XVII Reunión
Insuficiencia Cardiaca
y Fibrilación Auricular

DEBATE ABIERTO

FORTALECIENDO ALIANZAS CON LA SEC: NUEVOS DOCUMENTOS DE CONSENSO

Diagnóstico y tratamiento del déficit de Fe en la IC

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Servicio de Cardiología. Hospital Universitario Ramón y Cajal de Madrid

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FORTALECIENDO ALIANZAS CON LA SEC: NUEVOS DOCUMENTOS DE CONSENSO

Diagnóstico del déficit de hierro en la insuficiencia cardiaca

Dr. Juan Roberto Peraira Moral
Servicio de Cardiología. Hospital Universitario Ramón y Cajal de Madrid


Ferritina
Hepcidina
Saturación de transferrina
The Twilight Zone
Receptor soluble de transferrina
Amplitud de distribución eritrocitaria
Déficit de hierro
Anemia

ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure 2012

11.14 Iron deficiency

Iron deficiency may contribute to muscle dysfunction in HF and causes anaemia. In a single RCT, 459 patients with NYHA class II or III systolic HF, a haemoglobin concentration between 9.5 and 13.5 g/dL, and iron deficiency (see below) were randomized 2:1 to i.v. ferric carboxymaltose or saline. In this trial, iron deficiency was diagnosed when serum ferritin was <100 μ/L or when the ferritin concentration was between 100 and 299 μg/L and transferrin saturation was <20%.[208] Over 6 months of treatment, iron therapy improved self-reported patient global assessment and NYHA class (as well as 6-min walk distance and health-related quality of life) and may be considered as a treatment for these patients. The effect of treating iron deficiency in HF-PEF and the long-term safety of iron therapy in HF is unknown.

McMurray JJV. Eur Heart J 2012;33:1787-1847

ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure 2012

11.2 Anaemia

Anaemia (defined as a haemoglobin concentration <13 g/dL in men and <12 g/dL in women) is common in HF, particularly in hospitalized patients. It is more frequent in women, the elderly, and in patients with renal impairment. Anaemia is associated with more symptoms, worse functional status, greater risk of HF hospitalization, and reduced survival. A standard diagnostic work-up should be undertaken in anaemic patients. Correctable causes should be treated in the usual way, although no definite aetiology is identified in many patients. Correction of iron deficiency using i.v. iron has been specifically studied in patients with HF (see Section 11.14). The value of erythropoietin-stimulating agents as a treatment for anaemia of unknown aetiology is unknown but is currently being tested in a large mortality–morbidity RCT.[187]

McMurray JJV. Eur Heart J 2012;33:1787-1847

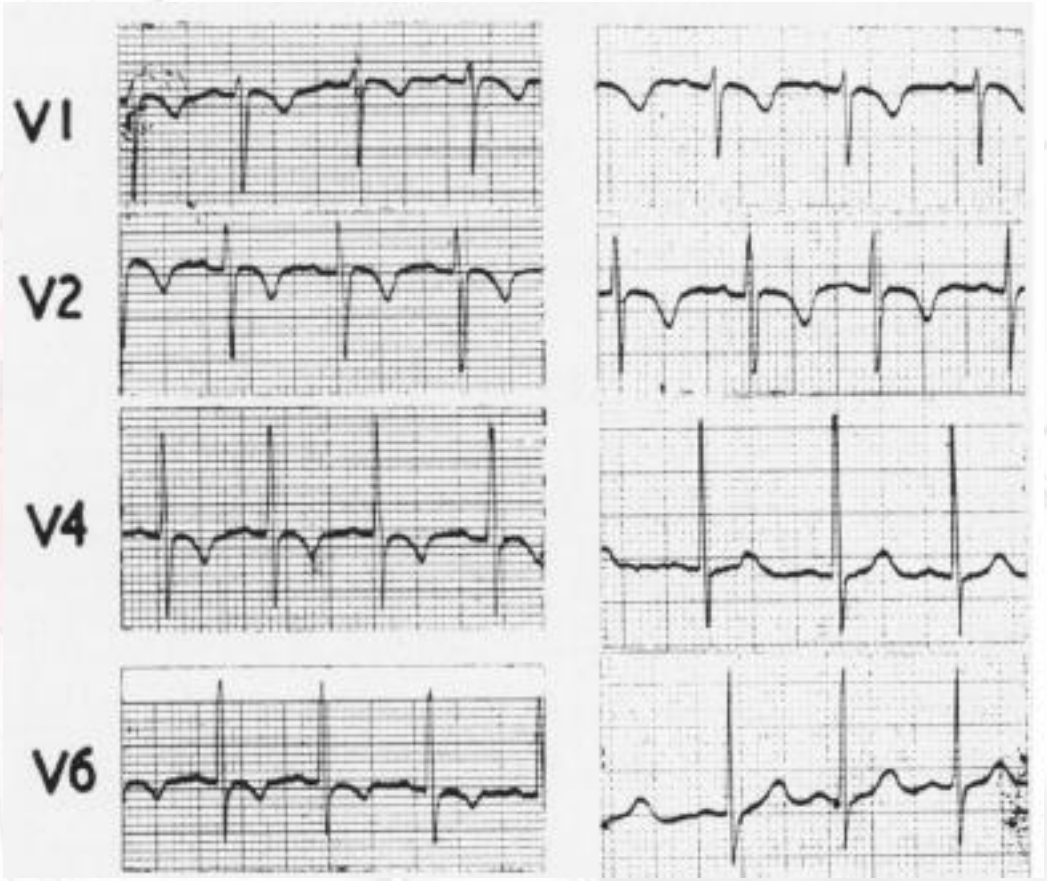
Acute Reversible Heart Failure in Severe Iron-Deficiency Anemia Associated with Hookworm Infestation in Uganda Africans

By K. Somers, M.B., B.Ch. (W'Rand), M.R.C.P., D.C.H.

- 15 pacientes (10 hombres, 5 mujeres) ingresados por IC aguda
- Edad media: 26.3 años (5-50 años)
- Hb media: 2.7 g/dl (1.1-4.6 g/dl)
- Tratamiento:
  - Hierro oral e intramuscular
  - Transfusiones
  - Digoxina
  - Dieta baja en sal
  - Tetracloroetileno
- 5 éxitus (AP: dilatación VI, edema pulmonar)

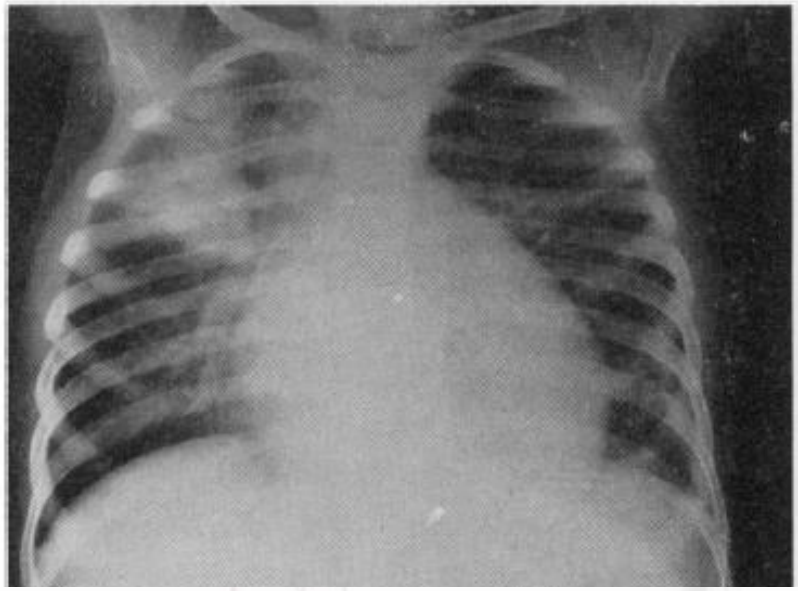
Somers K. Circulation 1959;19: 672-675

Antes del tratamiento **Después del tratamiento**

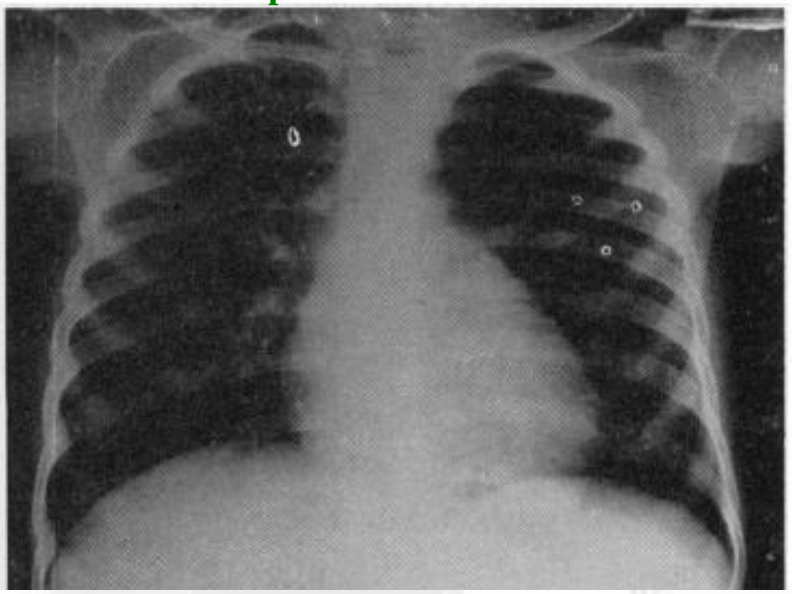


Somers K. Circulation 1959;19: 672-675

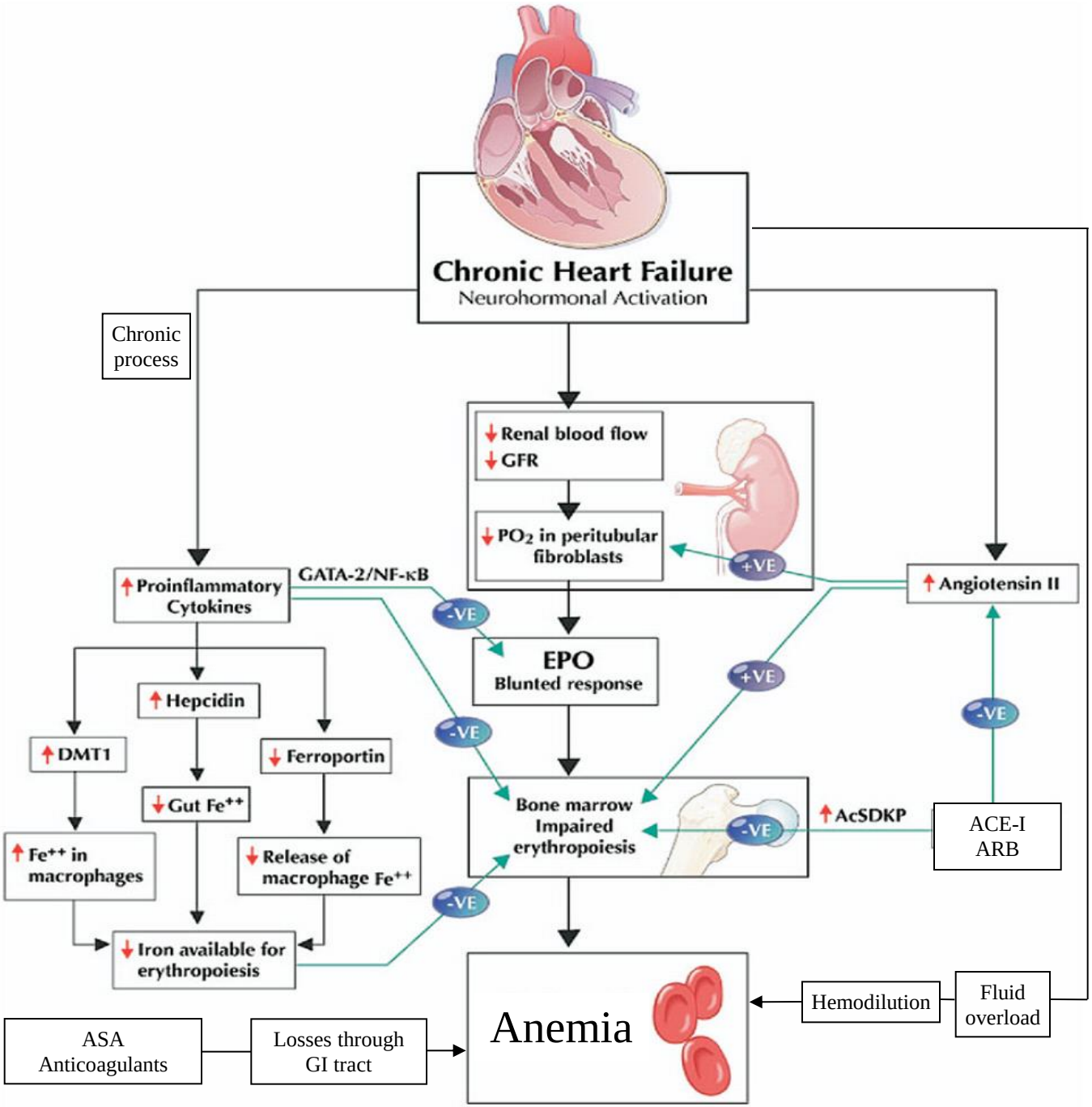
Antes del tratamiento



Después del tratamiento

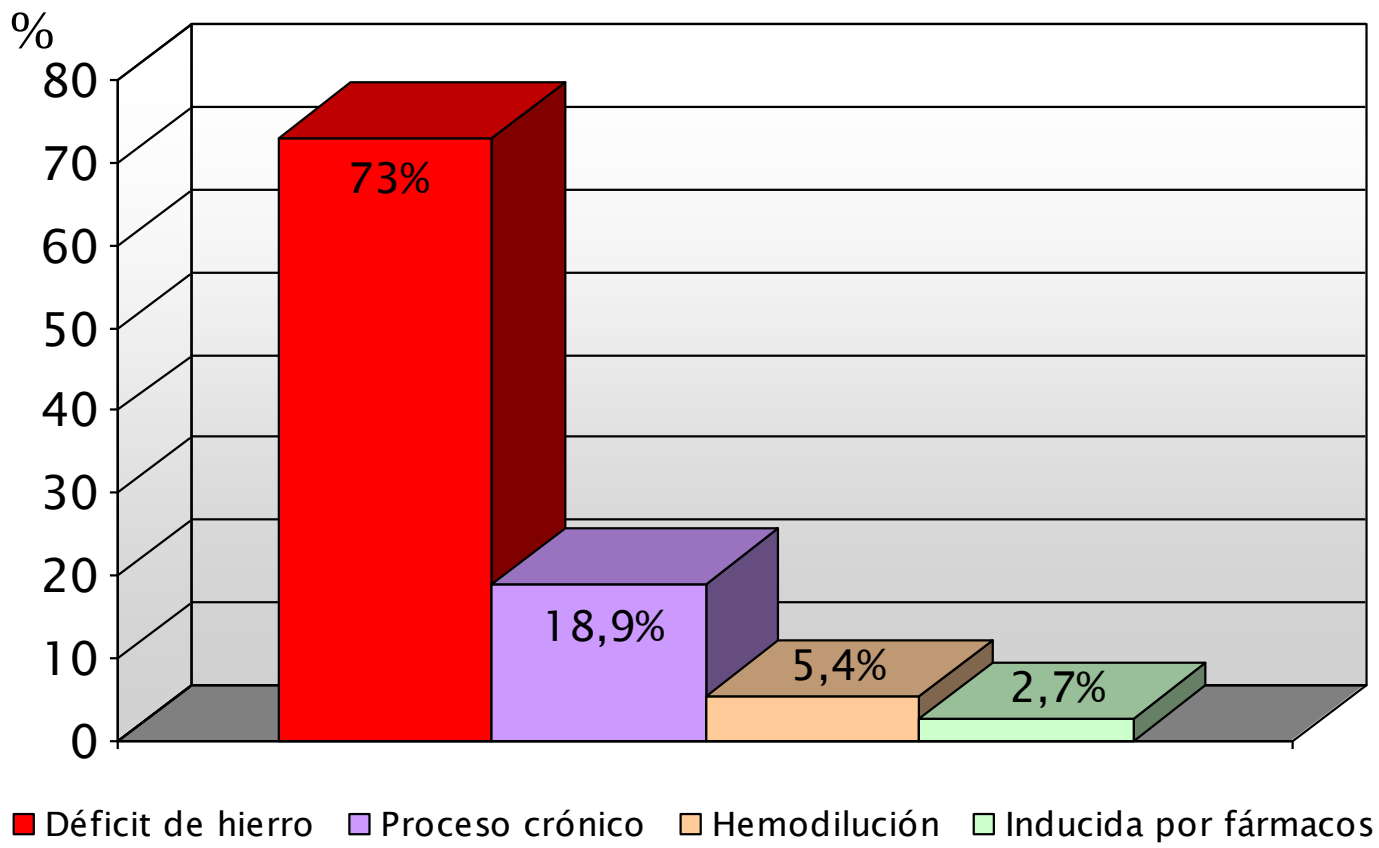


Somers K. Circulation 1959;19: 672-675

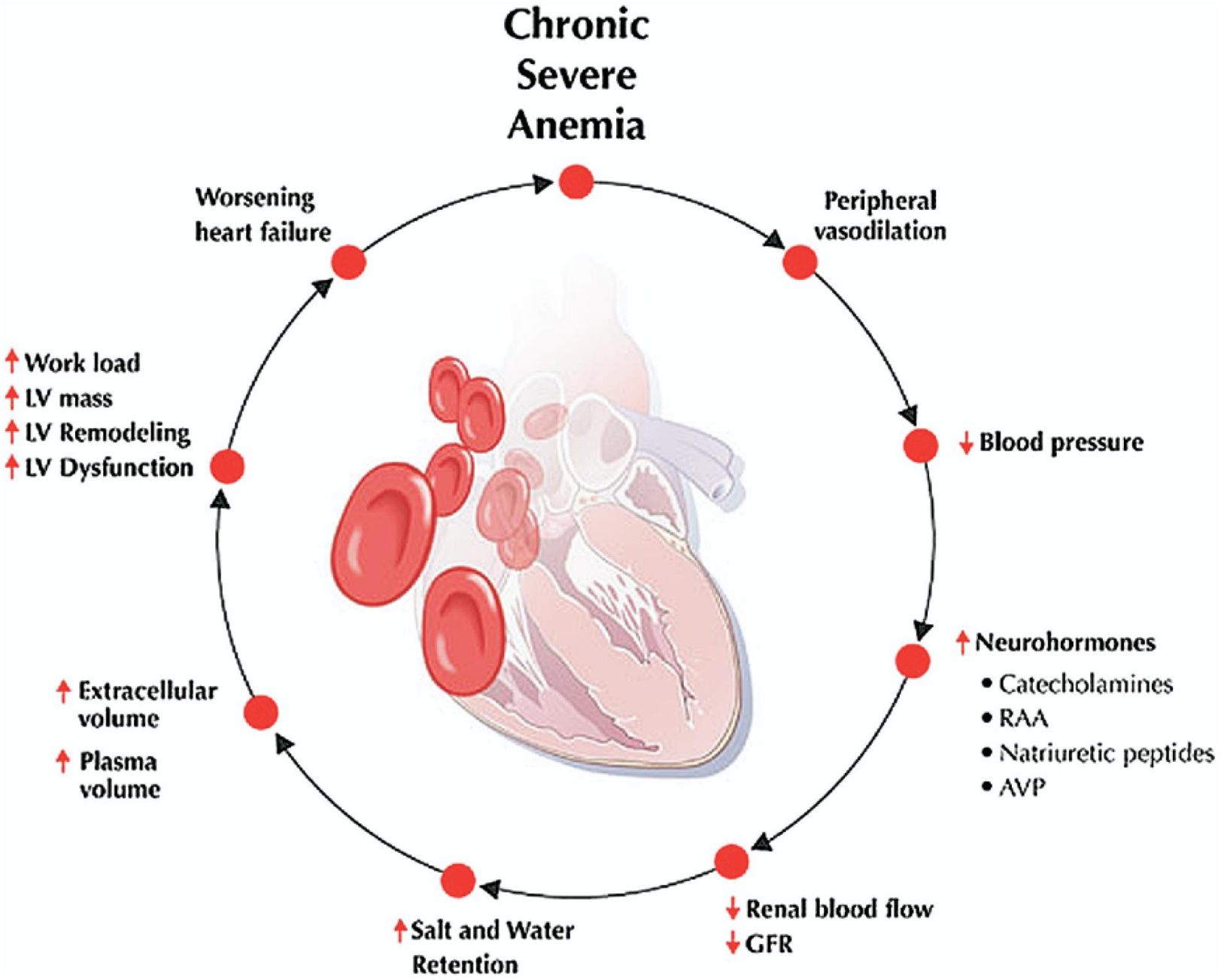


Anand IS. J Am Coll Cardiol 2008; 52: 501-511

Etiología de la anemia en la IC crónica



Nanas JN. J Am Coll Cardiol 2006; 48: 2485-2489



Anand IS. J Am Coll Cardiol 2008; 52: 501-511

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Heart Failure

Anemia and Mortality in Heart Failure Patients

A Systematic Review and Meta-Analysis

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Jan van Wijngaarden, MD, PhD,‡ Hans L. Hillege, MD, PhD,*
Dirk J. van Veldhuisen, MD, PhD, FACC,* Peter van der Meer, MD, PhD†

Groningen and Deventer, the Netherlands; and Boston, Massachusetts

Objectives The aim of this study was to assess the effect of anemia on mortality in chronic heart failure (CHF).

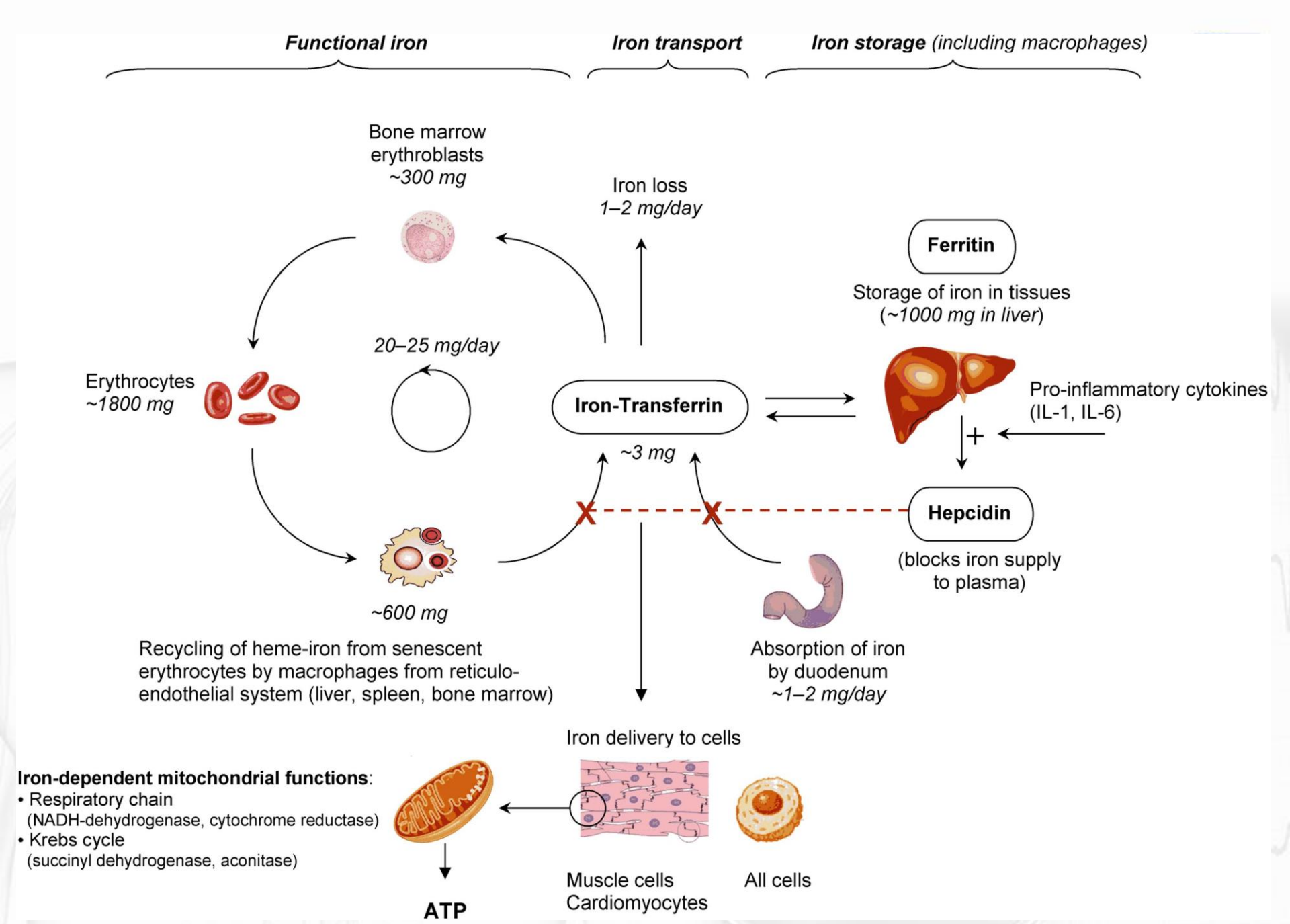
Background Anemia is frequently observed in patients with CHF, and evidence suggests that anemia might be associated with an increased mortality.

Methods A systematic literature search in MEDLINE (through November 2007) for English language articles was performed. In addition, a manual search was performed. We included cohort studies and retrospective secondary analyses of randomized controlled trials whose primary objective was to analyze the association between anemia and mortality in CHF. Of a total of 1,327 initial studies, we included 34 studies, comprising 153,180 patients. Information on study design, patient characteristics, outcome, and potential confounders were extracted.

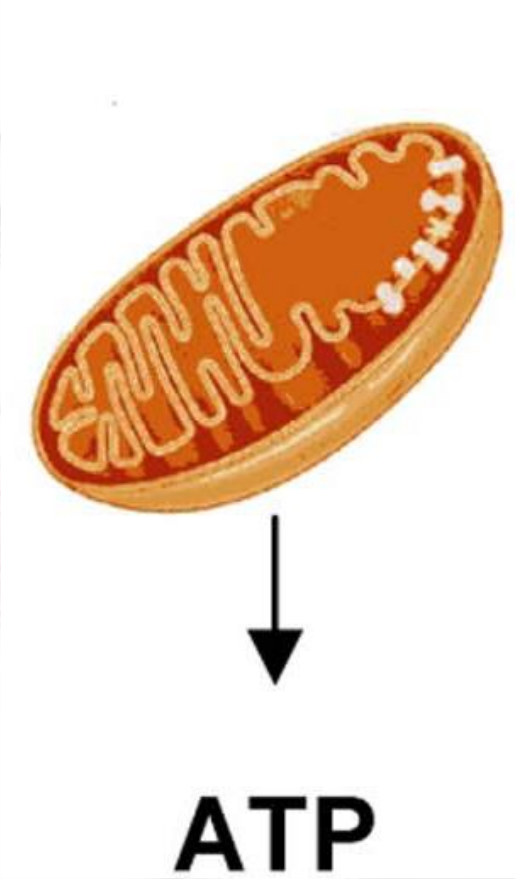
Results Anemia was defined by criteria used in the original articles. Of the 153,180 CHF patients, 37.2% were anemic. After a minimal follow-up of 6 months, 46.8% of anemic patients died compared with 29.5% of nonanemic patients. Crude mortality risk of anemia was odds ratio 1.96 (95% confidence interval: 1.74 to 2.21, $p < 0.001$). Lower baseline hemoglobin values were associated with increased crude mortality rates ($r = -0.396$, $p = 0.025$). Adjusted hazard ratios showed an increased adjusted risk for anemia (hazard ratio 1.46 [95% confidence interval: 1.26 to 1.69, $p < 0.001$]). Subgroup analysis showed no significant difference between mortality risk of anemia in diastolic or systolic CHF.

Conclusions Anemia is associated with an increased risk of mortality in both systolic and diastolic CHF. Anemia should, therefore, be considered as a useful prognosticator, and therapeutic strategies aimed to increase hemoglobin levels in CHF should be investigated. (J Am Coll Cardiol 2008;52:818–27) © 2008 by the American College of Cardiology Foundation

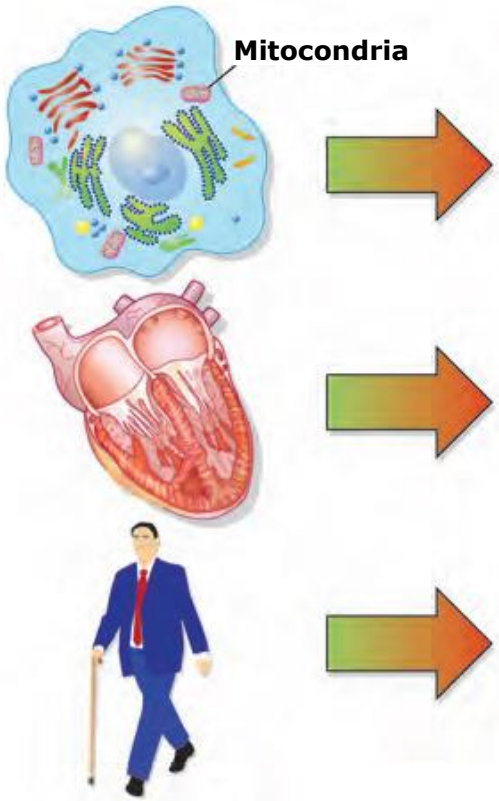
Groenveld HF. J Am Coll Cardiol 2008;52:818-827



Cohen-Solal A. Heart Published Online First 23 June 2014: doi:10.1136/heartjnl-2014-305669


ATP

Consecuencias del déficit de hierro



- Disfunción mitocondrial
- Metabolismo anaeróbico – ineficiencia energética
- Disfunción enzimática
- Alteración del transporte y la estructura de proteínas
- Apoptosis

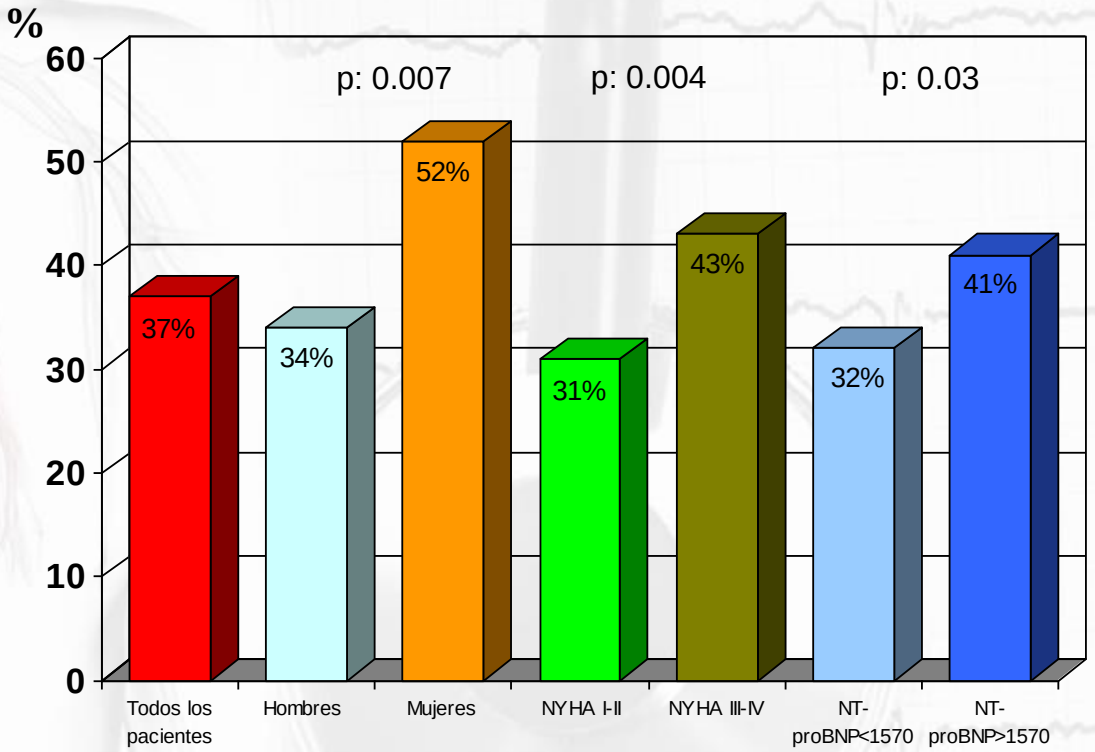
- Anemia
- Remodelado tisular (HVI, remodelado matriz extracelular, dilatación VI)
- Disminución de la eficacia de los órganos

- Alteración de la capacidad de ejercicio
- Disminución de la eficacia del trabajo
- Alteraciones cognitivas y del comportamiento
- Aumento de morbimortalidad

Jankowska EA. Eur Heart J 2013;34: 816-826
Cohen-Solal A. Heart Published Online First 23 June 2014: doi:10.1136/heartjnl-2014-305669

Déficit de hierro e IC crónica

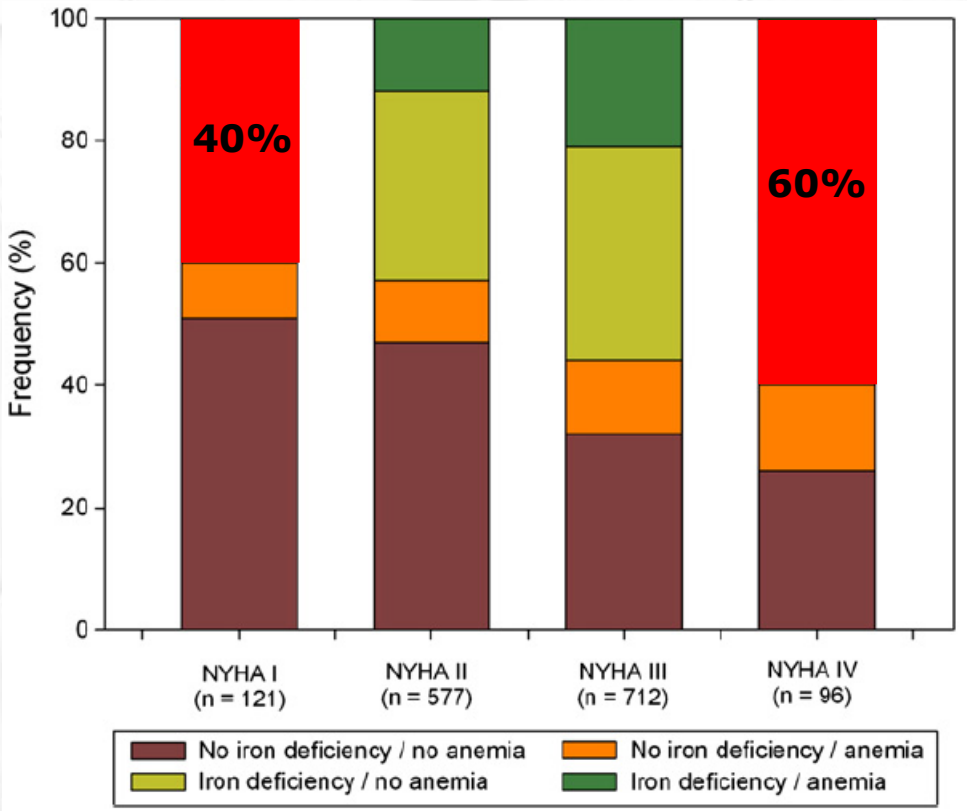
- 546 pacientes (22% mujeres) con IC sistólica estable, 82% clase II-III NYHA
- Edad: 55 ± 11 años
- FEVI: 26 ± 7%
- 67% de causa isquémica
- 27% Diabetes mellitus
- NT-proBNP medio: 1570 pg/ml
- Hb: 14.1 ± 1.7 g/dl
- 95% con IECA/ARA-II
- 98% con β-bloqueante
- 80% con diurético de asa
- 77% con antialdosterónico
- Seguimiento: 731 ± 350 días



Jankowska EA. Eur Heart J 2010;3:1872-1880

Iron deficiency in chronic heart failure: An international pooled analysis

- 1506 pacientes (26% mujeres) con IC crónica (97% ICFED), 93% clase I-III NYHA
- Edad: 64 ± 13 años
- FEVI: 33 ± 14% (13% con FEVI >45%)
- 60% de causa isquémica
- 20% HTA
- 35% Diabetes mellitus
- 20% Fibrilación auricular
- 91% con IECA/ARA-II
- 90% con β-bloqueante
- 79% con diurético de asa
- 48% con antialdosterónico
- Seguimiento: 8 años



Klip IT. Am Heart J 2013;165: 575-582.e3

The additive burden of iron deficiency in the cardiorenal–anaemia axis: scope of a problem and its consequences

- 1506 pacientes (26% mujeres) con IC crónica (97% ICFED), 93% clase I-III NYHA
- Edad: 64 ± 13 años
- FEVI: 33 ± 14% (13% con FEVI >45%)
- 60% de causa isquémica
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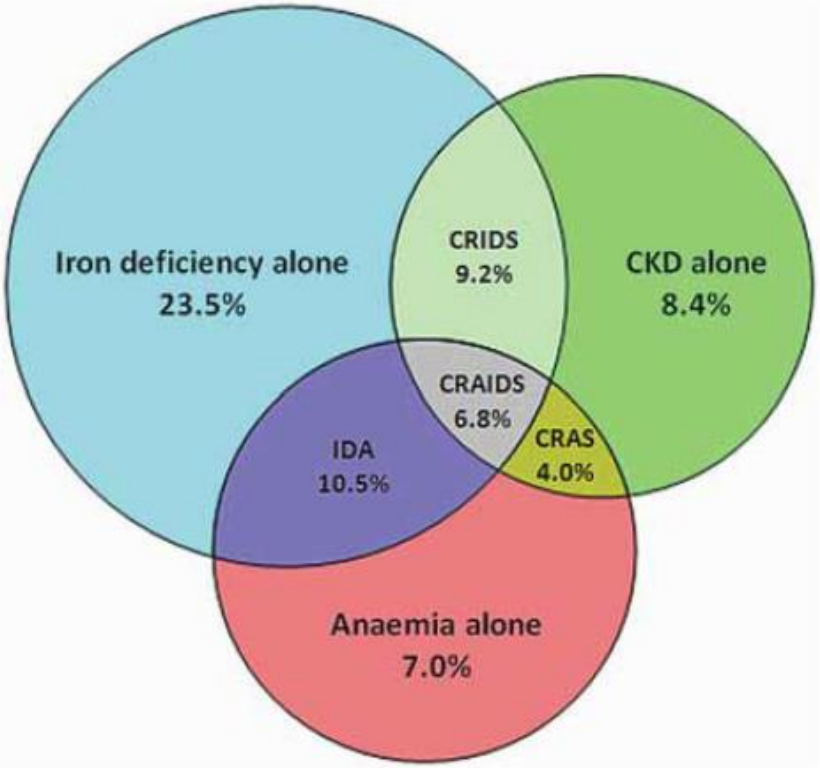
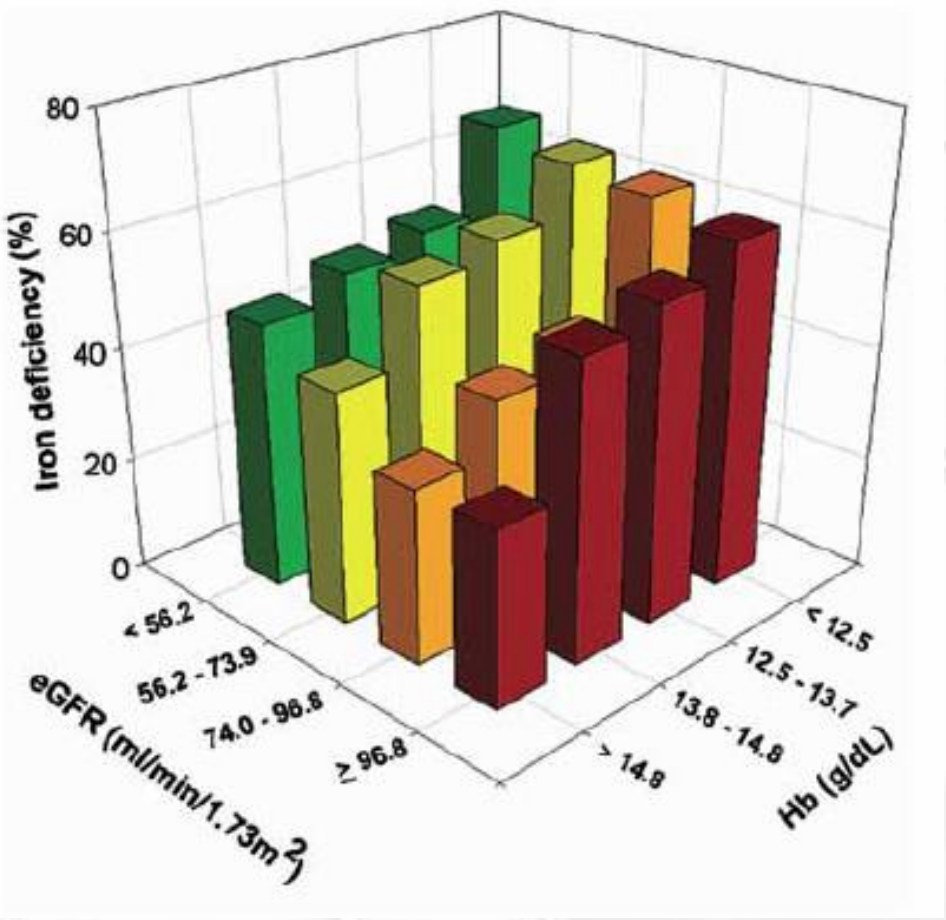
- 79% con diurético de asa
- 48% con antialdosterónico
- Seguimiento: 8 años



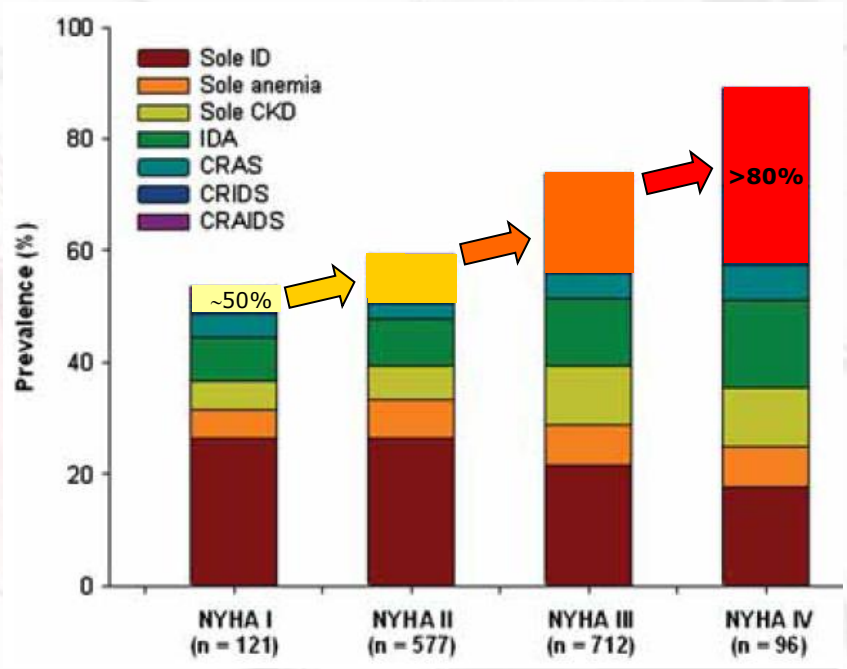
Klip IT. Eur J Hear Fail 2014;16: 655-662

Prevalencia de déficit de hierro según la función renal y la hemoglobina



Klip IT. Eur J Hear Fail 2014;16: 655-662

Prevalencia de déficit de hierro en pacientes con síndrome cardiorrenal



Klip IT. Eur J Hear Fail 2014;16: 655-662

Beyond the cardiorenal anaemia syndrome: recognizing the role of iron deficiency

Rationale for a new terminology

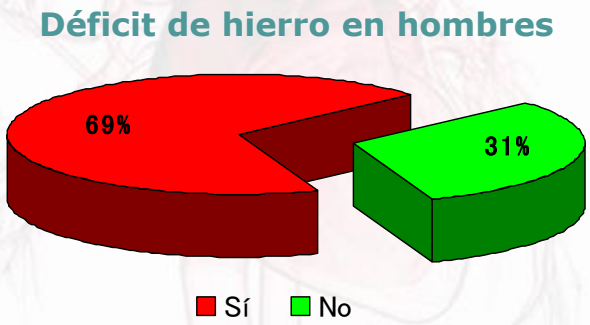
It is important that clinicians understand the interaction of iron deficiency, anaemia, renal dysfunction, and chronic HF, and development of more accurate terminology to describe specific combinations of these adverse phenomena may be one step towards improving awareness. The current term CRAS disregards the potential contribution of iron deficiency, a frequent finding in HF.

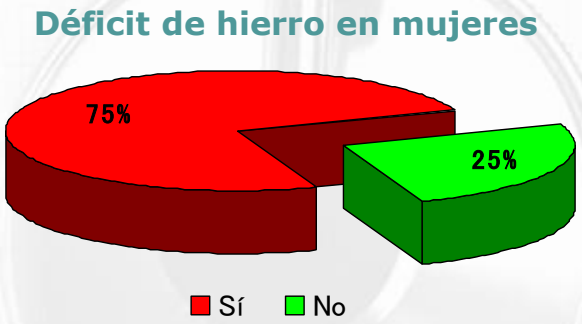
is it time for 'CRAS' to be supplemented with new acronyms such as CRIDS (cardiorenal–iron deficiency syndrome) or even CRAIDS (cardiorenal–anaemia–iron deficiency syndrome) (*Figure 1*) to permit a more subtle characterization of the patient's pathological condition and interventional requirements?

Macdougall IC et al. Eur J Hear Fail 2012;14: 882-88

Prevalencia del déficit de hierro en pacientes ingresados por IC aguda

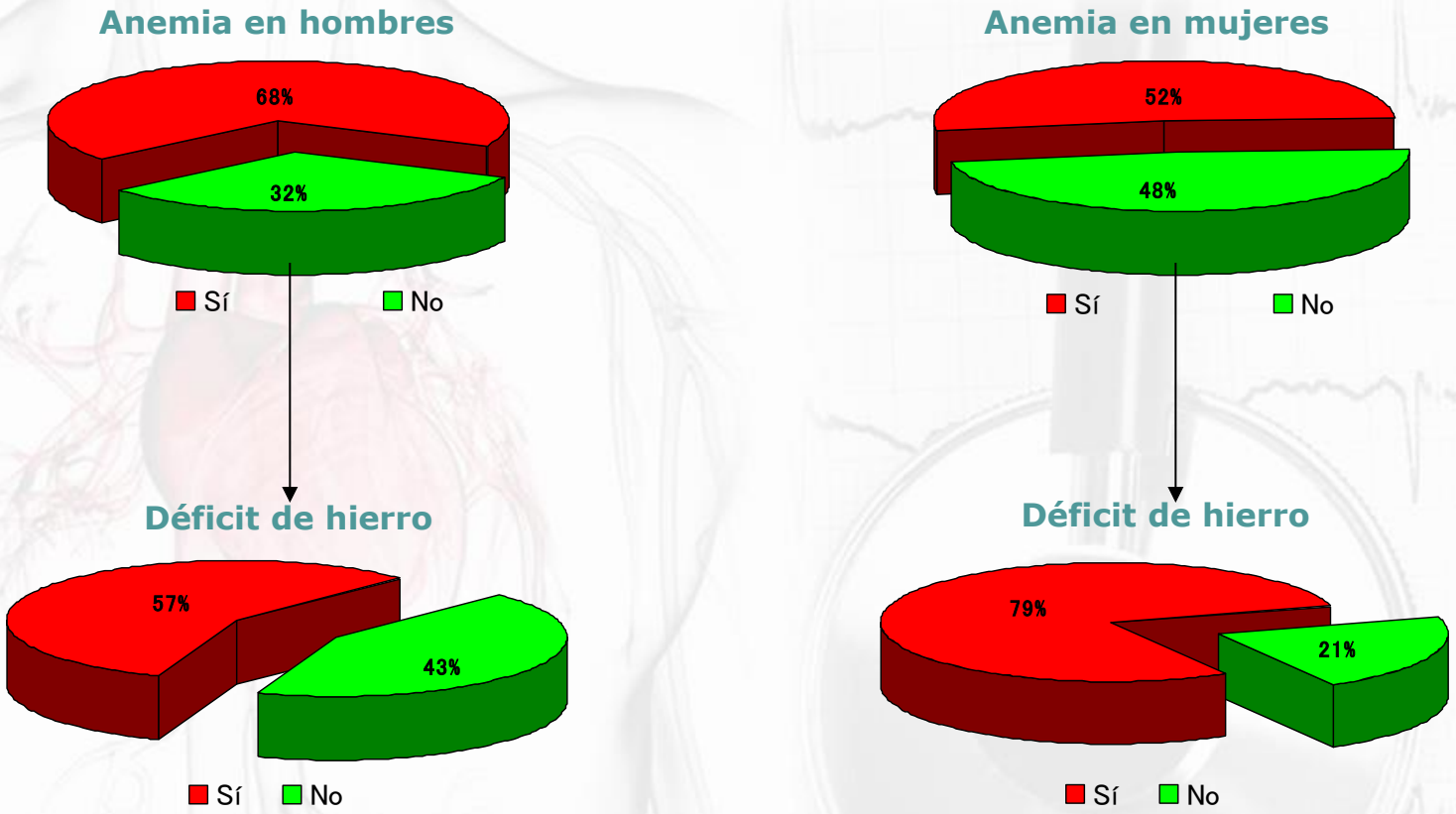
- 832 pacientes (51% mujeres) ingresados por IC descompensada
- Hombres: 75 ± 12 años, 75% clase III/IV NYHA
- Mujeres: 81 ± 11 años, 71% clase III/IV NYHA
- Estudio del déficit de hierro en las primeras 72 h tras ingreso





Cohen-Solal A. Eur J Heart Fail 2014 Jul 28. doi: 10.1002/ejhf.139. [Epub ahead of print]

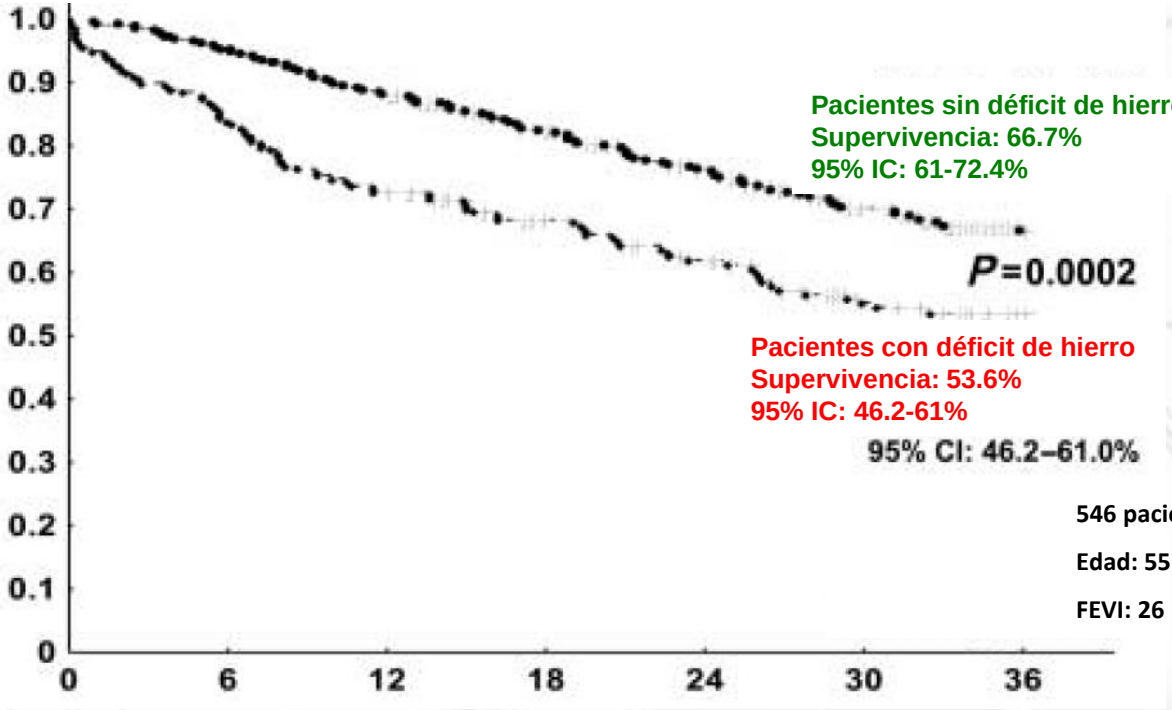
Prevalencia del déficit de hierro en pacientes ingresados por IC aguda



Cohen-Solal A. Eur J Heart Fail 2014 Jul 28. doi: 10.1002/ejhf.139. [Epub ahead of print]

Implicaciones pronósticas del déficit de hierro en la IC crónica

Supervivencia libre de eventos (muerte por cualquier causa / trasplante cardiaco)



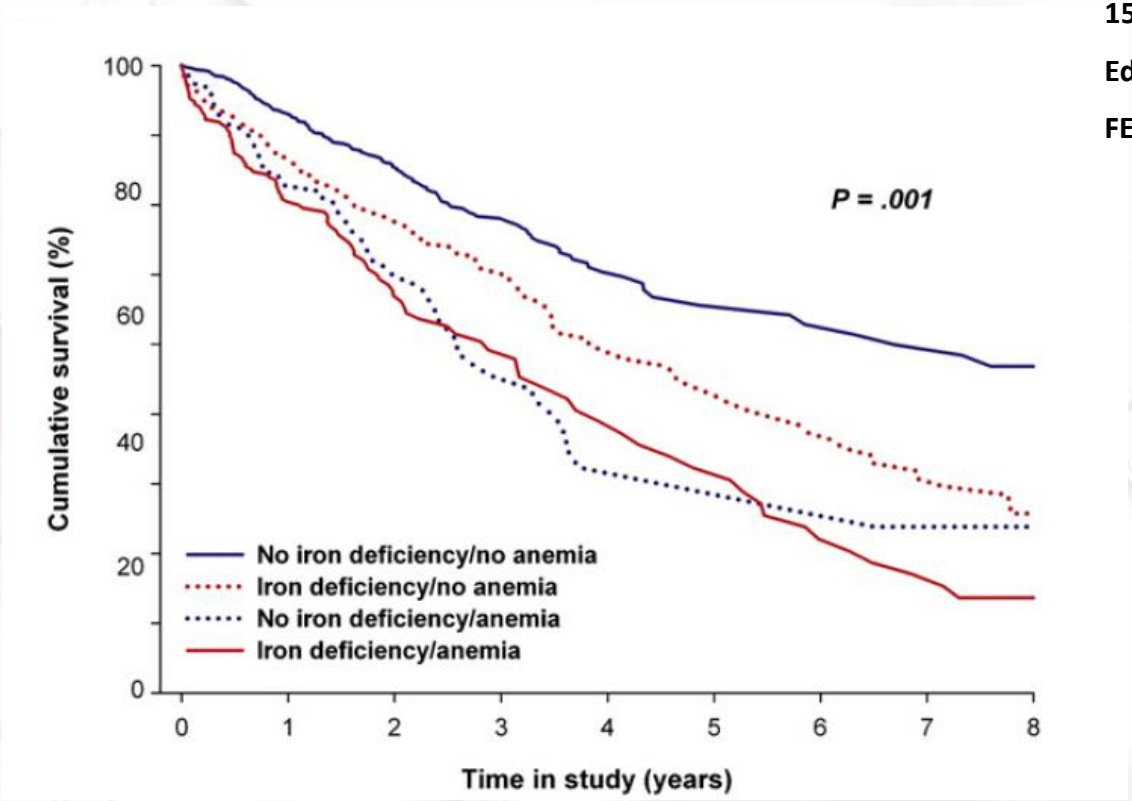
546 pacientes con IC sistólica estable

Edad: 55 ± 11 años

FEVI: 26 ± 7%

Jankowska EA. Eur Heart J 2010;3:1872-1880

Déficit de hierro y mortalidad en IC crónica



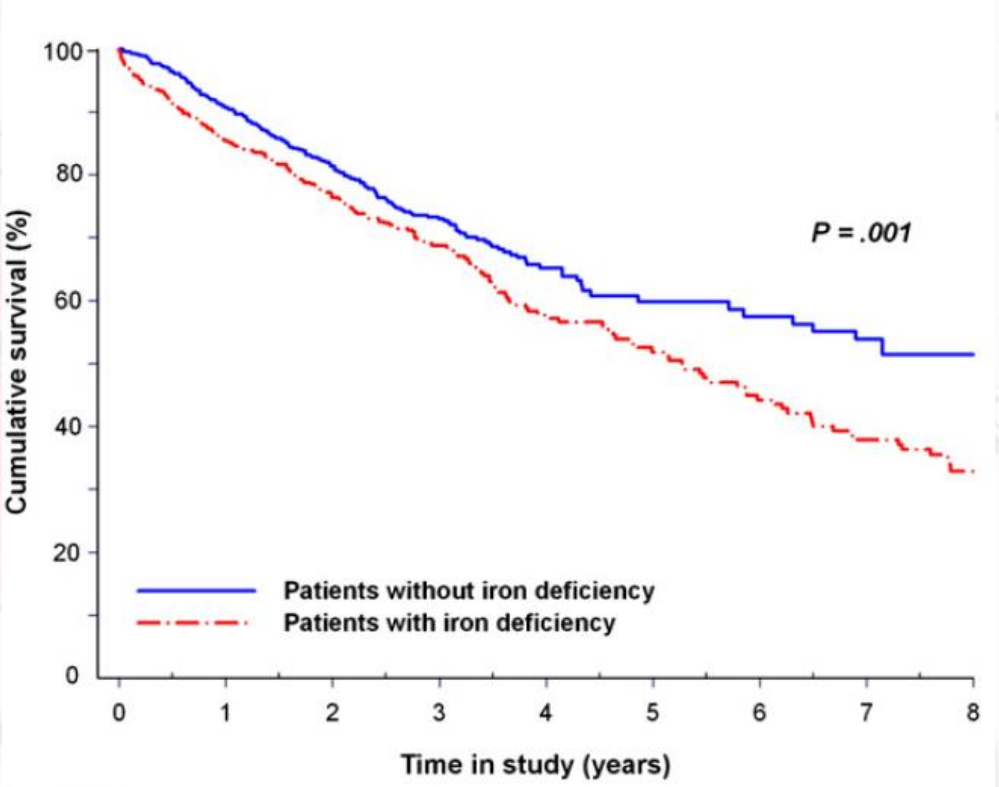
1506 pacientes con IC crónica

Edad: 64 ± 13 años

FEVI: 33 ± 14%

Klip IT. Am Heart J 2013;165: 575-582.e3

Déficit de hierro y mortalidad en IC crónica



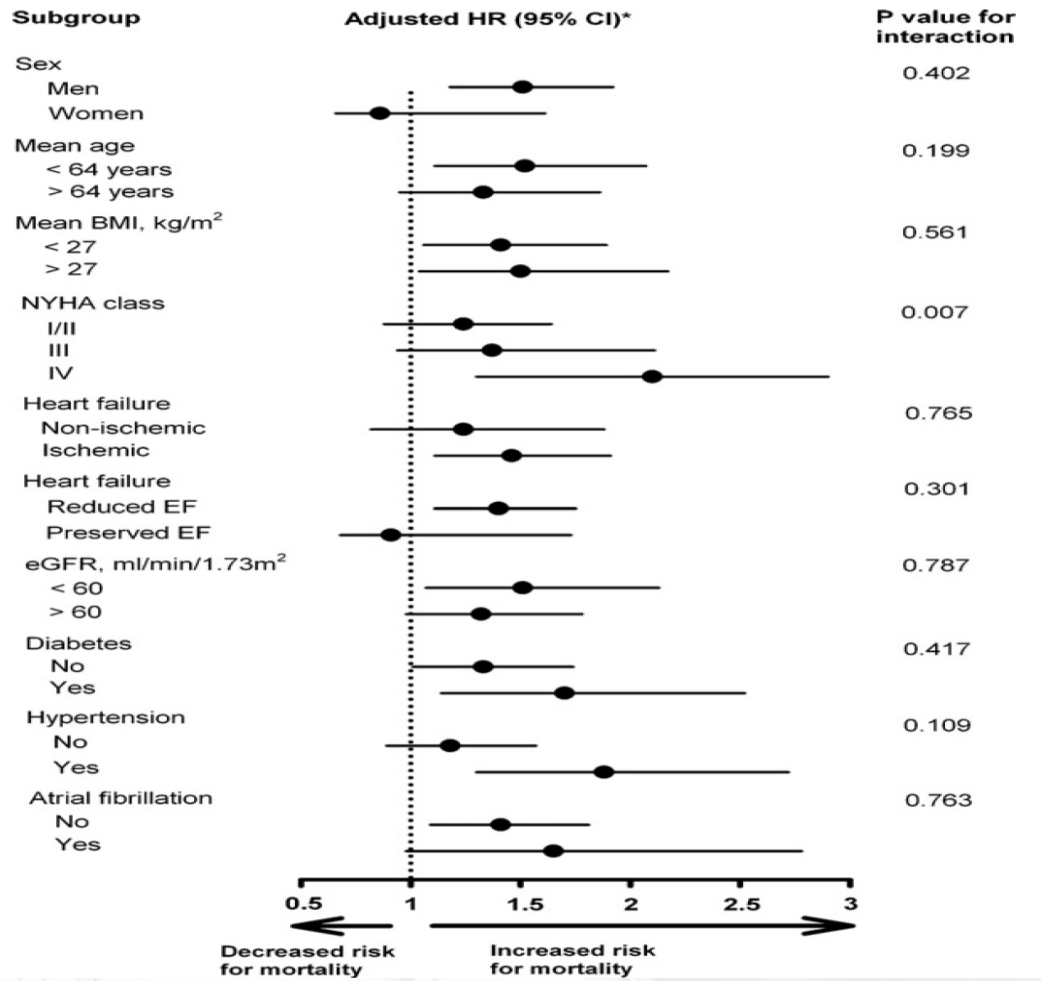
1506 pacientes con IC crónica

Edad: 64 ± 13 años

FEVI: 33 ± 14%

Klip IT. Am Heart J 2013;165: 575-582.e3

Déficit de hierro y mortalidad en IC crónica

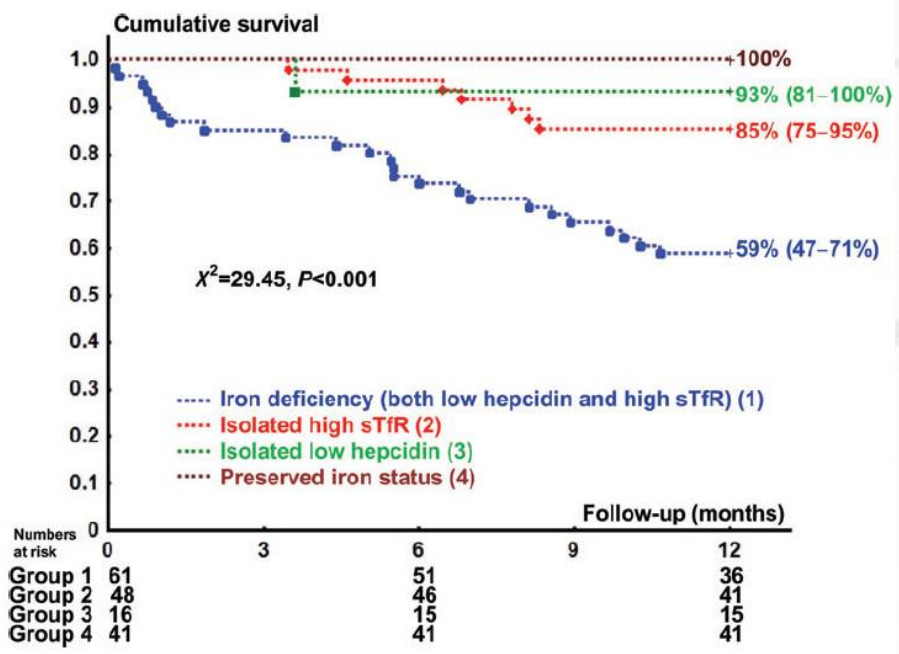


Klip IT. Am Heart J 2013;165: 575-582.e3

Déficit de hierro y pronóstico tras ingreso por IC aguda

- 165 pacientes (19% mujeres) con IC aguda (31% de novo)
- Edad: 65 ± 12 años
- FEVI: 33 ± 13%
- NT-proBNP medio: 4800 pg/ml
- 55% de causa isquémica
- 66% HTA
- 39% Diabetes mellitus
- 51% Fibrilación auricular
- Estudio basal del déficit de hierro
  - ↓ Hepcidina (depósitos deplecionados)
  - ↑ sTfR (déficit de hierro celular)

  } 37% pacientes
- Seguimiento: 1 año



Jankowska EA. Eur Heart J 2014; doi:10.1093/eurheartj/ehu235

Diagnóstico del déficit de hierro en la IC

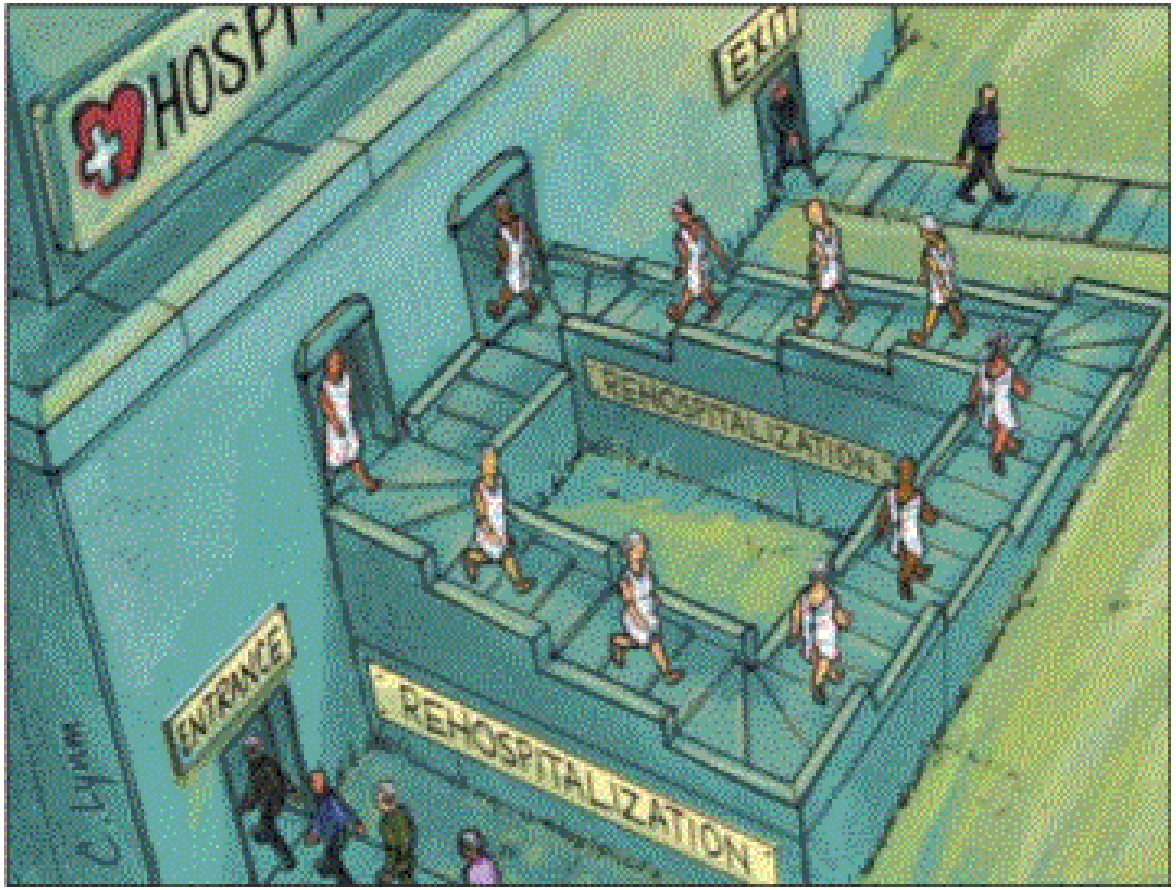
Manifestaciones clínicas del déficit de hierro en la IC

↓ capacidad ejercicio
↑ clase NYHA

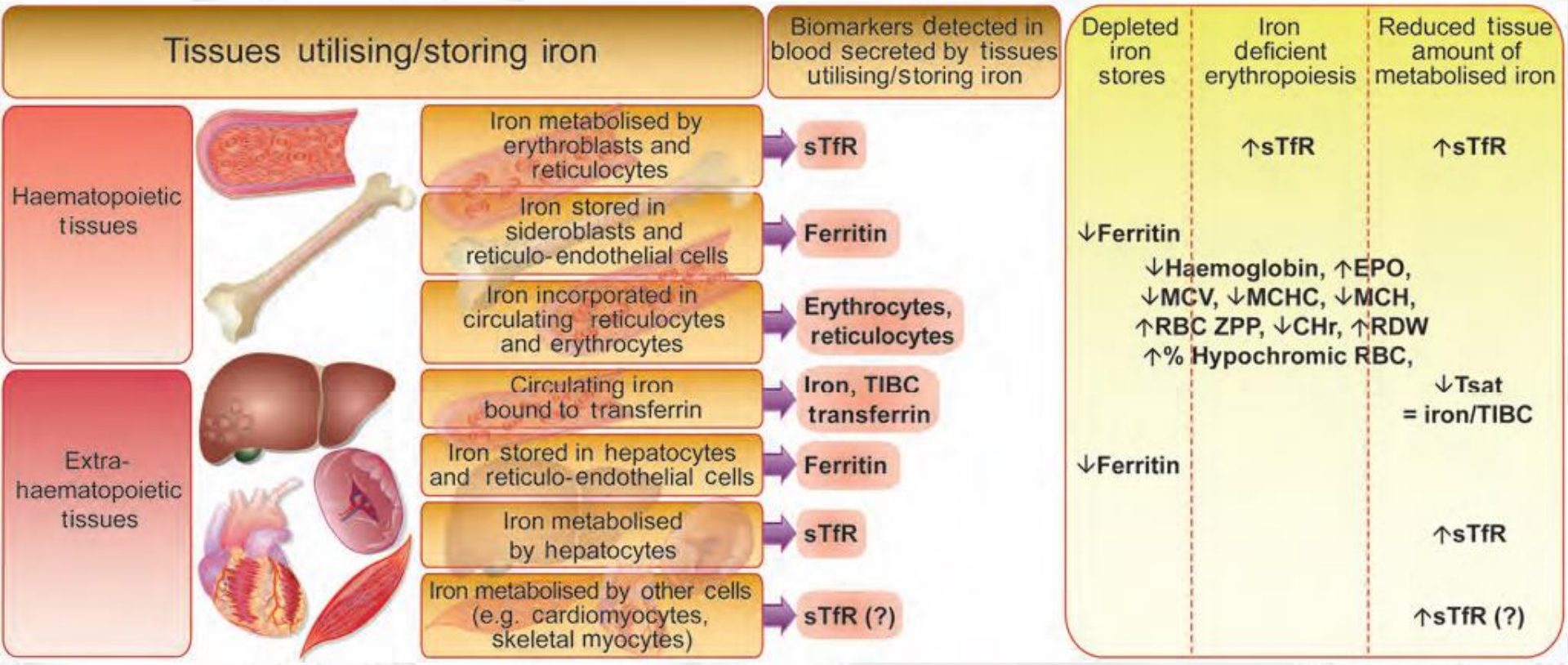
↓ capacidad aeróbica (↓ VO_2 máx)
↓ resistencia al ejercicio

Alteraciones cognitivas
Alteraciones del comportamiento
Síntomas depresivos

Manifestaciones clínicas del déficit de hierro en la IC



Biomarcadores de utilización y almacenamiento de hierro



Jankowska EA. Eur Heart J 2013;34: 816-826

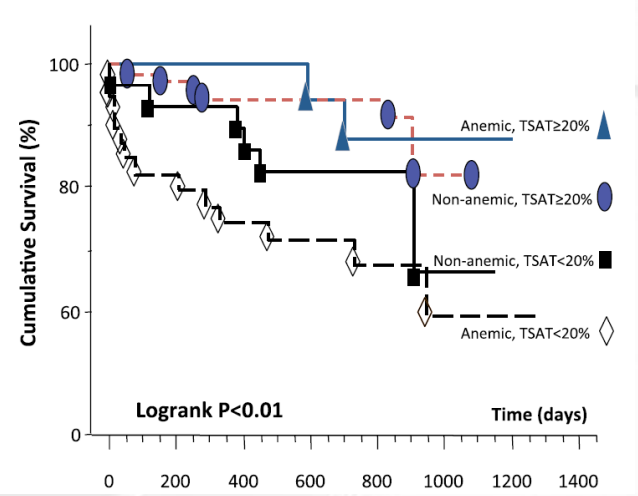
Ferritina sérica y saturación de transferrina (TSAT)

- **Ferritina = depósitos**
- **TSAT = hierro disponible** para **eritropoyesis** y **otros procesos metabólicos**

	Características	Criterios diagnósticos en IC
Déficit absoluto de hierro	**Depósitos insuficientes** para satisfacer las necesidades del organismo	Ferritina <100 µg/ml
Déficit funcional de hierro	**Depósitos intactos** pero su **movilización** está **comprometida**	Ferritina 100-299 µg/ml TSAT <20%

Ferritina sérica y saturación de transferrina (TSAT)

- **TSAT más sensible** que ferritina **para diagnóstico de DH**[1,2]
- **Valores más bajos** de **TSAT ↔ clase NYHA avanzada, ↓ VO pico y ↑ mortalidad**[3]
- **Limitaciones** de la **TSAT:**
  - fluctuaciones diurnas de su valor (17-70%)
  - ↑ en malnutrición y procesos crónicos (↓ transferrina)
  - ↓ en procesos inflamatorios (↑ transferrina)



[1]Fishbane S et al. J Am Soc Neprol 1996;7:2654-7
[2]Tessitore N et al. Nephrol Dial Transplant 2001;16:1416-123
[3]Okonko DO et al. J Am Coll Cardiol 2011;58:1241-51

Ferritina sérica y saturación de transferrina (TSAT)

Intravenous iron reduces NT-pro-brain natriuretic peptide in anemic patients with chronic heart failure and renal insufficiency

Methods

Forty patients with hemoglobin (Hb) <12.5 g/dl, transferrin saturation <20%, ferritin <100 ng/ml, creatinine clearance (CrCl) <90 ml/min, and left ventricular ejection fraction (LVEF) < or =35% were randomized into 2 groups (n = 20 for each). For 5 weeks, group A received isotonic saline solution and group B received iron sucrose complex, 200 mg weekly. Minnesota Living with Heart Failure Questionnaire (MLHFQ) and 6-min walk (6MW) test were performed. NT-pro brain natriuretic peptide and CRP were evaluated throughout the study. No patients received erythroprotein any time.

Effect of Intravenous Iron Sucrose on Exercise Tolerance in Anemic and Nonanemic Patients With Symptomatic Chronic Heart Failure and Iron Deficiency

FERRIC-HF: A Randomized, Controlled, Observer-Blinded Trial

Methods

We randomized 35 patients with CHF (age 64 ± 13 years, peak oxygen consumption [pVo_2] 14.0 ± 2.7 ml/kg/min) to 16 weeks of intravenous iron (200 mg weekly until ferritin >500 ng/ml, 200 mg monthly thereafter) or no treatment in a 2:1 ratio. Ferritin was required to be <100 ng/ml or ferritin 100 to 300 ng/ml with transferrin saturation <20%. Patients were stratified according to hemoglobin levels (<12.5 g/dl [anemic group] vs. 12.5 to 14.5 g/dl [nonanemic group]). The observer-blinded primary end point was the change in absolute pVo_2.

Toblli JE. J Am Coll Cardiol 2007; 50: 1657-1665
Okonko DO. J Am Coll Cardiol 2008; 51: 103-112

Ferritina sérica y saturación de transferrina (TSAT)

FAIR-HF

METHODS

We enrolled 459 patients with chronic heart failure of New York Heart Association (NYHA) functional class II or III, a left ventricular ejection fraction of 40% or less (for patients with NYHA class II) or 45% or less (for NYHA class III), iron deficiency (ferritin level <100 μg per liter or between 100 and 299 μg per liter, if the transferrin saturation was <20%), and a hemoglobin level of 95 to 135 g per liter. Patients were randomly assigned, in a 2:1 ratio, to receive 200 mg of intravenous iron (ferric carboxymaltose) or saline (placebo). The primary end points were the self-reported Patient Global Assessment and NYHA functional class, both at week 24. Secondary end points included the distance walked in 6 minutes and the health-related quality of life.

CONFIRM-HF

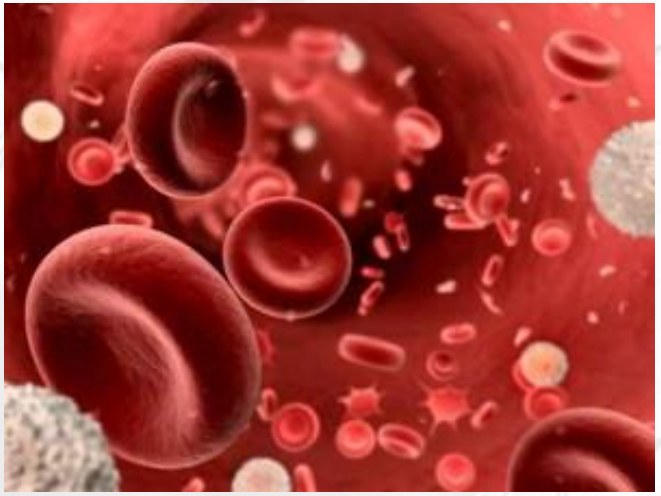
Methods and results

CONFIRM-HF was a multi-centre, double-blind, placebo-controlled trial that enrolled 304 ambulatory symptomatic HF patients with left ventricular ejection fraction ≤45%, elevated natriuretic peptides, and iron deficiency (ferritin <100 ng/mL or 100–300 ng/mL if transferrin saturation <20%). Patients were randomized 1 : 1 to treatment with i.v. iron, as ferric carboxymaltose (FCM, $n = 152$) or placebo (saline, $n = 152$) for 52 weeks. The primary end-point was the change in 6-min-walk-test (6MWT) distance from baseline to Week 24. Secondary end-points included changes in New York Heart Association (NYHA) class, Patient Global Assessment (PGA), 6MWT distance, health-related quality of life (QoL), Fatigue Score at Weeks 6, 12, 24, 36, and 52 and the effect of FCM on the rate of hospitalization for worsening HF. Treatment with FCM significantly prolonged 6MWT distance at Week 24 (difference FCM vs. placebo: 33 ± 11 m, $P = 0.002$). The treatment effect of FCM was consistent in all subgroups and was sustained to Week 52 (difference FCM vs. placebo: 36 ± 11 m, $P < 0.001$). Throughout the study, an improvement in NYHA class, PGA, QoL, and Fatigue Score in patients treated with FCM was detected with statistical significance observed from Week 24 onwards. Treatment with FCM was associated with a significant reduction in the risk of hospitalizations for worsening HF [hazard ratio (95% confidence interval): 0.39 (0.19–0.82), $P = 0.009$]. The number of deaths (FCM: 12, placebo: 14 deaths) and the incidence of adverse events were comparable between both groups.

Anker SD. N Engl J Med 2009; 361: 2436-48
Ponikowski P. Eur Heart J 2014 Aug 31. pii: ehu385. [Epub ahead of print]

Hemograma y eritropoyesis deficitaria en hierro

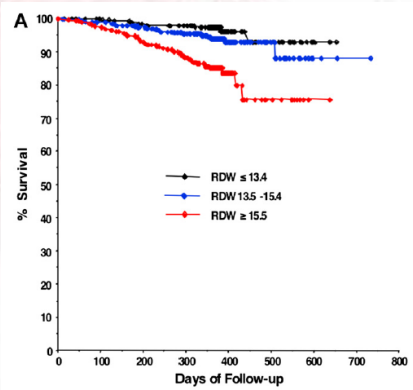
- **Precozmente** ↓ contenido de hemoglogina de los reticulocitos (CHR) **<28 pg**
- **Más tarde:**
  - ↑ % hematíes hipocrómicos (S y E del 78.1% si >6%)
  - ↓ VCM
  - ↓ HCM
  - ↓ CHCM
  - ↓ Hemoglobina



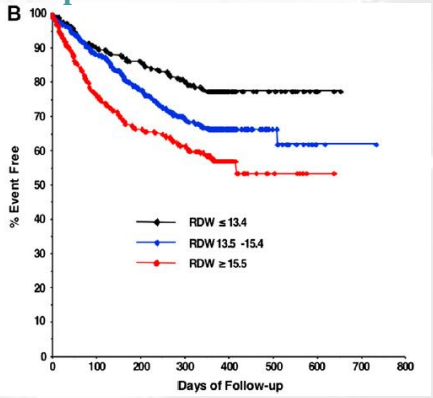
Hemograma y eritropoyesis deficitaria en hierro: ADE

- Índice cuantitativo de **anisocitosis** (↑ hematíes microcíticos)
- Normal: 11.5-14.5%
- Aumenta **precozmente**
- Permite **monitorizar** el **efecto** de la **ferroterapia**
- Marcador de **peor pronóstico** en **IC crónica** e **IC aguda**

Mortalidad



Hospitalización o mortalidad

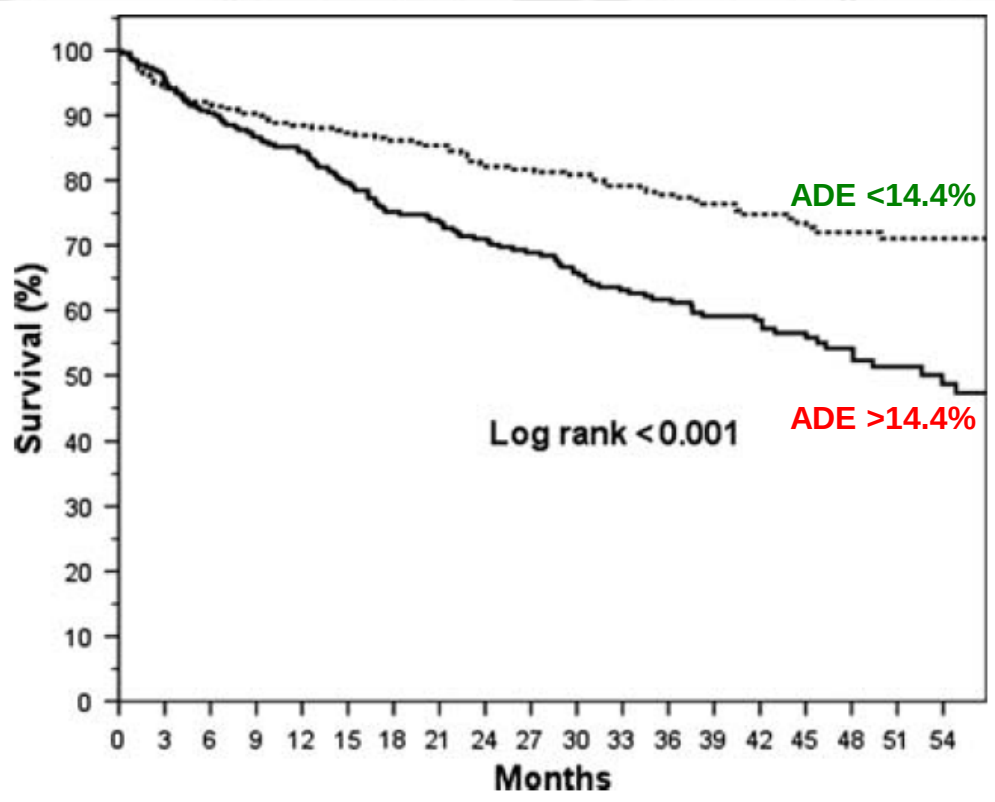


- Puede alterarse si déficit de vitamina B12 / ácido fólico o anemia sideroblástica

Allen LA. J Card Fail 2010; 16: 230-238
Constantino BT. Hematology 2013;44:e2-e9

ADE y pronóstico en IC aguda

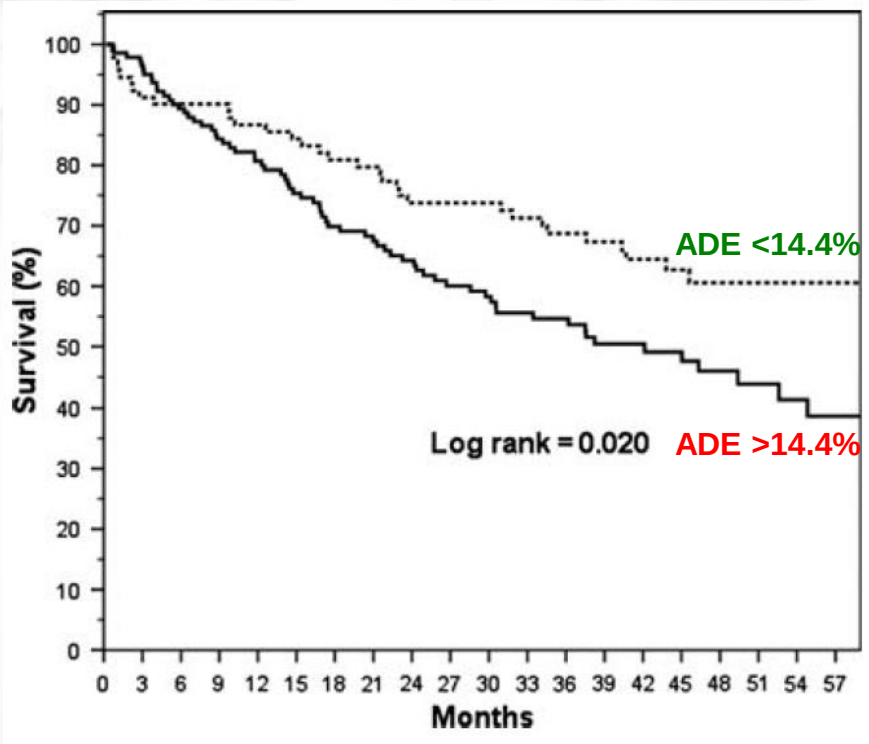
- 628 pacientes ingresados por IC aguda en planta de Cardiología (Enero 2002-Diciembre 2003)
- Edad media: 71 años (61-77)
- FEVI media: 37 % (30-45)
- 55% causa isquémica
- 57% HTA
- 39% diabetes mellitus
- 26% fibrilación auricular
- Hb y ADE previos al alta
- Supervivencia tras el alta
- Seguimiento medio: 38.1 meses



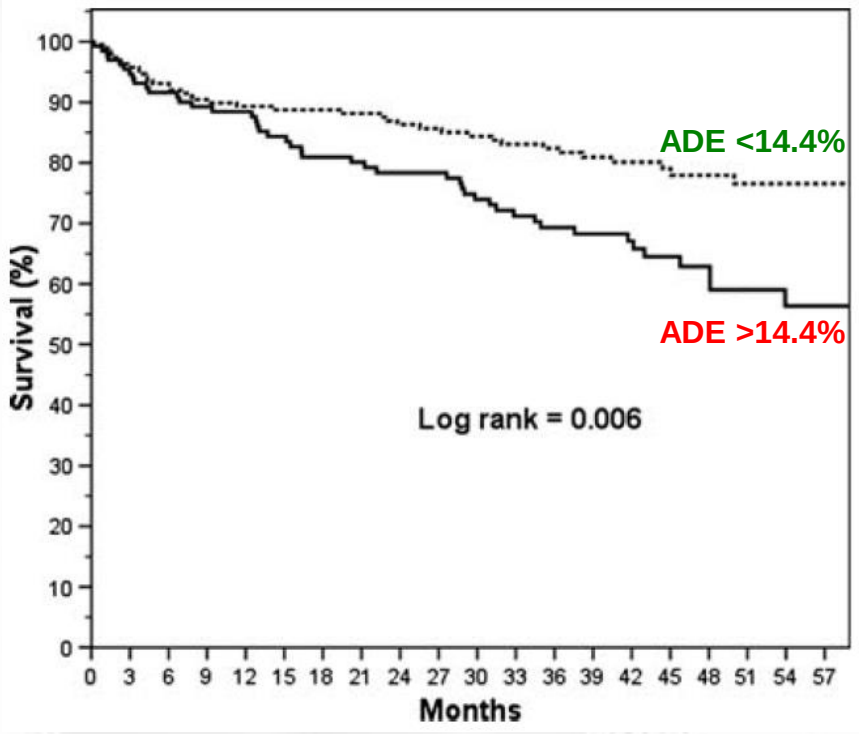
Pascual-Figal DA. Eur J Heart Fail 2009;11: 840-846

ADE y pronóstico en IC aguda

Pacientes con anemia



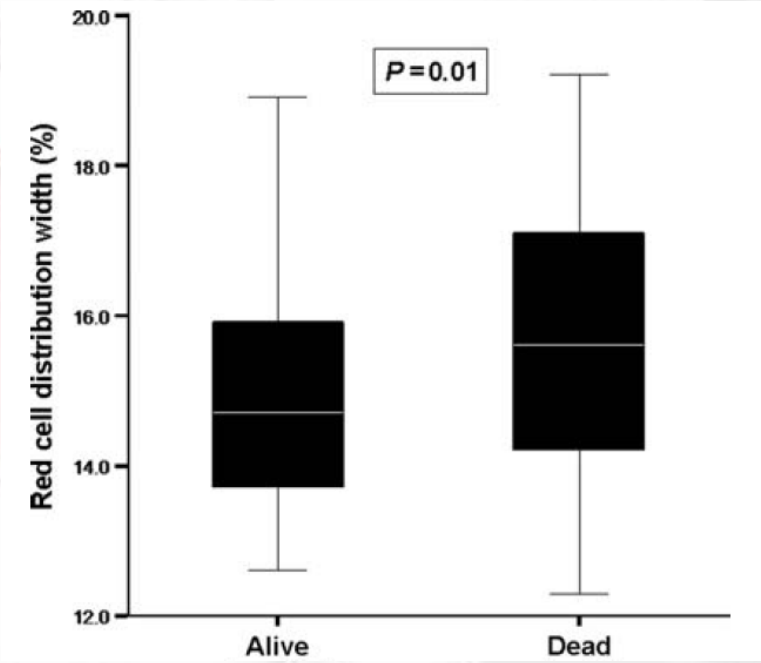
Pacientes sin anemia



Pascual-Figal DA. Eur J Heart Fail 2009;11: 840-846

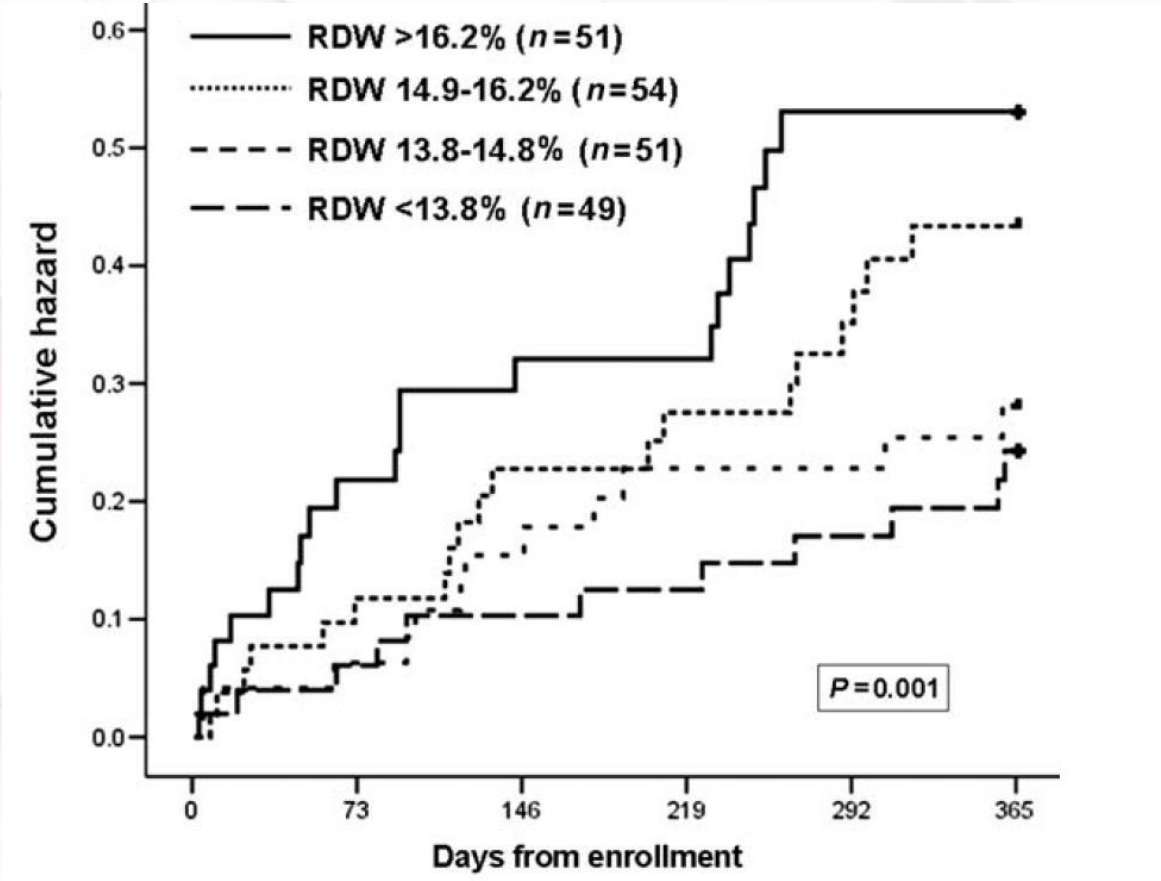
ADE y pronóstico en IC aguda

- 205 pacientes que acuden a Urgencias por IC aguda (42% con FEVI preservada)
- ADE en Urgencias
- Mortalidad al año



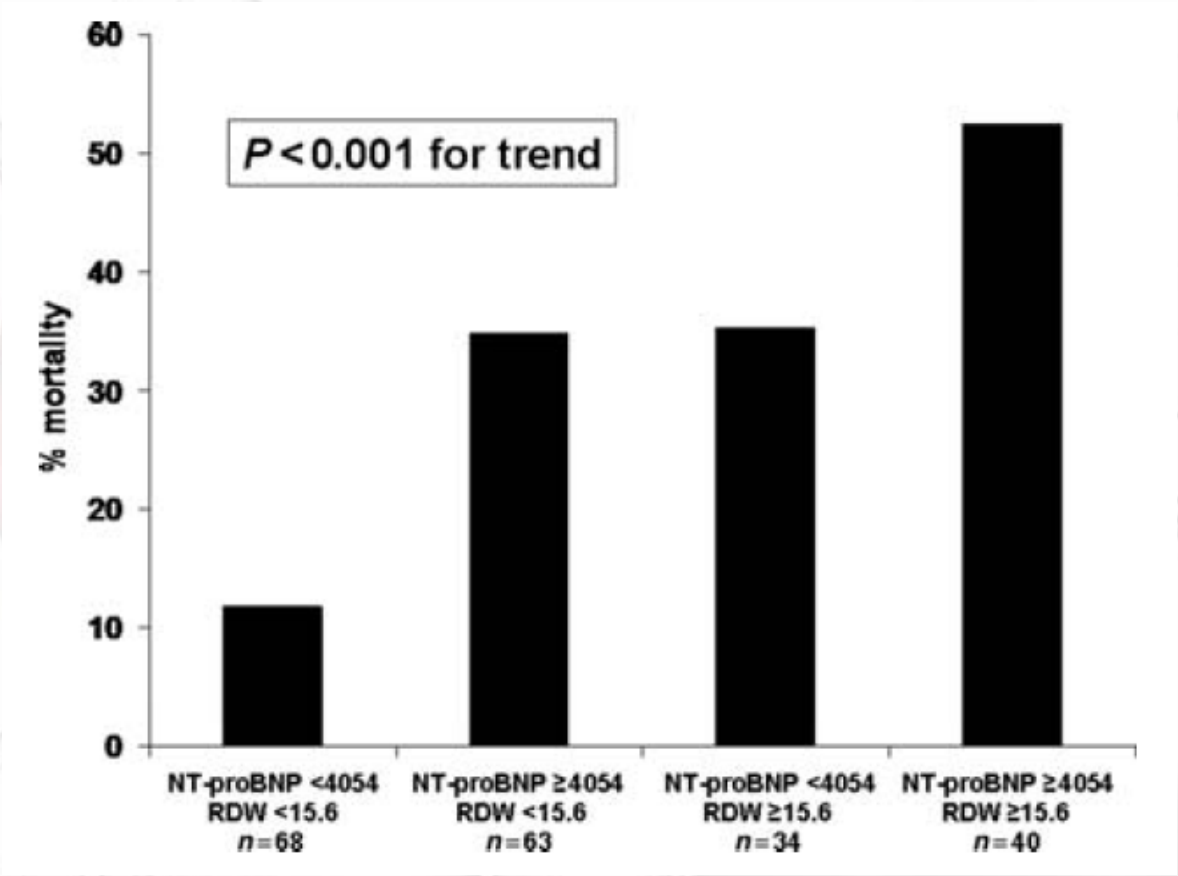
Van Kimmenade RRJ. Eur J Heart Fail 2010;12: 129-136

ADE y pronóstico en IC aguda



Van Kimmenade RRJ. Eur J Heart Fail 2010;12: 129-136

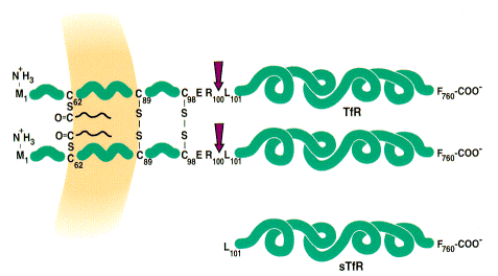
ADE y pronóstico en IC aguda

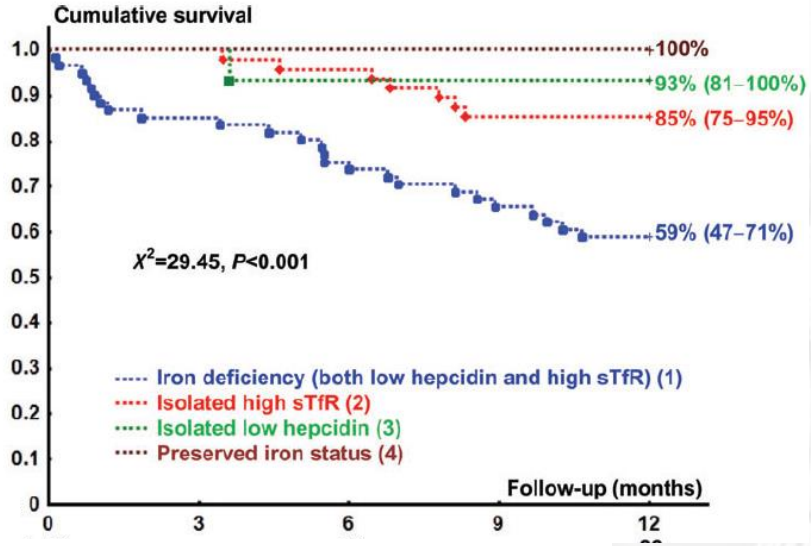


Van Kimmenade RRJ. Eur J Heart Fail 2010;12: 129-136

Receptor soluble de la transferrina

- Responsable de la **incorporación intracelular de Fe**
- Sus **niveles plasmáticos aumentan** en pacientes con **DH =** requerimientos celulares insatisfechos
- **Valores >1.5 mg/l** tienen una **sensibilidad** del **81%** y una **especificidad** del **71%**
- **No influido por estado inflamatorio** (a diferencia de la ferritina)

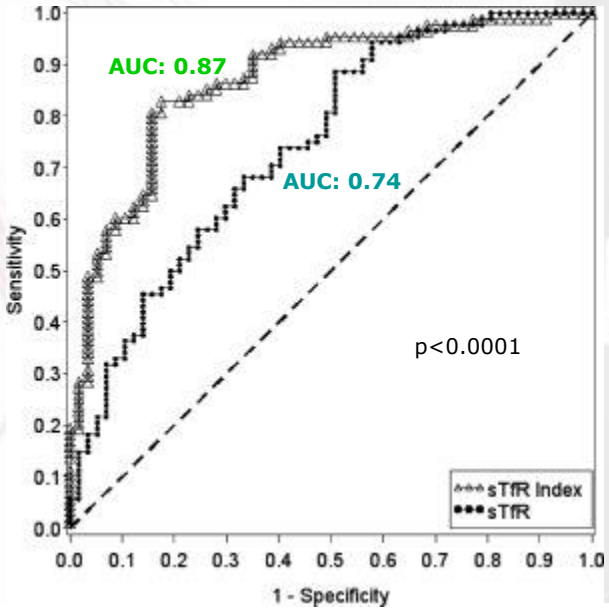




Jankowska EA. Eur Heart J 2014; doi:10.1093/eurheartj/ehu235

Índice de ferritina (sTfR/log ferritina)

- 145 pacientes con anemia (67% mujeres)
  - -Anemia por déficit de hierro (n: 27)
  - -Anemia de proceso crónico (n: 57)
  - -Ambas (n: 61)
- Edad media: 62 años
- Ferritina
- sTfR
- sTfR/log ferritina

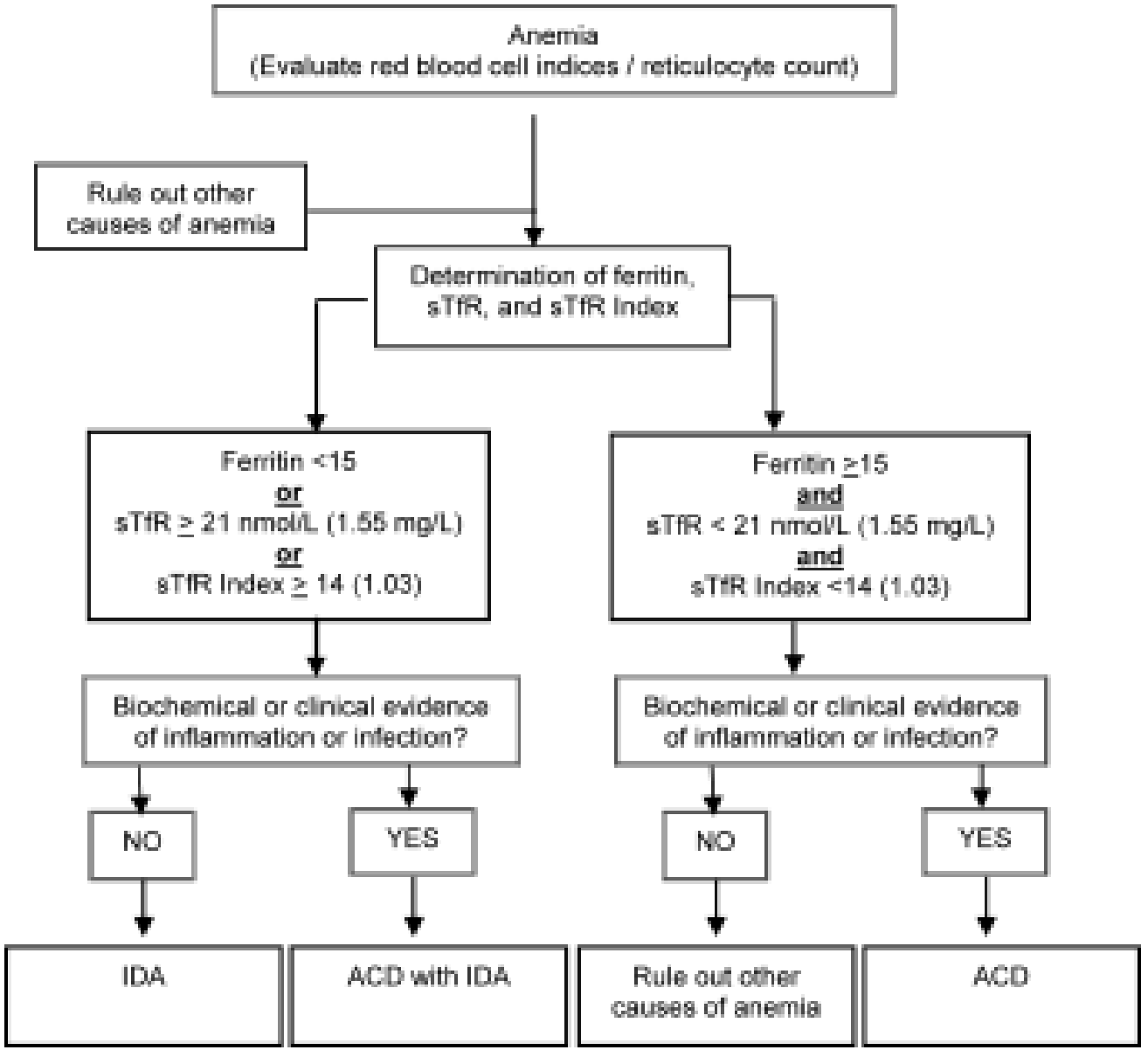


Skikne BS. Am J Hematol 2011; 86:923-927

Índice de ferritina (sTfR/log ferritina)

	Sensibilidad (% pacientes con anemia por DH y anemia de proceso crónico correctamente diagnosticados)	**Especificidad** (% pacientes con anemia de proceso crónico, sin anemia por déficit de hierro, correctamente diagnosticados)
Ferritina < 15 ng/ml	41	96
Ferritina < 30 ng/ml	59	93
sTfR ≥21 nmol/l (≥1.55 mg/l)	86	49
Índice de ferritina ≥14	**81**	**83**
Ferritina <15 ng/mL ó sTfR ≥21 nmol/L (≥1.55 mg/L) ó Índice de ferritina ≥14	92	49

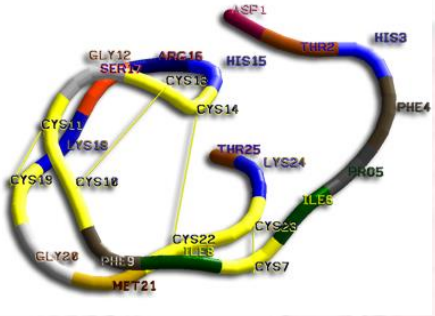
Skikne BS. Am J Hematol 2011; 86:923-927

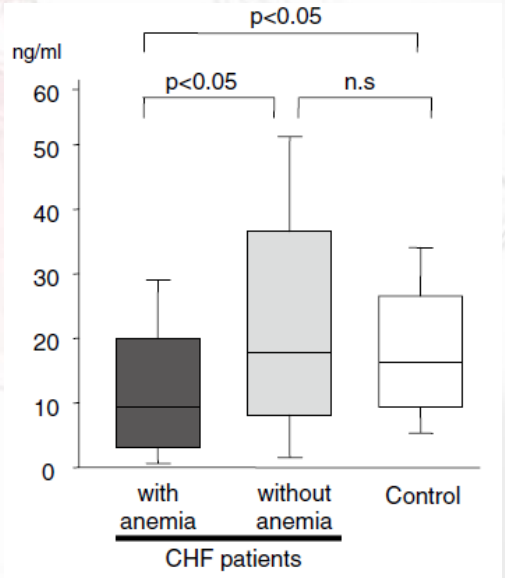


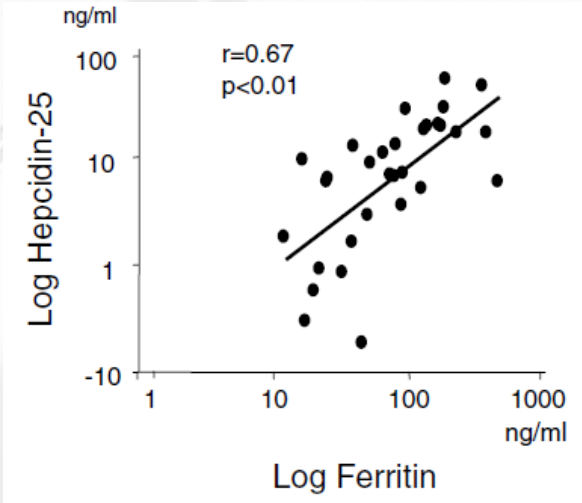
Skikne BS. Am J Hematol 2011; 86:923-927

Hepcidina

- **Determinación plasmática / urinaria** (ELISA / cromatografía líquida con espectrometría de masa)
- **Niveles disminuidos** se correlacionan con el grado de **depleción** de los **depósitos** de Fe **(ferritina)**[1]
- Probable utilidad en **IC aguda** (niveles bajos en 46% pacientes)[2]



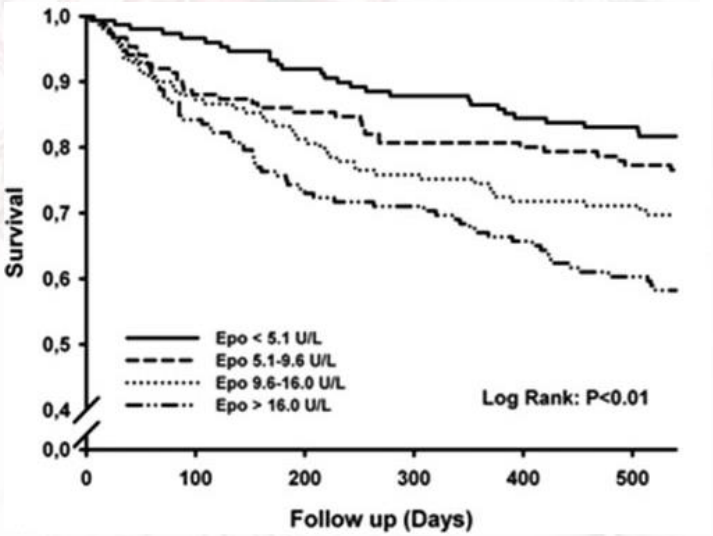


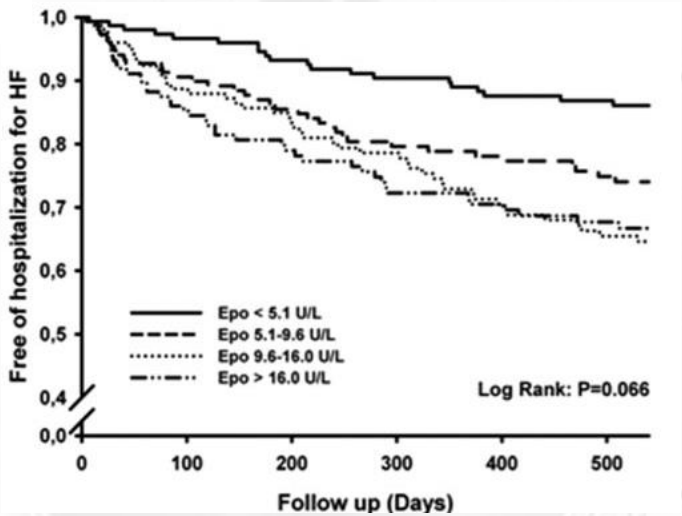


[1]Matsumoto M et al. Circ J 2010; 74:301-6
[2]Jankowska EA et al. Eur Heart J 2014;35:2468-76

Eritropoyetina plasmática

- **Niveles elevados** indican **eritropoyesis deficitaria en Fe**
- Se relacionan con **peor evolución** de la IC
  - Empeoramiento de clase NYHA
  - Menor supervivencia independientemente de Hb





Belonje AMS et al. Circulation 2010;121:245-251

Protoporfirina zinc

- Producto de la **síntesis anómala** de grupos **hemo**
- Refleja la **incorporación** de **Zn** (en vez de Fe) en la **protoporfirina IX** (eritropoyesis deficitaria en Fe)
- Valores normales ≤40 mmol/mol hemo
- **Aumenta** de forma **más precoz** que el **descenso de la Hb *y* se normaliza tras ferroterapia**
- **No se correlaciona con** la **ferritina** (principal indicador de depósitos de Fe)

	Ferritina	Protoporfirina zinc	Hemoglobina
Grado I: Depleción de hierro	↓	Normal	Normal
Grado II: Eritropoyesis deficitaria en hierro	↓	↑	Normal
Grado III: Anemia por déficit de hierro	↓	↑	↓

Hierro sérico

- Muestra **importantes variaciones individuales**
- Aporta **menos información** sobre metabolismo del Fe **que ferritina**
- No debería emplearse en la valoración del DH

Hastka J. Clin Chem 1994;40:768-773

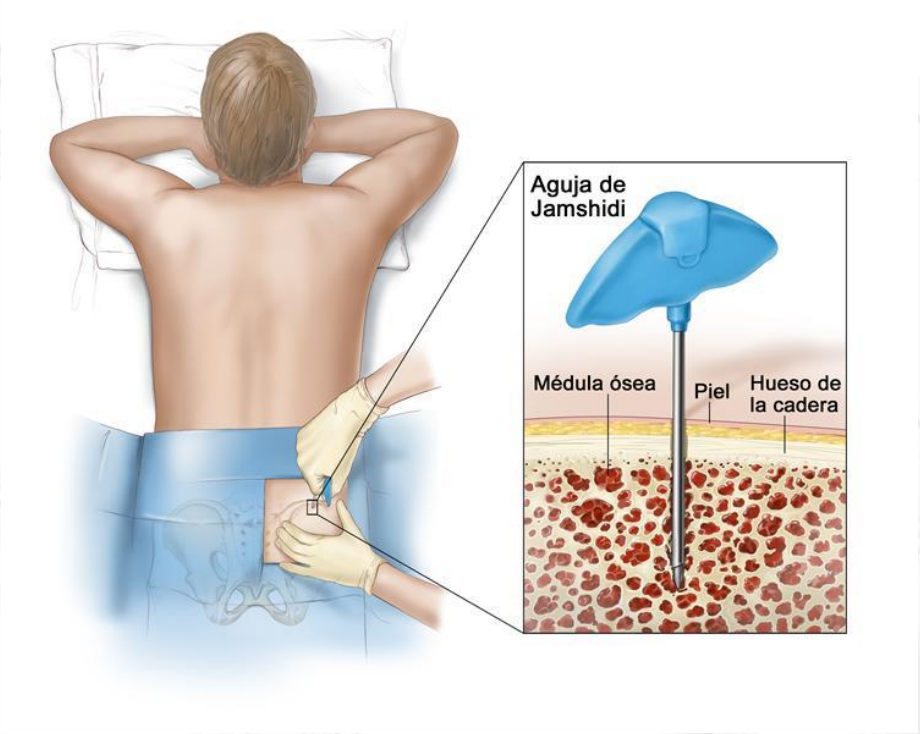
Déficit de hierro absoluto vs déficit de hierro funcional

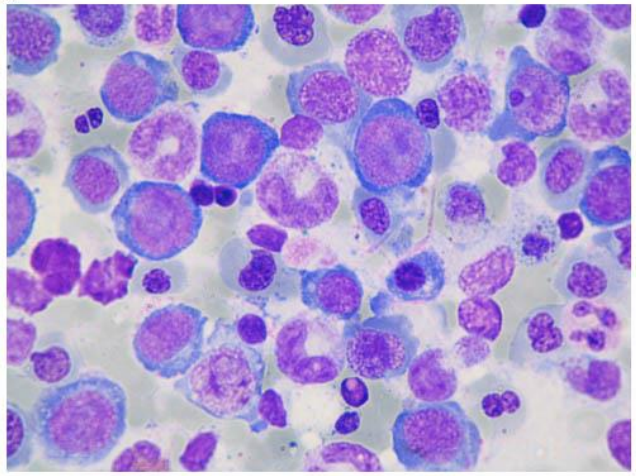
	DÉFICIT DE HIERRO	
	Absoluto	Funcional
Hierro sérico	↓	↓
Ferritina	↓	Normal / ↑
TSAT	↓	↓

Déficit de hierro absoluto vs déficit de hierro funcional

	DÉFICIT DE HIERRO	
	Absoluto	Funcional
Hierro sérico	↓	↓
Ferritina	↓	Normal / ↑
Transferrina	↑	Normal / ↓
TSAT	↓	↓
sTfR	↑	Normal / ↓
Hepcidina	↓	Normal / ↑
Depósitos medulares de hierro	↓	↑

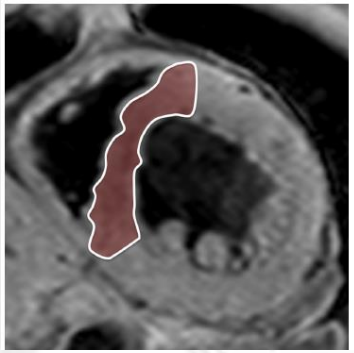
Biopsia de médula ósea





Quantification of Myocardial Iron Deficiency in Nonischemic Heart Failure by Cardiac T2* Magnetic Resonance Imaging

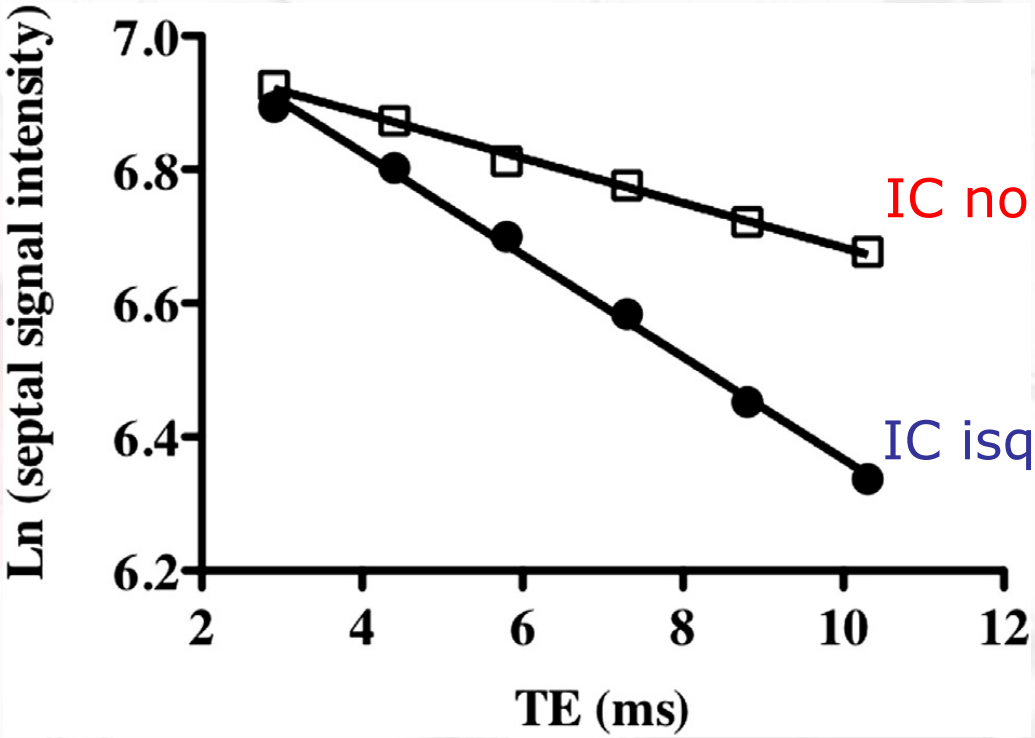
- Cardiorresonancia (secuencia T2 con valoración de volúmenes ventriculares y FEVI)
  - 167 pacientes con IC no isquémica
  - 31 pacientes con IC isquémica
  - 50 personas sin IC (BNP y FEVI normales)



- MACE (muerte cardiaca, ingreso por IC ó implante de asistencia ventricular)
- Seguimiento medio: 20 meses

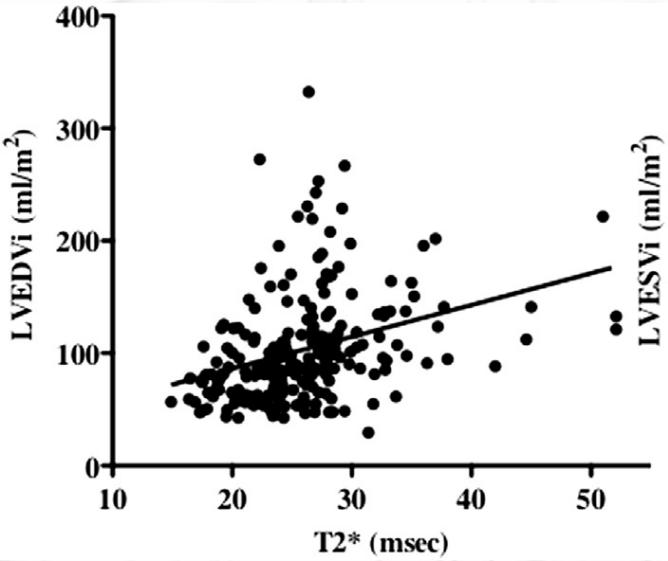
Nagao M et al. Am J Cardiol 2014;113:1024-30

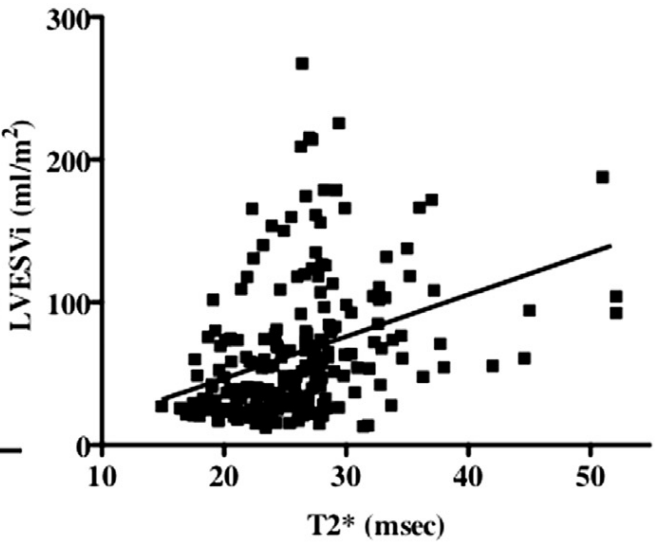
Quantification of Myocardial Iron Deficiency in Nonischemic Heart Failure by Cardiac T2* Magnetic Resonance Imaging

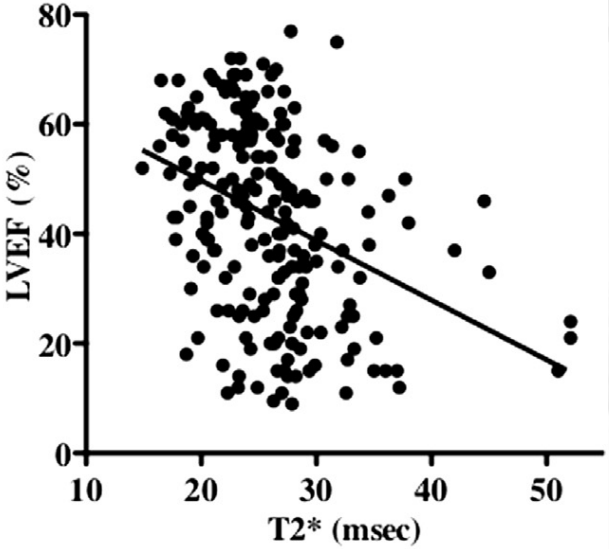


Nagao M et al. Am J Cardiol 2014;113:1024-30

Quantification of Myocardial Iron Deficiency in Nonischemic Heart Failure by Cardiac T2* Magnetic Resonance Imaging







Nagao M et al. Am J Cardiol 2014;113:1024-30

Quantification of Myocardial Iron Deficiency in Nonischemic Heart Failure by Cardiac T2* Magnetic Resonance Imaging

Risk for major adverse cardiac event in patients with nonischemic heart failure (n = 167)

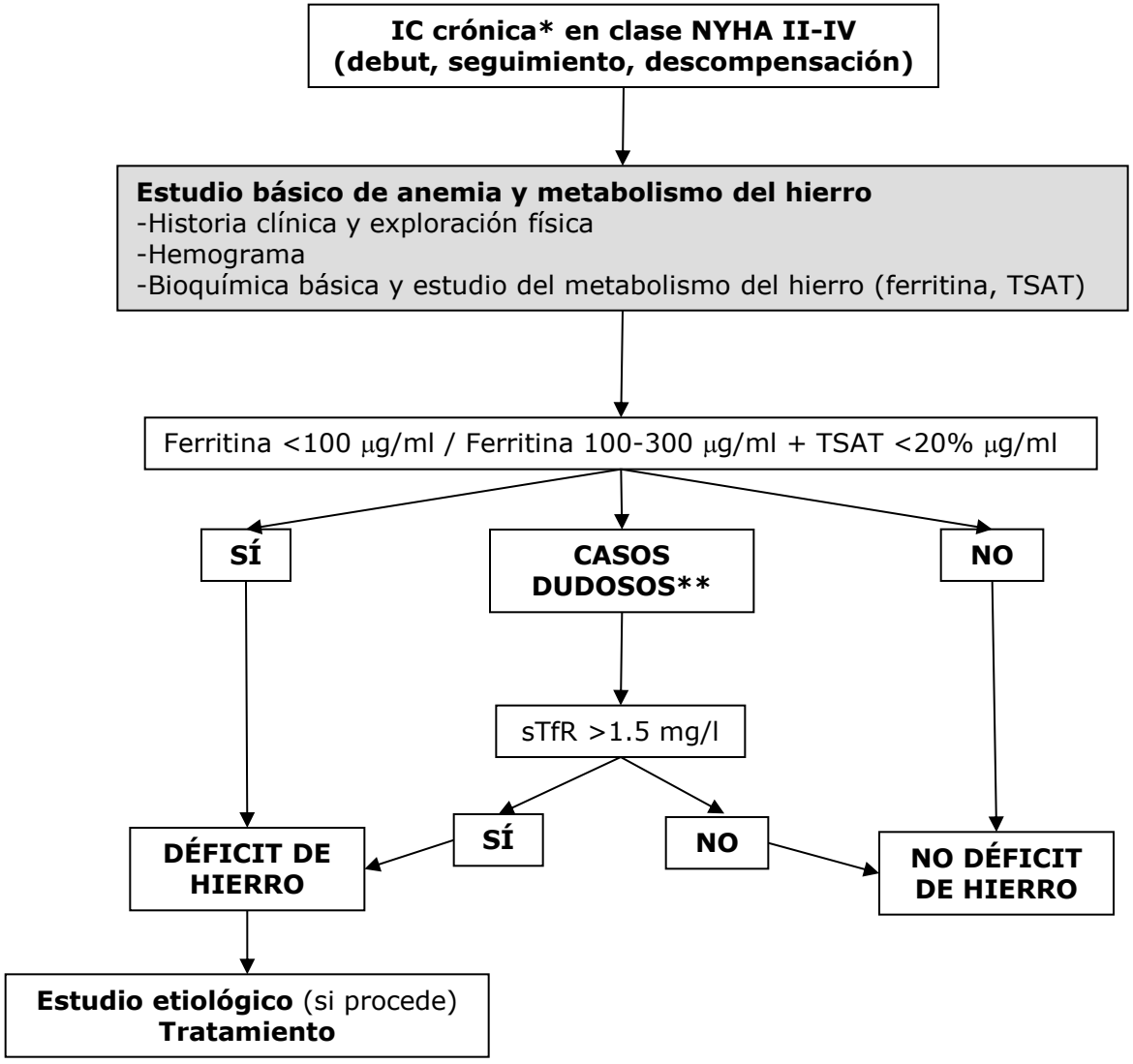
Parameter	Univariate Logistic Analysis			Multivariate Logistic Analysis			
	Chi-Square	Odds Ratio	p Value	Chi-Square	Odds Ratio	95% CI	p Value
Age	0.04	0.99	0.85				
Gender	1.05	0.57	0.43				
LVEF	7.05	0.95	0.01	2.58	0.97	0.93–1.01	0.11
Systolic blood pressure	5.99	0.96	0.014	6.31	0.96	0.93–0.99	0.012
Hemoglobin	0.04	1.03	0.83				
Estimated glomerular filtration rate	0.41	0.99	0.52				
BNP	0.44	1.0001	0.51				
T2*	5.83	1.09	0.016	4.7	1.1	1.01–1.21	0.03
LGE	1.98	0.44	0.27				

Umbral óptimo de T2 para predecir MACE: 25.1 ms

(S: 94%, E: 40%)

Nagao M et al. Am J Cardiol 2014;113:1024-30

Algoritmo diagnóstico del déficit de hierro en la insuficiencia cardiaca



IC: insuficiencia cardiaca, sTfR: receptor soluble de transferrina, TSAT: saturación de transferrina

*En IC aguda con diagnóstico dudoso tras determinación de ferritina y TSAT podría ser útil la determinación de hepcidina y sTfR (Jankowska EA et al. Eur Heart J 2014; doi:10.1093/eurheartj/ehu235)
**Coexistencia de otros procesos crónicos


Muchas gracias por vuestra atención