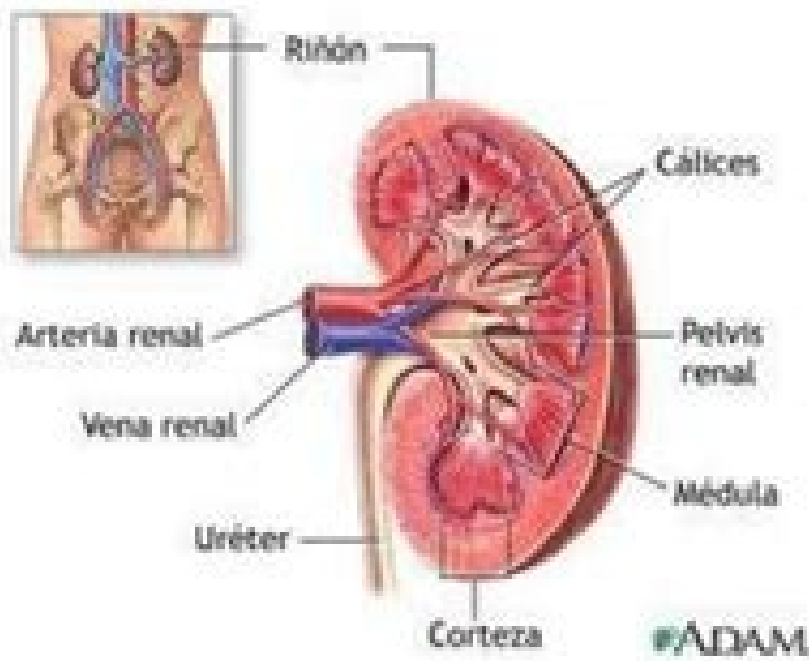
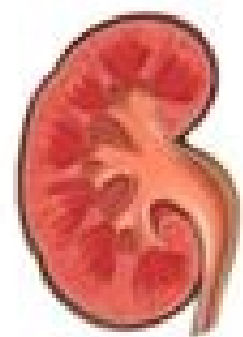


I'm not robot!

FISIOPATOLOGÍA

- IRC tiende a progresar a uremia terminal en un tiempo prolongado, aunque no persista la causa de nefropatía inicial.
- 2 Mecanismos básicos:
 - Lesiones estructurales residuales producidas por la enfermedad causal.
 - «Teoría de la hiperfiltración»



Cuadro N°3. Indices urinarios en IRA prerrenal y NTA.

	Personal	NTA
Desnsidad urinaria	>1020	<1010
Osmolalidad	>500	<350
U/P osmolalidad	>1.3	<1.1
Sodio urinario	<20	>40
U/P creatinina	>40	<20
U/P úrea	>10	<3
Indice de insuficiencia renal	<1	>1
fracción excretada de sodio	<1	>1

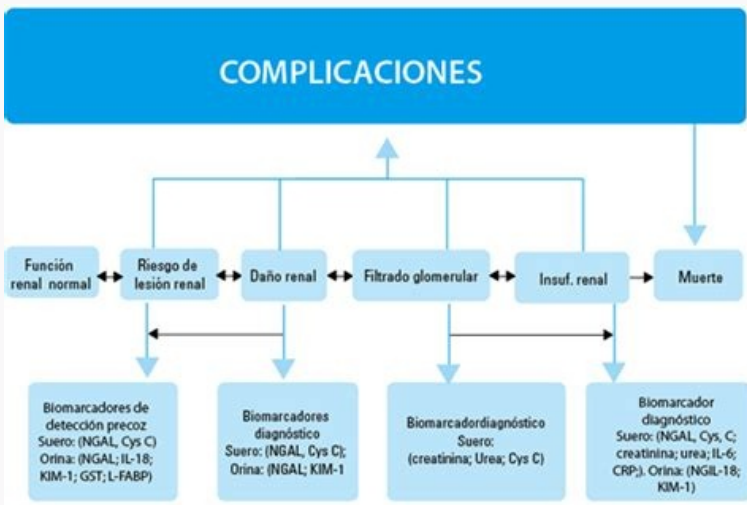


TABLA 4			
Tratamiento para la prevención de recurrencias			
	Litiasis cálcicas	Litiasis ácido úrico ^b	Litiasis cistina
Aumento de la ingesta hídrica	Si (grado de recomendación A)	Poco importante	Si
Si se observa hipercalcúria (mujeres > 300 mg/día, hombres > 250 mg/día)	• Restringir proteínas, oxalato y sodio (grado de recomendación C) • Hidroclorotiazida 25 mg/12 h (grado de recomendación B) • Citrato potásico^a (grado de recomendación B) • No restringir la ingesta de calcio		
Si se observa hipocitratúria (< 320 mg/día)	• Restringir proteínas y sodio (grado de recomendación C) • Citrato potásico (grado de recomendación B)		
Si se observa hiperuricosuria (mujeres > 750 mg/día, hombres > 800 mg/día)	• Restringir purinas (grado de recomendación C) • Alopurinol 100-300 mg/día (grado de recomendación B) (No tiene efecto en la recurrencia de litiasis sintomáticas)^c	• Restringir proteínas y sodio (grado de recomendación C) • Alopurinol (grado de recomendación C)	
Si se observa pH urinario ácido		• Restringir proteínas y sodio (grado de recomendación C) • Citrato potásico (grado de recomendación C)	• Citrato potásico (grado de recomendación C)

^a Citrato potásico (Acalta®) 1-2 comprimidos/8 horas, Uralyt® 1 cucharada/8-12 horas). Efectos adversos gastrointestinales frecuentes; precaución en la insuficiencia renal crónica; riesgo de hipopotasemia.

^b Litiasis ácido úrico: más relacionadas con pH urinarios bajos que con hiperuricosuria (alcalinizar la orina con citrato potásico).

^c Las litiasis de estruvita requieren la extracción completa del cálculo, la mayoría de las veces con nefrolitotomía percutánea, y tratar las posibles infecciones de orina.

Tabla 3. Indicadores de Riesgo Cardiometabólico en adolescentes con y sin exceso de peso, según presencia de CHT.

Indicador	Normopeso			Sobrepeso/Obesidad		
	CHT(-)*	CHT(+)**	p	CHT(-)***	CHT(+)*****	p
Presión sistólica (mm Hg)	102.2±10.1	115.0±5	0.020	110±7.6	120.0±6.3	0.008
Presión diastólica (mm Hg)	64.1±7.2	57±6.1	0.107	64.9±7.0	74±5.8	0.013
Glicemia (mg/dL)	84.9±5.6	88.3±2.9	0.252	86.3±7.4	83.3±2.1	0.241
CT (mg/dL)	136.2±26.9	152.3±31.3	0.280	142.1±30.3	172.5±33.9	0.031
LDLc (mg/dL)	90.8±23.5	93.1±29.2	0.796	97.8±27.6	110.1±32.5	0.932
HDLc (mg/dL)	33.7±6.5	35±3.6	0.487	32.0±5.4	31.5±7.3	0.372
TGL (mg/dL)	58.6±24.1	121±7	<0.001	62.7±23.2	154.3±45.4	<0.001
Col no-HDL (mg/dL)	102.5±24.3	117.3±27.9	0.309	110.1±29.5	141.0±31.4	0.015
Relación CT/HDLc	4.1±0.8	4.3±0.5	0.411	4.5±1.1	5.6±1.2	0.045
Relación LDLc/HDLc	2.7±0.7	2.6±0.5	0.703	3.1±1	3.6±1.1	0.348
Relación TGL/HDLc	1.8±0.9	3.5±0.5	0.002	2±0.8	5.3±2.4	<0.001
Insulina (mU/mL)	10.8±4.8	15±3.6	0.123	14.6±6.3	17.8±5.6	0.241
Índice HOMA-IR	2.3±1	3.3±0.8	0.127	3.1±1.3	3.6±1.2	0.324
IRCMa	-1.39±2.41	3.34±1.16	0.001	2.56±2.48	7.79±2.86	0.001

Resultados expresados como media aritmética±desviación estándar. *n=59; **n=3; ***n=27; ****n=6. U de Mann-Whitney según presencia de cintura hipertriglicéridémica en cada grupo estudiado. CHT: circunferencia hipertriglicéridémica; CT: colesterol total; LDLc: colesterol unido a la lipoproteína de baja densidad; HDLc: colesterol unido a la lipoproteína de alta densidad; TGL: triglicéridos; Col no-HDL: colesterol no HDL; HOMA-IR: homeostasis model assessment insulin resistance; IRCMa: índice de riesgo cardiometabólico agrupado.

INTRODUCTION Acute renal failure (RAF) is a clinical syndrome, secondary to multiple etiologies, which is characterized by a sudden deterioration of the renal function, whose common expression is an increase in the concentration of nitrogenated products in blood, with/without decreased urinary volume. First of all, deterioration of the renal function, we must be able to distinguish from what type of kidney failure we find ourselves, and at the same time try to discover its etiology. To do this, we will rely on three basic actions:1. The realization of a detailed medical history that includes anamnesis of personal history.2. A thorough physical exploration.3. The use of the various complementary diagnostic tests in a phased manner depending on their effectiveness and safety for the sick. It is important to remember that acute renal failure on multiple occasions is multifactorial, especially the one that develops in the hospital. And, moreover, it is a dynamic process and can evolve from one stage to another more serious. We must address the differential diagnosis of the FRA from four points of view (Table 1):1. Syndrome diagnosis, acute or chronic deterior?2. Functional diagnosis, how much has the kidney function deteriorated?3. Physiopathological diagnosis, prerenal, parenchymatous or obstructive?4. Etiological diagnosis, what causes it? SINDROMIC DIAGNOSTICOAn important aspect in the evaluation of the patient with kidney disease is to know the duration of the disease, so that knowing it, the differential diagnosis can be limited frequently. Usually, when the elevation of nitrogenous products or alterations in urinary parameters develop within hours or consecutive days before a sharp process; if the evidence of kidney disease extends to several weeks represents a rÃ¡pidamente rÃ¡pidamente progresivo (subagudo) y, por Ã ltimo, cuando es conocido desde meses o aÃ±Aos llevarÃ¡ un curso crÃ¡nico, pero que puede asociarse con exacerbaciones agudas.La etiologÃ¡a del FRA es muy amplia y debemos considerar que su distribuciÃ³n puede variar con la regiÃ³n geogrÃ¡fica e incluso con el tipo de hospital. En el estudio de Madrid (GEFRAM), en el queA se evaluaron 748 casos de FRA en 13 hospitales, las causas mÃ¡s frecuentes fueron: necrosis tubular aguda en el 45%, prerenal en el 21%, enfermedad crÃ¡nica agudizada en el 13% (principalmente por necrosis tubular aguda o enfermedad prerenal) yÃ obstructiva en el 10% de los casos.2. En otro estudio, realizado en 5 unidades de cuidados intensivos en Estados Unidos: Program to Improve Care in Acute Renal Disease (PICARD), incluyen a 618 pacientes, siendo las principales causas de FRA: necrosis tubular aguda (isquÃ©mica y tÃ¡xica), en torno al 70%, prerenal en el 16%, y la patologÃ¡a obstructiva representÃ¡ apenas el 1% de los casos.3. Se han realizado pocos estudios sobre la etiologÃ¡a del FRA en pacientes con enfermedad renal crÃ¡nica. En un estudio retrospectivo, realizado sobre 104 casos de enfermedad renal crÃ¡nica en China, las causas del FRA se atribuyeron a necrosis tubular aguda/nefritis intersticial por fÃ¡rmacos en el 36% de los casos y a enfermedad prerenal en el 24%. La necesidad de diÃ¡lisis, el antecedente de hipertensiÃ³n y los niveles mÃ¡s elevados de creatinina sÃ©rica fueron factores predictores independientes de escasa recuperaciÃ³n renal.4. Por todo ello, ante un paciente con elevaciÃ³n de los productos nitrogenados en sangre, la primera pregunta que debemos plantearnos es si nos encontramos ante un caso deÃ insuficiencia renal crÃ¡nica o de FRA. La distinciÃ³n entre estas dos situaciones, en ocasiones, Ã resulta difÃcil. Inicialmente, deberemos averiguar la posible existencia de controles analÃticos previos de la funciÃ³n renal obtenidos rutines, or by previous pathological processes, which allow us to know if the current deterioration is acute or chronic, as well as to know personal or family history of kidney disease or other systemic diseases with frequent kidney involvement (e.g., diabetes). On the other hand, periodic controls will be very orient: in cases of FRA the daily increase of serum creatinine should be greater than 0.3 mg/dl/day, while in chronic kidney failure the values of creatinine will continue to be constant. In the absence of previous data from renal function, the existence of symptoms such as anorexia, astenia, cramps, nausea, morning vomiting, polyuria, nicturia, etc., of long evolution, oriented towards a chronic process. The urochrome dye practically discards the FRA. Well-tolerated anemia, hypocalcemia, hyperphosphoemia and metabolic acidosis not justified by other reasons usually a chronic process, although it should not be forgotten that these analytical alterations can also be seen in a patient with FRA.5. With all previous considerations, the distinction between acute or chronic kidney disease can remain uncertain. We need the support of other complementary tests, as well as the observation of the patient's clinical evolution. PHASES OF RENAL FRACASE ACUIDOThe most separate form of FRA is acute tubular necrosis (NTA) and is characterized by the sublethal and lethal lesion of the tubular cells, mainly in the distal portions of the proximal tubulum and in the thick ascending portion of the Henle asa. In the physiopathology of acute renal ischemia, hemodynamic factors, tubular lesions and inflammatory processes are involved. Basically, the ischemic FRA has been divided into three phases: initiation, maintenance and recovery. Recently Sutton et al. have established aof vital importance in the physiopathology of the FRA.6. This recognizes five physiopathological stages in the course of the NTA (Figure 1):1. Hemodynamic or toxic aggression (prerenal, English). We can consider that it forms a continuum with the initiation phase. It occurs when the renal blood flow decreases, but the cellular integrity is maintained.2 Start phase. It appears when the renal blood flow causes ATP depletion. The lesion of the epithelial tubular cell (loss of microvilli, exfoliation, etc.) 3. Extension phase. It is characterized by the persistence of hypoxia and the inflammatory response, both more pronounced events in the corticomedular union. It is at this stage that the dysfunction of the endothelial cell plays a fundamental role: alteration of permeability, procoagulant state, alteration in the regulation of pro-inflammatory cells, liberation of cytokines, etc. Cell death occurs: necrosis and apoptosis.4 Maintenance phase. In it, cells begin to repair: dedifference, migration, apoptosis, proliferation in an attempt to maintain cellular and tubular integrity.5 Recovery phase. Cellular differentiation is maintained and epithelial polarity is restored. FUNCIONAL DIAGNOSTICOThere is no universal definition of acute kidney failure. In general, all definitions of kidney failure emphasize the immediate character of renal functional impairment and the importance of abrupt decline in glomerular filtration and/or elevation of nitrogenated products in blood, as a universal marker of acute renal failure independent of its etiology.7 The knowledge that kidney function requires three premises: proper blood perfusion, the integrity of the renal parenchyma and the permeability of the pathwaysIt allows us to classify acute kidney failure, depending on the altered functional element, as a prerenal if what fails is renal perfusis; parenchymatous, if the alteration occurs in renal structures, and obstructive or postrenal if the urinary flow is obstructed. This classification has been used universally in the last 50 years and is still in force today, and it has also served as a basis for most epidemiological studies carried out in this period.8 From an operational point of view, each author uses a particular definition to identify the group of patients that includes in his study (instrumental definition)7. These definitions vary from considering the absolute value of sane creatinine, the increase in it, the determination of glomerular filtration or the volume of diuresis. On occasion only particular situations are studied, such as patients admitted to the ICU or only those treated with dialectical techniques.In view of this disparity of criteria, different study groups such as Acute Dialysis Quality Initiative (ADQI) and working networks such as Acute Kidney Injury Network (AKIN) have been formed in the last day to develop clinical recommendations. They recognize the need to unify the definition of the FRA, but to date they have only agreed different functional classifications. Due to its universality, low cost and frequency of use, the determination of creatinine has been the path used for clinical practice in the diagnosis and treatment of acute kidney failure. However, it suffers from several inconveniences: it rises when the glomerular filtration has descended in half; its concentration is very influenced by changes in muscle mass, by the increase in tubular secretion.presence of functional impairment and numerous extrarenal factors such as body weight, race, age, sex, etc. In short, the glomerular filtration by creatinine clarification does not appear to be an exact tool in unstable metabolic situations such as those that accompany acute kidney failure. However, the data of Herrera et al., which use creatinine clarifications done in 2 hours in chronic patients, is simple and can be useful.9 The related inconveniences associated with the use of creatinine may condition the delay in the early diagnosis of the acute renal alteration, so that when it is particularly detected, it is in a particularly serious stage of damage. Trying to solve this situation, thanks to the technological, protein, generic and a new physiopathological consideration established by Sutton et al., the interest in the development and use of new biomarkers with greater sensitivity, specificity, prognostic capacity, location of the daÃ±o produced and monitoring of the response to the treatment has been triggered in the last year. In short, we must find one or more substances that in the FRA area comply with the definition of biomarker: "a characteristic capable of being measured objectively and evaluated as an indicator of normal biological processes, or of pharmacological responses to a treatment."10. The ideal characteristics of a FRA marker are summarized in table 2.11. Advanced in this approach, several authors have proposed the development of phase biomarkers, which conceptually refer to substances of a tissue that are released at a specific time with respect to the time in which harm is produced.12.13. In this way, we can distinguish four types of biomarkers depending on the phaseThe is detected: 1. Phase 1 or pre -in -art: the marker will serve to identify subjects at risk. 2. Phase 2 or early harm: the marker will serve to identify the appearance of the lesion. 3. Phase 3 or LesiÃ³n late: It will serve to monitor the evolution of the lesion and its repair (3rd (3rd) and to evaluate the function (3b) .4. Phase 4 or recovery: the marker will be used for pronostical purposes. 2002.14. Rifle is the acridemate of the English words corresponding to risk (risk), daÃ±a ± o and irreversible of renal function (end). Therefore, it includes three stages of renal lesion and increasing severity (risk-injury-failure) and two of clogeric pronists Acute are percentage descents of glomerular filtrate, relative elevations of the symore creatinine with respect to basal value and the decrease in diuresis (Table 3). It takes into account the presence of prior chronic renal insufficiency, as well as those patients with symore creatinine values> 4 mg/dl are considered in stage F, provided that the increase has been at least 0.0 . 5 mg/dl. To classify a patient, the worst criterion (creatinine, glomerular filtering or diuresis) should be used, which site it in a greater stadium of gravity. The time permit, considered to evaluate the changes is 7 days. This classification has proven to be a boy to diagnose acute renal failure and classify patients according to their functional severity, but also also © n has demonstrated its correlation as a pronostical marker. In a systematic review, which included a more than 71,000 patients, 13 studies have been found that 13 studies have been found the mortality of patients without and with FRA estimated by the RIFLE system. Mortality was 6.95 and 31.2%, respectively. When they analyze the relative risk of patients who did not develop FRA, it increased with the severity of the RIFLE scale (R = 2.40; I = 4.15; F = 6.15). Similar results have been published in Spain (R = 2.77; I = 3.7; F = 3.52)16. AKINES classification a modification of the RIFLE system, proposed by the Agute Kidney injury Network17. It is based on the emergence of new epidemiological data that show an increase of 80% in the risk of mortality with changes so minimal in the concentration of serum creatinine as of 0.3 to 0.5 mg/dl.18. Instead of using the letters of an acronym they employ a system of functional stadiums identified by numbers that correspond to the first three stages of the RIFLE (Table 4). Therefore, there are only two differences between both systems: the AKIN classification includes in its small stage increases in serum creatinine (0.3 mg/dl) and changes observed in renal function should occur in 48 hours, add two premises:1. Diagnostic criteria should only be applied after optimizing the patient's volatile state.2. When we only consider oliguria as a diagnostic criterion, the existence of obstructive uropathy must be discarded. Creatinine kineticsThe use of percentage changes in creatinine in the diagnosis of acute kidney failure has recently been questioned. The inverse relationship between glomerular and creatinine filtration is known, and the elevation of the second can be estimated depending on the descent of the first, being independent of the basal renal function of which it is split. Using a mathematical model of creatinine kinetics, Waikar and Bonventre demonstrate that the absolute changes of creatinine are capableDetect any decrease in the glomerular filtering rate before the percentage changes, especially in patients with prior chrhenal renal disease19. Provide a classification that also contemplates three stages (Table 5) based on absolute changes. Ã ¢ strongly remembered (0.3, 0.5, 1 and 1.5 mg/dl) in two defined time intervals (24 and 48 hours). Each stage corresponds to an approximate decrease in creatinine clearance: Stage 1, 19-33%; Stage 2, 30-57%; Stage 3, 49-68%. BiomarCadores:creatinina es an inhibitor of proteases, constant by all the c nucleated lulas. It filters freely in the glomÃ¡f © rulo, it is completely reabsorbed into the proximal bullshit and is not secreted. Cistatin levels are not modified by age, sex, race or muscle mass. It is a better predictor of glomerular function than creatinine sÃ¡f © in patients with chronic renal disease. Some authors find that an increase of 50% in cystatin c sÃ¡f © rica predicts 1-2 days before the appearance of acute renal failure that the elevation of creatinine of the creatinine only as associated associated With neutritide lipocaline (N-Gal) it is a 25 kd protection known as siderocaline or lipocaline 2. It usually expresses low levels in several human tissues such as riÃ¡f ± ageÃ³n, lung fÃ¡Mago and colon. It belongs to the family of lipocalins. In general they are protection of small size that have the ability to join small moisturizers and transport them to the interior of the CÃ¡f © lula.22. 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