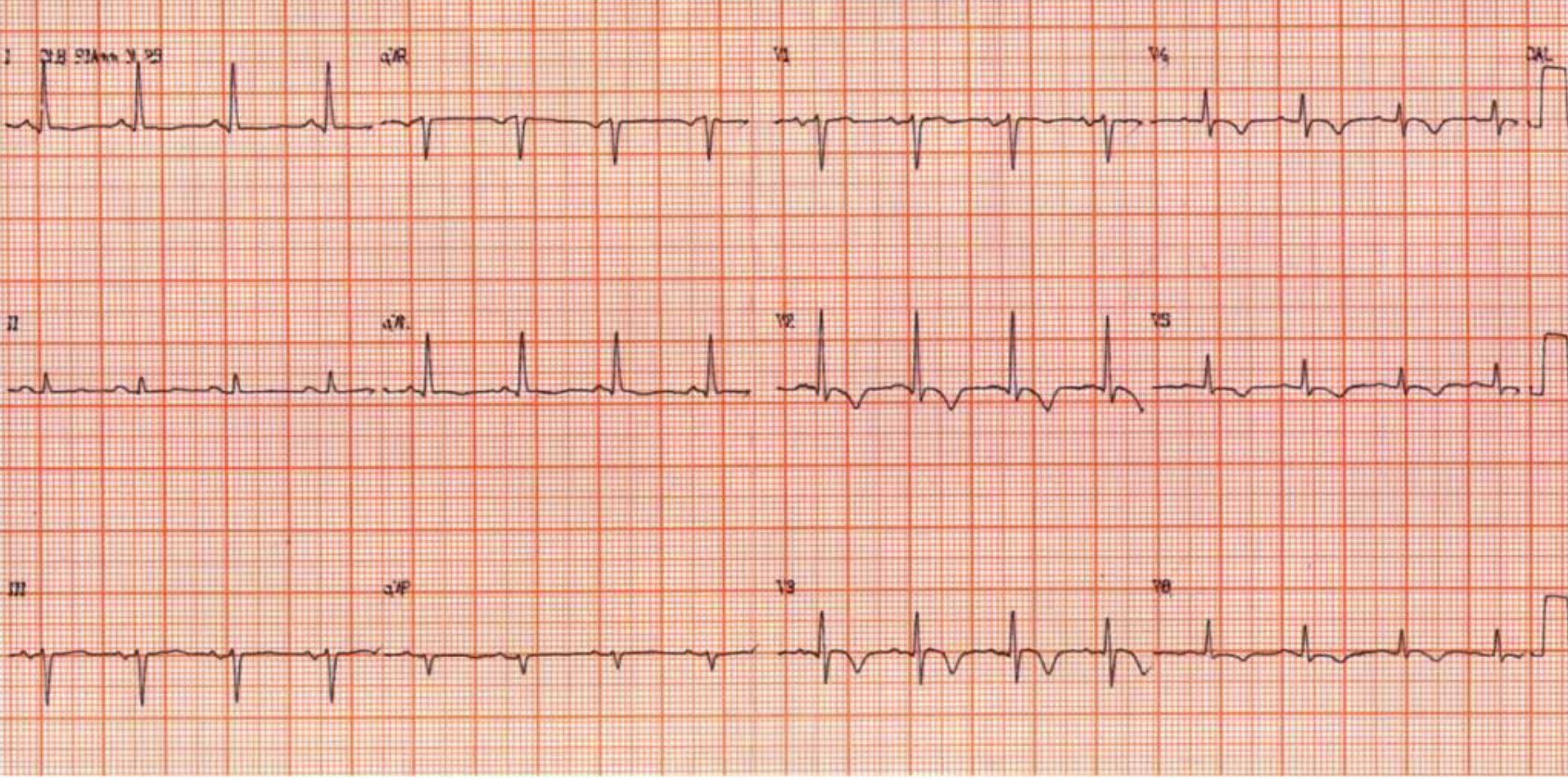
First ECG: September 28, 2009. 10 AM. Two days after admission



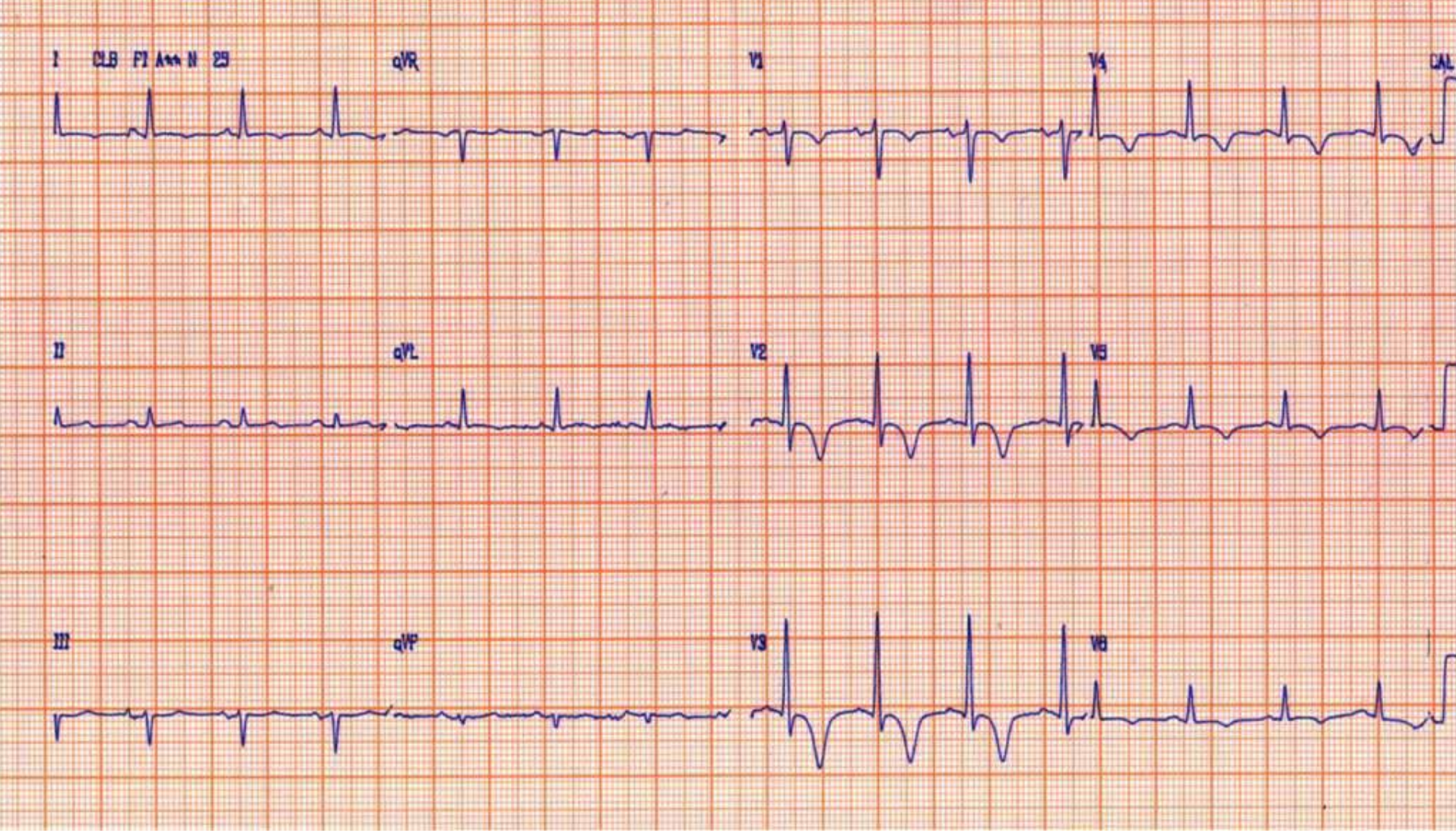
Second ECG: September 29-2009 9. AM
Patient with chest pain.



Patients with worsening chest pain.
What is the diagnosis and management?



ECG one week after admission.
T wave inversion with no formation of Q wave form V_2 to V_6.
Early appearance of q wave and prominent R waves in V_2 and in precordial leads indicative of counterclockwise heart rotation in longitudinal axis: It could be an indicative of LVH associated with QRS axis on frontal plane located in -10º .



ECG two week after admission

COLLEAGUES OPINIONS
OPINIÃO DOS COLEGAS

Classic pericarditis Spodick ! A possible uremic pericarditis.

Pericardite clássica de Spodick! Uma possível pericardite urémica.

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¡Hermoso ECG! Se trata de una oclusión completa de una arteria descendente anterior muy larga después de la 1ª diagonal y 1ª septal. El vector lesión se dirigue hacia delante y hacia abajo. Hay por tanto, afectación antero-apical y de la zona inferior por long wrap-around Left anterior descending. Isquemia grado 3. Reperfusión inmediata.

Un cordial saludo,

Javier García Niebla
Servicios Sanitarios del Area de Salud de El Hierro, Valle del Golfo Health Center, Islas Canarias, Spain.

Beautiful ECG! This is a complete occlusion of left anterior descending artery very long after the 1st diagonal and 1 st septal. The injury vector is directed forward and downward. There is therefore affected antero-apical and the inferior wall by a long wrap-around left anterior descending. Grade 3 ischemia. Immediate reperfusion.
Sincerily

Javier Garcia Niebla
Health Area Service Health of El Hierro, Bay Valley Health Center, Canary Islands, Spain.

FINAL CONCLUSIONS

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FINAL DIAGNOSIS/ DIAGNÓSTICO FINAL

Diagnóstico clínico: ***Pericardite urémica***

Diagnóstico ECGs o segundo e terceiro traçado mostram elevação do segmento ST de concavidade superior de V2 a V6 e nas tres derivações inferiores. (os desnivelamentos do segmento ST são extensos) Imagem em espelho ou recíproca é observada na derivação aVR. Na pericardite ocasilonalmente cambios recíprocos do segmento ST são observados em aVR. Estos ECG encontram-se na primeira fase da pericarditis.

O terceiro e quarto ECGs encontram-se no terceiro estagio da pericarditis aguda: onda T invertida sem formação de onda Q

ECO mostrou derrame percicárdico

Infelizmente a paciente também realizou coronariografia que resultou normal

Instituida hemodiálise.

Clinical Diagnose: ***Uremic Pericarditis***

ECGs Diagnose: ***the second and third ECGs show ST segment elevation concave to the top from V_2 to V_6 and all inferior leads. (The*** ST segment changes are not segmental)

Mirror image is observed in aVR lead. *In pericarditis occasionally reciprocal ST changes are observed in aVR. These ECGs are in the first stage of acute pericariditis.*

The third and fourth ECGs are in the third stage of acute pericarditis: Why? Because we observe an inversion of T wave, with no formation of Q wave. Additionally, we observe early appearance of q wave and prominent R waves in V_2 indicative of counterclockwise heart rotation in longitudinal axis: It could be an indicative of LVH.

ECHO showed pericardial efusion

Unfortunatelly, the patient also performed coronariography, which was normal Instituted hemodialysis.

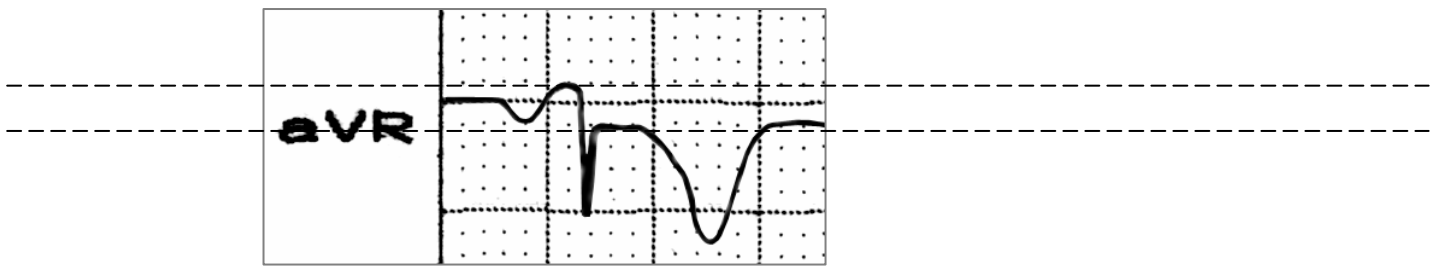
The fourth stages or phases of acute pericarditis/ Os quatro estágios o fases da pericardite aguda

FIRST PHASE: ST segment elevation (<5mm) of superior concavity. It is observed only two hours before chest pain and it lasts for several days. ST segment changes are *EXTENSIVE AND NOT TOO INTENSE,* normally noticeable in several leads simultaneously, excluding V_1. Occasionally reciprocal alterations are observed in aVR.

PRIMEIRA FASE: Elevação do segmento ST < 5mm e de concavidade superior. Estos cambios são observados duas hóras após a dor precordial e se extende por vários dias.

O cambios do segmento ST são extensos mas não intensos, normalmente observados em várias derivações simultaneamente(não segmentar), excluindo V1 Ocasionalmente se observam cambios recíporcos ou em espelho em aVR.

RECIPROCAL OR MIRROR CHANGES/ CÂMBIOS RECÍPROCOS O EM ESPELHO



SECOND PHASE: ST segment returns to baseline and flat T wave.

SEGUNDA FASE: **O segmento ST retorna a linha de base e a onda T fica aplanada.**

THIRD PHASE: inversion of T wave, with no formation of pathological Q wave.

TERCEIRA FASE: **inversão de onda T sem formação de onda Q patológica**

FOURTH PHASE: ECG normalization with gradual reversion of T wave inversion.

QUARTA FASE: **Normalização do ECG com reversão da inversão da onda T.**

PERICARDITIS: EVOLUTIONARY CLASSIFICATION
PERICADITE: CLASSIFICAÇÃO EVOLUTIVA

1. Acute: **< 6 weeks (fibrinous with pericardial effusion).**

2. Sub-acute: **6 weeks to 6 months (constrictive and pericardial effusion).**

3. Chronic: **>6 months it divides into:**

Constrictive
Pericardial effusion
Adhesive without constriction.

OVERVIEW

Renal Failure(RF) is a common cause of pericardial disease, including pericarditis and pericardial effusions, and a less frequent cause of chronic constrictive pericarditis[1;2]. Advances in management have decreased the incidence of pericarditis in patients with RF, but this problem is still associated with significant morbidity and occasional mortality.

Two forms of pericarditis in renal failure have been described:

I) Uremic pericarditis: results from inflammation of the visceral and parietal membranes of the pericardial sac. There is a correlation with the degree of azotemia (the BUN is usually >60 mg/dL (22 mmol/L), although the pathogenesis is poorly understood. Except in the case of systemic immune disorders (such as lupus erythematosus or scleroderma), there is no relationship with the underlying cause of RF.

Prevalence: 6-10% in patients with acute or chronic RF. When patients with large effusions are studied, uremia may account for up to 20% of cases in some series.

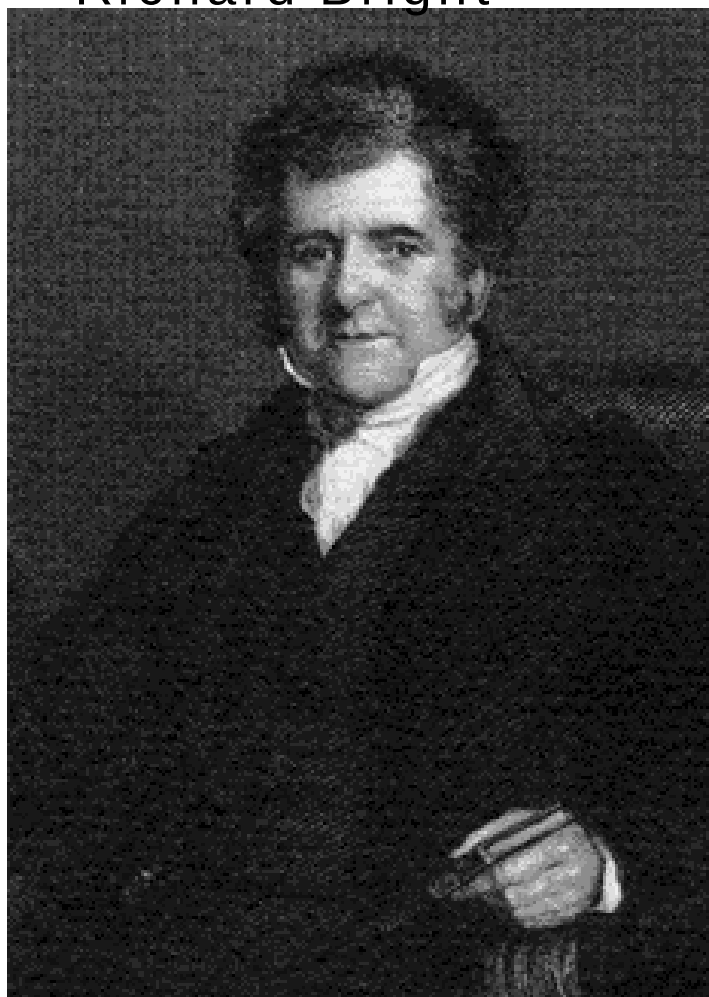
Mobility/mortality: Uremic pericarditis continues to be associated with significant morbidity and occasional mortality. Mortality may occur in 3-5% of cases resulting from cardiac tamponade or arrhythmias.

History: Richard Bright (28 September 1789 – 16 December 1858) was an English physician and early pioneer in the research of kidney disease. Richard make the first description of uremic pericarditis a long time ago. His research into the causes and symptoms of kidney disease led to his identifying what became known as Bright's disease. For this, he is considered the "father of nephrology". He was born in Bristol, Gloucestershire, the third son of Sarah and Richard Bright Sr., a wealthy merchant and banker. Bright Sr. shared his interest in science with his son, encouraging him to consider it as a career.

1. **Alpert MA, Ravenscraft MD SOPericardial involvement in end-stage renal disease.Am J Med Sci. 2003;325:228-236.**
2. **Maisch, B, Seferović, PM, Ristić, AD, et al. Guidelines on the diagnosis and management of pericardial diseases executive summary; The Task force on the diagnosis and management of pericardial diseases of the European society of cardiology. Eur Heart J 2004; 25:587.**

In 1808, Bright Jr. joined the University of Edinburgh to study philosophy, economics and mathematics, but switched to medicine the following year. In 1810, he accompanied Sir George Mackenzie on a summer expedition to Iceland where he conducted naturalist studies. Bright then continued his medical studies at Guy's Hospital in London and in September 1813 returned to Edinburgh to be granted his medical doctorate. His thesis was *De erysipelate contagioso* (*On contagious erysipelas*).During the 1820s and 1830s Bright again worked at Guy's Hospital, teaching, practising and researching medicine. There he worked alongside two other celebrated medical pioneers, Thomas Addison and Thomas Hodgkin.

Richard Bright



Since that classic description, this common complication of chronic RF has evolved from an ominous event heralding the terminal stages of disease to an event that, with early management, is likely to have a good outcome. Furthermore, advances in dialysis technology with early and timely management of chronic renal failure have dramatically reduced the prevalence of uremic pericarditis. Uremic pericarditis has a prevalence of 6-10% in patients with acute or chronic RF, and it continues to be associated with significant morbidity and occasional mortality.

PATHOPHYSIOLOGY

Uremic pericarditis is thought to result from inflammation of the visceral and parietal layers of the pericardium by metabolic toxins that accumulate in the body owing to kidney failure. Other factors may be involved, however, because pericarditis also may occur in patients with chronic RF who already are receiving dialysis therapy. The putative toxins suggested to precipitate uremic pericarditis when they accumulate are poorly characterized, but they may include urea, creatinine, methylguanidine, guanidinoacetate, parathyroid hormone, beta2-microglobulin, uric acid, and others. More than one toxin apparently may be involved, though considerable controversy surrounds this point. The precise pathogenetic changes induced by these toxins when causing uremic pericarditis have not been elucidated, though a rough correlation with the degree and the duration of azotemia exists; the blood urea nitrogen (BUN) level is usually greater than 60 mg/dL (22 mmol/L). Histopathological examination often reveals adhesions between the pericardial membranes, which are thickened. Uremic pericarditis may be associated with hemorrhagic or serous effusion, although considerable overlap exists. Hemorrhagic effusions are more common and result in part from uremia -induced platelet dysfunction.

Anamnesis:

Patients with uremic pericarditis often complain of chest pain. This chest pain characteristically is pleuritic and is worse in the recumbent position, but it is relieved by leaning forward. Some patients may have fever. Patients with sizable effusions may present with dyspnea. Palpitations may be the presenting complaint.

Physical

On examination, patients with uremic pericarditis may have tachycardia or hypotension in cases with impending tamponade. A pericardial friction rub is present in most cases, but the rub may be transient. Signs of tamponade, including inspiratory jugular venous distension and paradoxical pulse, also may be present. Aside from the cardiac findings, patients may be febrile and present with confusion.

DIFFERENTIAL DIAGNOSIS

	PERICARDITIS	CORONARY ARTERY DISEASE
Number of involved leads	More (diffuse) extensive	Lesser (SEGMENTAR)
Intensity of the phenomena	Lesser	More
Reciprocal effect Mirror image	Only in aVR	Present
	PERICARDITIS	EARLY REPOLARIZATION PATTER
Response to strain	ST segment elevation is not modified.	Frequent return of ST to baseline. T wave may normalize.
Hyperventilation	T polarity is not modified.	T polarity may be modified.
Presentation	Transitory.	Stable.
HR	Frequent tachycardia.	Frequent bradycardia.
Clinics	Marked alteration.	Asymptomatic.
Age range:	40 or more.	20 to 40 years old.

CLINICAL CAUSES OF J POINT AND ST SEGMENT ELEVATION UPWARDLY CONVEX

1.Acute Coronary Syndromes (ACSs): STE-ACS[1].

ST segment elevation myocardial infarction (STEMI) Acute Myocardial Infarction (30%)

ST segment elevation Unstable Angina (UASTE) 38%

Lambda-like ST segment elevation in AMI[2] CAD is the major determinant of SCD, and its predisposing genetic background is complex. Very recently, a first genome-wide association study on primary VF was published (See next slide).

3. Percutaneous Coronary Intervention (PCI) enhanced P2Y12 inhibition (protein is found on the surface of blood platelet cells and is an important regulator in blood clotting) with either higher clopidogrel dosing or new oral antiplatelet agents, including prasugrel and ticagrelor, in the setting of STEMI, focusing on results in the setting of primary PCI[3].

4. Prinzmetal's angina, *variant angina* or angina inversa[4].

1.Nikus K, Pahlm O, Wagner G, Birnbaum Y, Cinca J, Clemmensen P, et al. J Electrocardiographic classification of acute coronary syndromes: a review by a committee of the International Society for Holter and Non-Invasive Electrocardiology.Electrocardiol. 2010 Mar-Apr;43:91-103.

2.Kukla P, Jastrzebski M, Sacha J, Bryniarski L. Lambda-like ST segment elevation in acute myocardial infarction - a new risk marker for ventricular fibrillation? Three case reports. Kardiol Pol. 2008 Aug;66(8):873-7;

3.Capranzano P, Mehran R, Tamburino C, Stone GW, Dangas G. Clinical Impact of Enhanced Inhibition of P2Y 12-Mediated Platelet Aggregation in Patients with ST-Segment Elevation Myocardial Infarction Undergoing Percutaneous Coronary Intervention. Hosp Pract (Minneap). 2010 Nov;38:38-43.

4.Shah RV, Januzzi JL Jr. Images in cardiovascular medicine. ST-elevation alternans and nonsustained polymorphic ventricular tachycardia in a patient with Prinzmetal (variant) angina.Circulation. 2010 Mar 23;121:1371-1373.

5. ST elevation during exercise treadmill testing:
Suggests extremely tight coronary artery stenosis or spasm[1] (transmural ischemia)
ST elevation in the lead aVR during exercise treadmill testing may indicate left main coronary artery disease[2].

6. Tako-Tsubo Cardiomyopathy (TTC)[3]

7. Kounis syndrome [4]. associated with the Greek physician Nicholas Kounis, is defined as "the concurrence of ACS with conditions associated with mast cell activation, involving interrelated and interacting inflammatory cells, and including allergic or hypersensitivity and anaphylactic or anaphylactoid insults." "It is caused by inflammatory mediators such as histamine, neutral proteases, arachidonic acid products, platelet activating factor and a variety of cytokines and chemokines released during the activation process.

1.Norgaz T, Gorgulu S. ST elevation on the exercise ECG: only severe stenosis? Heart. 2010 Jun;96(12):995; author reply 995-6.
2.Ozmen N, Yiginer O, Uz O, Kardesoglu E, Aparci M, Isilak Z, et al. ST elevation in the lead aVR during exercise treadmill testing may indicate left main coronary artery disease. Kardiol Pol. 2010 Oct;68:1107-1111.
3.Bielecka-Dabrowa A, Mikhailidis DP, Hannam S, Rysz J, Michalska M, Akashi YJ,et al. Takotsubo cardiomyopathy--the current state of knowledge.Int J Cardiol. 2010 Jul 9;142:120-125.
4.Biteker M. Current understanding of Kounis syndrome. Expert Rev Clin Immunol. 2010 Sep;6:777-788.

8. **Persistent ST-segment elevation after anterior myocardial infarction consequence of left ventricular aneurysm development**[1]
9. **Massive Pulmonary Embolism**[2]
10. **Severe hypothermia (Osborn wave**[3]**)**
11. **Idiopathic Ventricular Fibrillation or atypical BrS: Lambda wave**[4]
12. **Brugada syndrome**[5]
    12. **Brugada phenocopyes, Brugada-like patterns, or acquired Brugada syndrome**[6]
13. **Congenital Short QT associated with BrS**[5]**. Overlap? Or BrS type 4?**[7]
14. **Hypertrophic cardiomyopathy (HCM).** the ECG finding of convex ST-segment elevation and abnormal Q waves could be valuable for detection of disease progression in patients with HCM[8].

1.Napodano M, Tarantini G, Ramondo A, Cacciavillani L, Corbetti F, Marra MP, et al. Myocardial abnormalities underlying persistent ST-segment elevation after anterior myocardial infarction. J Cardiovasc Med (Hagerstown). 2009 Jan;10:44-50.

2.Lin JF, Li YC, Yang PL. A case of massive pulmonary embolism with ST elevation in leads V1-4.Circ J. 2009 Jun;73:1157-9.

3.Pavlidis AN, Giannakopoulos A, Manolis AJ. Iatrogenic giant Osborn waves. Circulation. 2010 Oct 12;122(15):1519.

4.Gussak I, Bjerregaard P, Kostis J. Electrocardiographic "lambda" wave and primary idiopathic cardiac asystole: a new clinical syndrome?J Electrocardiol. 2004 Apr;37:105-107.

5.Wilde AA, Antzelevitch C, Borggrefe M, Brugada J, Brugada R, Brugada P, et al. ; Study Group on the Molecular Basis of Arrhythmias of the European Society of Cardiology. Proposed diagnostic criteria for the Brugada syndrome. Eur Heart J 2002;23:1648-1654.

6.Riera AR, Uchida AH, Schapachnik E, Dubner S, Filho CF, Ferreira C.Propofol infusion syndrome and Brugada syndrome electrocardiographic phenocopy.Cardiol J. 2010;17(2):130-5.

7.Antzelevitch C, Pollevick GD, Cordeiro JM, Casis O, Sanguinetti MC, Aizawa Y, Loss-of-function mutations in the cardiac calcium channel underlie a new clinical entity characterized by ST-segment elevation, short QT intervals, and sudden cardiac death..Circulation. 2007 Jan 30;115(4):442-9.

8.Furuki M, Kawai H, Onishi T, Hirata K. Value of convex-type ST-segment elevation and abnormal Q waves for electrocardiographic-based identification of left ventricular remodeling in hypertrophic cardiomyopathy. Kobe J Med Sci. 2009 Jun 5;55:E16-29.

Hypothermic J wave

Non-hypothermic J wave: The J wave syndrome is characterized by a prominent J wave accompanied by ST-segment elevation in the absence of structural heart disease. It includes the benign early repolarization pattern, the highly arrhythmogenic BrS and IVF.

Classical Early repolarization Pattern (ERP) or Early Repolarization Variant (ERV) or type 1, which displays an ERP predominantly in the lateral precordial leads, is prevalent among healthy male athletes and is rarely seen in VF survivors.

Early repolarization syndrome as a new electrical disorder associated with SCD this can include the type 2, which displays an ERP predominantly in the inferior or inferolateral leads, is associated with a higher level of risk; and type 3, which displays an ERP globally in the inferior, lateral, and right precordial leads, is associated with the highest level of risk for development of malignant arrhythmias and is often associated with VF storms[1].

Early repolarization in short QT syndrome[2].

Severe hypercalcemia: Electrocardiographically, relatively short QT interval (mean QTc of 340 ms) , ST segment is depressed, and T wave becomes negative in hypercalcemia. In severe cases is possible to observe rarely a J wave and Brugada type1 phenotype or mimicking acute myocardial infarction with prominent J-waves in the inferior leads[3;4].

1. **Antzelevitch C, Yan GX. J wave syndromes. Heart Rhythm. 2010 Apr;7: 549-58**
2. **Zeb M, McKenzie DB, Naheed B, Gazis T, Morgan JM, Staniforth AD. Hypercalcaemia and a Brugada-like ECG: An independent risk factor for fatal arrhythmias. Resuscitation. 2010 Aug;81:1048-1050.**
3. **.Watanabe H, Makiyama T, Koyama T, Kannankeril PJ, Seto S, Okamura K, et al. High prevalence of early repolarization in short QT syndrome. Heart Rhythm. 2010 May; 7: 647-652.**
4. **Mehta S, Parameswaran AC, Greenspan A, Figueredo VM. Hypercalcemia due to rhabdomyolysis mimicking Brugada syndrome. Pacing Clin Electrophysiol. 2009 Nov;32:e14-25.**

Injuries in the central nervous system: Subarachnoid hemorrhage, post-heart arrest and in cervical sympathetic system dysfunction**.**

Brugada "entities": result of a preferential abbreviation of the right ventricular epicardial action potential, With mutation cases (≈17%): true Brugada disease (autosomal dominant pattern). Sporadic cases (≈63%): Brugada syndrome.

Brugada phenotypes, Brugada phenocopies, Brugada-like ECG pattern or acquired Brugada syndrome: they are those clinico-pharmacological entities or circumstances, where Brugada phenotype or sign in ECG, may be found as a consequence of causing increase in Ito channel function in the ventricular epicardium or decrease of slow calcium channel5. Concealed forms of arrhythmogenic dysplasia of the right ventricle;

Idiopathic ventricular fibrillation related to a prominent J wave in the inferior/lateral leads, variant of BrS or atypical BrS: These forms are distinguished by ECG abnormalities of the J wave, and ST-segment elevation appeared in the inferior and lateral leads.

Early repolarization syndrome as a new electrical disorder associated with SCD: gain-of-function mutation s422l in the KCN8-encoded cardiac ATP-sensitive potassium channel KATP channel kir6.1 as a pathogenic substrate (Haïssaguerre syndrome[1]).

CACNA2D1 as possible novel ERS susceptibility genes[2].

Early repolarization in short QT syndrome. There is a high prevalence of early repolarization in patients with SQTS. Early repolarization may be useful in identifying risk of cardiac events in SQTS[3].

Family IVF with mutation on DPP6 (dipeptidyl-peptidase 6) gene on chromosome 7 has not early repolarization on ECG[4].

Eventually, vagally mediated VF initiated by PVCs arising from the RVOT[5].

1.Miyazaki S, Shah AJ, Haïssaguerre M. 2010 Sep 11.Early repolarization syndrome – a new electrical disorder associated with sudden cardiac death –. Circ J. 2010 Oct;74:2039-2044.

2.Burashnikov E, Pfeiffer R, Barajas-Martinez H, Delpón E, Hu D, Desai M, et al. Mutations in the cardiac L-type calcium channel associated with inherited J-wave syndromes and sudden cardiac death. Heart Rhythm. 2010 Oct 13.

3. Watanabe H, Makiyama T, Koyama T, Kannankeril PJ, Seto S, Okamura K, et al. High prevalence of early repolarization in short QT syndrome. Heart Rhythm. 2010 May;7:647-652.

4.Alders M, Koopmann TT Christiaans I, Postema PG, Beekman L, Tanck MW.Haplotype-sharing analysis implicates chromosome 7q36 harboring DPP6 in familial idiopathic ventricular fibrillation. Am J Hum Genet. 2009 Apr;84:468-476.

5. Kataoka M, Takatsuki S, Tanimoto K, Akaishi M, Ogawa S, Mitamura HA case of vagally mediated idiopathic ventricular fibrillation. Nat Clin Pract Cardiovasc Med. 2008 Feb;5:111-115.

ST SEGMENT ELEVATION CONCAVE UPWARD

Causes

1) Vagotonia

2) Afro-descendents

3) Early Repolarization Variant (ERV) or pattern (ERP)

4) Juvenile pattern

5) Asthenic habitus

6) Athlete heart

7) Hyperacute phase of myocardial infarction

8) Acute phase of pericarditis(The present case)

9) Artifact caused by excessive inertia of the needle of the device

10) Saddle types 2 or 3 ECG Brugada pattern

II) Dialysis-associated pericarditis **— Pericarditis also occurs in approximately 13 percent of patients on maintenance hemodialysis[1], and may occasionally be seen with chronic peritoneal dialysis. At least two factors may contribute to this problem: inadequate dialysis (ie, the patient has uremic pericarditis) and/or fluid overload[2].**

It has been suggested that the two forms of uremic pericarditis can be distinguished by the type (serous versus hemorrhagic) of effusion that is present, but there is significant overlap. Pathologic examination of the pericardium typically shows adhesions between the pericardial membranes, which are thicker than normal. Loculated, bloody fluid when present, is due in part to the frequent impairment in platelet function in renal failure and the use of anticoagulation during hemodialysis. The clinical features of pericarditis in renal failure are similar to those observed with other causes. Most patients complain of fever and pleuritic chest pain, the intensity of which is quite variable[3].The pain is characteristically worse in the recumbent position. A pericardial rub is generally audible, but is frequently transient. Signs of cardiac tamponade may be seen, particularly in patients with rapid pericardial fluid accumulation. However, the high prevalence of autonomic impairment in this patient population may hinder the normally observed rise in heart rate[4]. Moreover, some patients with uremic pericarditis present without symptoms or suggestive findings (chest pain or pericardial rub) on physical examination[4]. Cardiac ultrasonography reveals a pericardial effusion in at least 50 percent of cases. A concomitant pleural effusion, which is commonly exudative, may be observed, findings consistent with generalized serositis[5].

1. Rutsky EA, Rostand SGTreatment of uremic pericarditis and pericardial effusion.Am J Kidney Dis. 1987;10:2-8.
2. Lundin, AP. Recurrent uremic pericarditis: A marker of inadequate dialysis. Semin Dial 1990; 3:5.
3. Alpert MA, Ravenscraft MD SOPericardial involvement in end-stage renal disease.Am J Med Sci. 2003;325:228-236.
4. Gunukula SR, Spodick DHPericardial disease in renal patients. Semin Nephrol. 2001;21: 52-56.
5. Bakirci T, Sasak G, Ozturk S, Akcay S, Sezer S, Haberal M. Pleural effusion in long-term hemodialysis patients.Transplant Proc. 2007;39:889-891.

In an observational study, Tseng et al[1] investigated whether the improvement rate for dialysis-associated pericarditis following the administration of aggressive dialysis differs between patients on dialysis who have diabetes and those who do not. The study employed data from 88 maintenance hemodialysis patients (47 with diabetes and 41 without diabetes) with dialysis-associated.

The authors found that following the intensification of hemodialysis, pericarditis improved in 85.1% of patients with diabetes and in 82.9% of those without diabetes. Among patients with diabetes, 85.1% survived without recurrence of pericarditis, 4.3% survived but did suffer recurrence, and 10.6% died, with similar outcomes recorded in the group without diabetes (87.8%, 4.9%, and 7.3%, respectively). The authors concluded that in patients with or without diabetes, intensive dialysis is the most effective treatment for dialysis-associated pericarditis.

1. **Tseng JR, Lee MJ, Yen KC, et al. Course and outcome of dialysis pericarditis in diabetic patients treated with maintenance hemodialysis. *Kidney Blood Press Res*. 2009;32:17-23.**