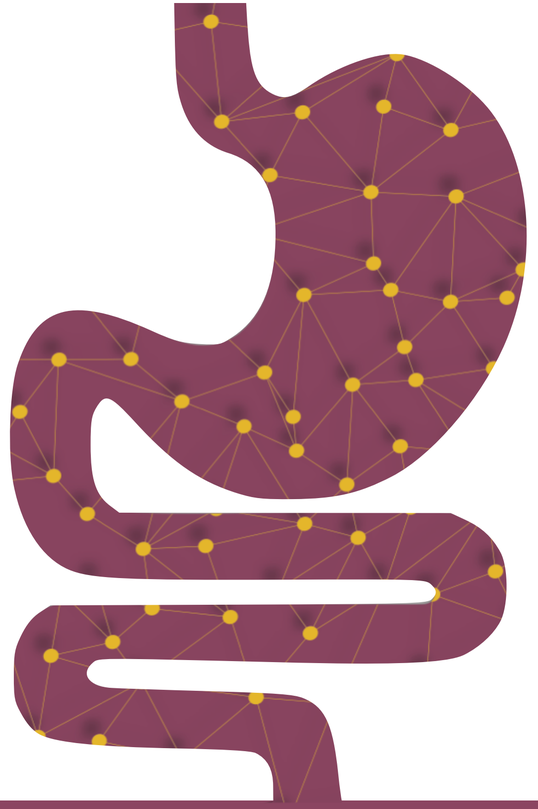

II JORNADA DE FORMACIÓN CONTINUADA EN GASTROENTEROLOGÍA Y HEPATOLOGÍA PARA RESIDENTES

24 y 25 de septiembre de 2021
Palacio de Congresos y Exposiciones de Ronda

www.sapd.es

TALLER CLÍNICO: CIRROSIS DESCOMPENSADA

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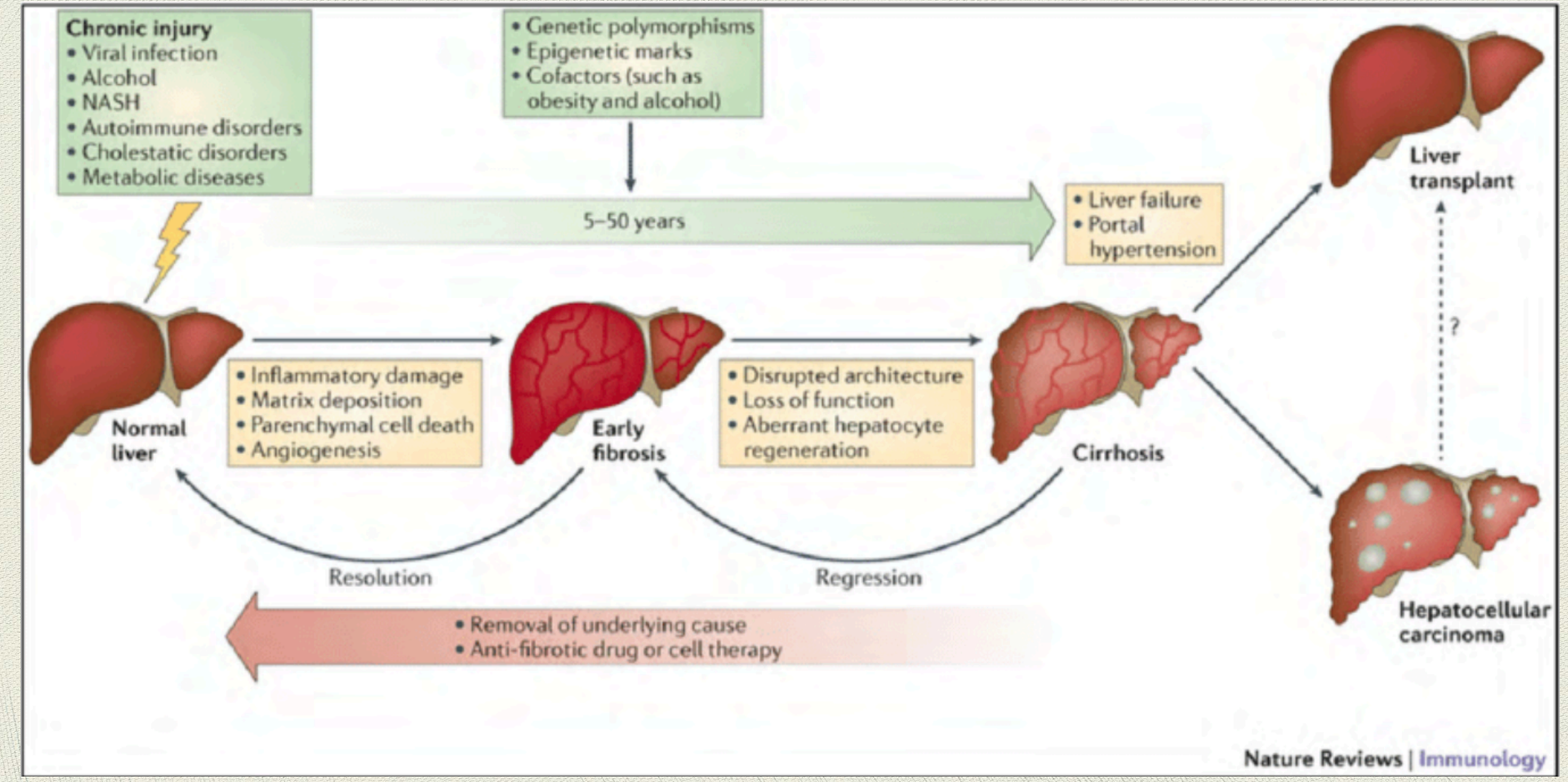
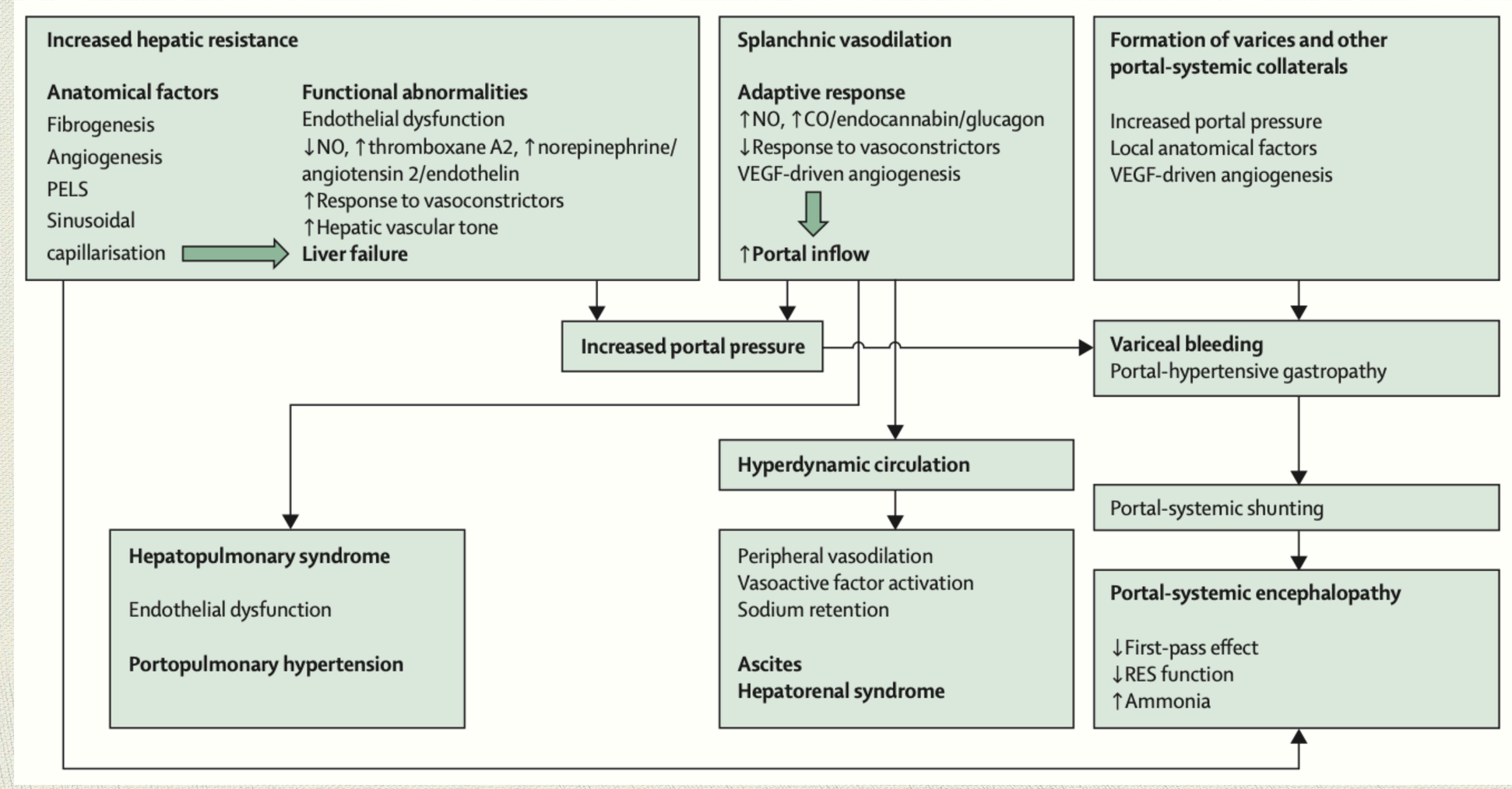
Patricia Cordero Ruíz
Hospital Universitario Virgen Macarena (Sevilla)

De hepatopatía a cirrosis hepática

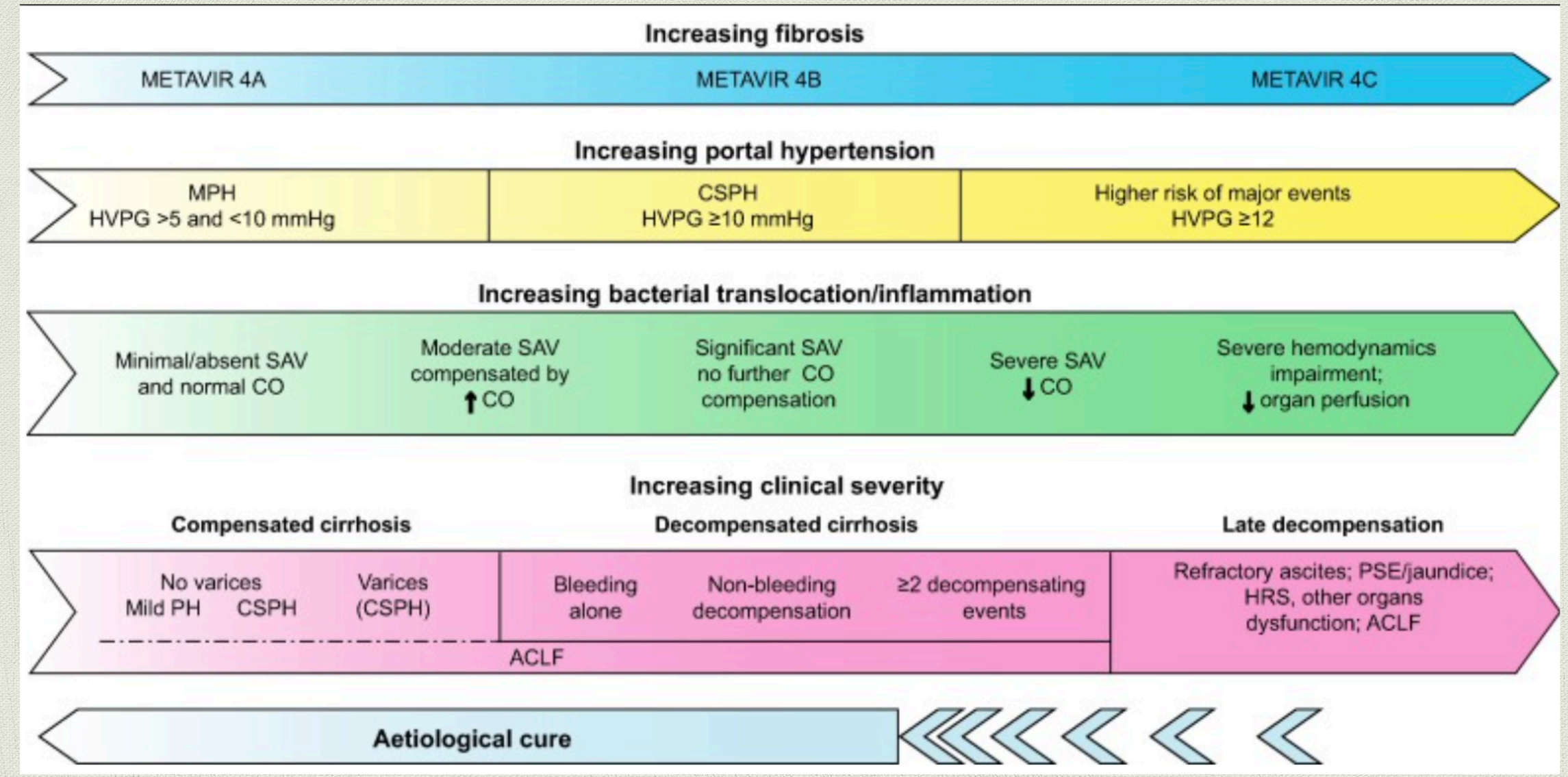


The natural history of chronic liver disease. Nat Rev Immunol. 2014;14(3):181-194

Fisiopatología de la cirrosis

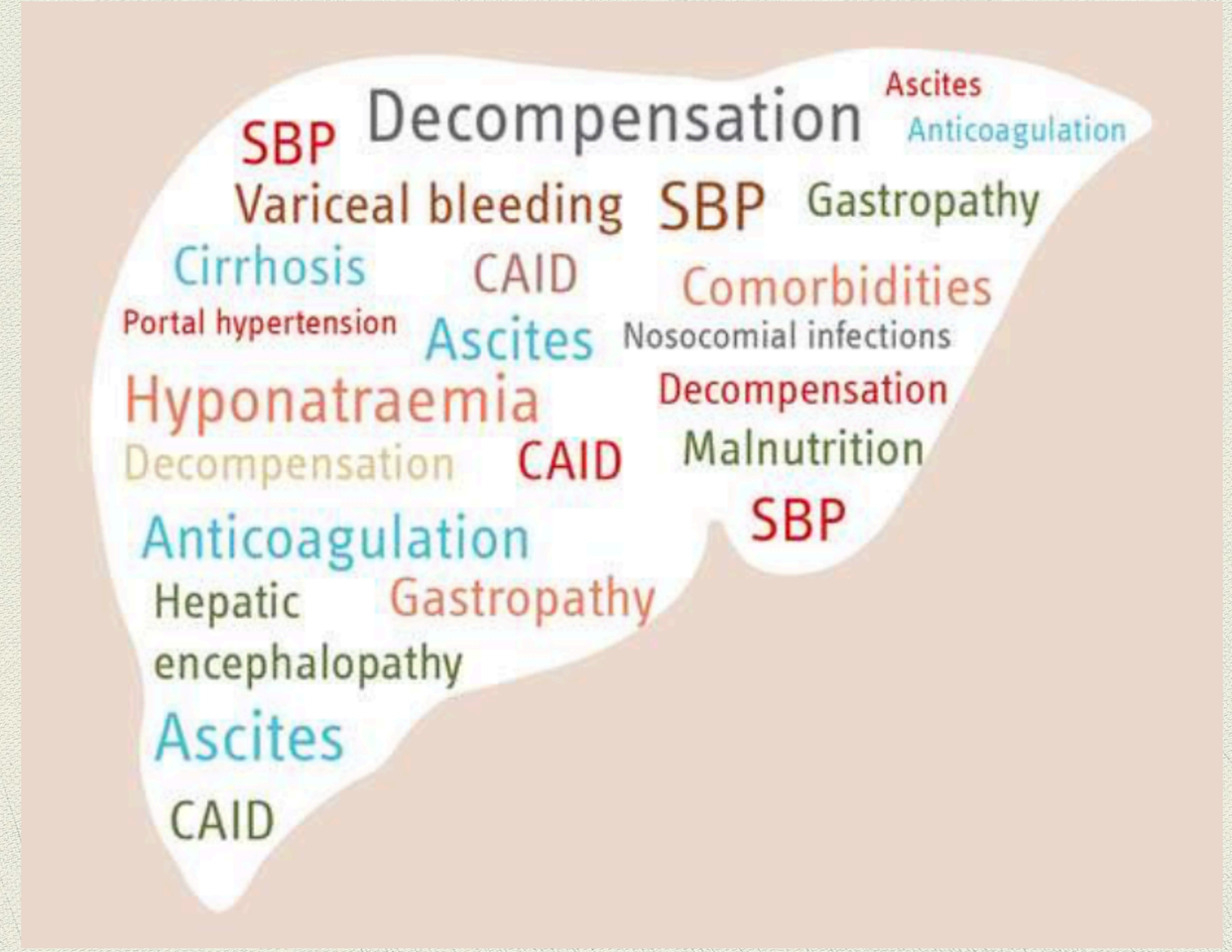


Liver cirrhosis. Emmanuel A Tsochatzis, Jaime Bosch, Andrew K Burroughs. The Lancet. 2014.



Clinical states of cirrhosis and competing risks. D´Amico. Journal of hepatology. 2020.

Manifestaciones de la cirrosis descompensada



Caso Clínico

ANTECEDENTES PERSONALES

- Varón 53 años
- Tóxicos: Ex bebedor de 15 UBE (Abstinencia desde julio 2017). Fumador de 20 cigarrillos al día.
- Factores de riesgo cardiovascular: Obesidad (IMC 31Kg/m2), HTA, DL.
- **Cirrosis hepática de origen mixto (OH + metabólica). Estadio funcional: CPT A6, MELD 9, MELD-Na 13. Cribado VVEE en 2018: Gastropatía por HTP. Ascitis leve controlada con Espironolactona.**
- En contexto de pandemia SARS-CoV **pierde seguimiento: última consulta septiembre 2019.**
- Última analítica Abril 2021: Hb 12.1 PQ 125.000 **Cr 1.1** AST 45 ALT 51 Bt 1.2

HISTORIA ACTUAL

Trasladado a urgencias por la familia en junio 2021 por **distensión abdominal, intensa astenia y "comportamiento extraño"** de una semana de evolución.

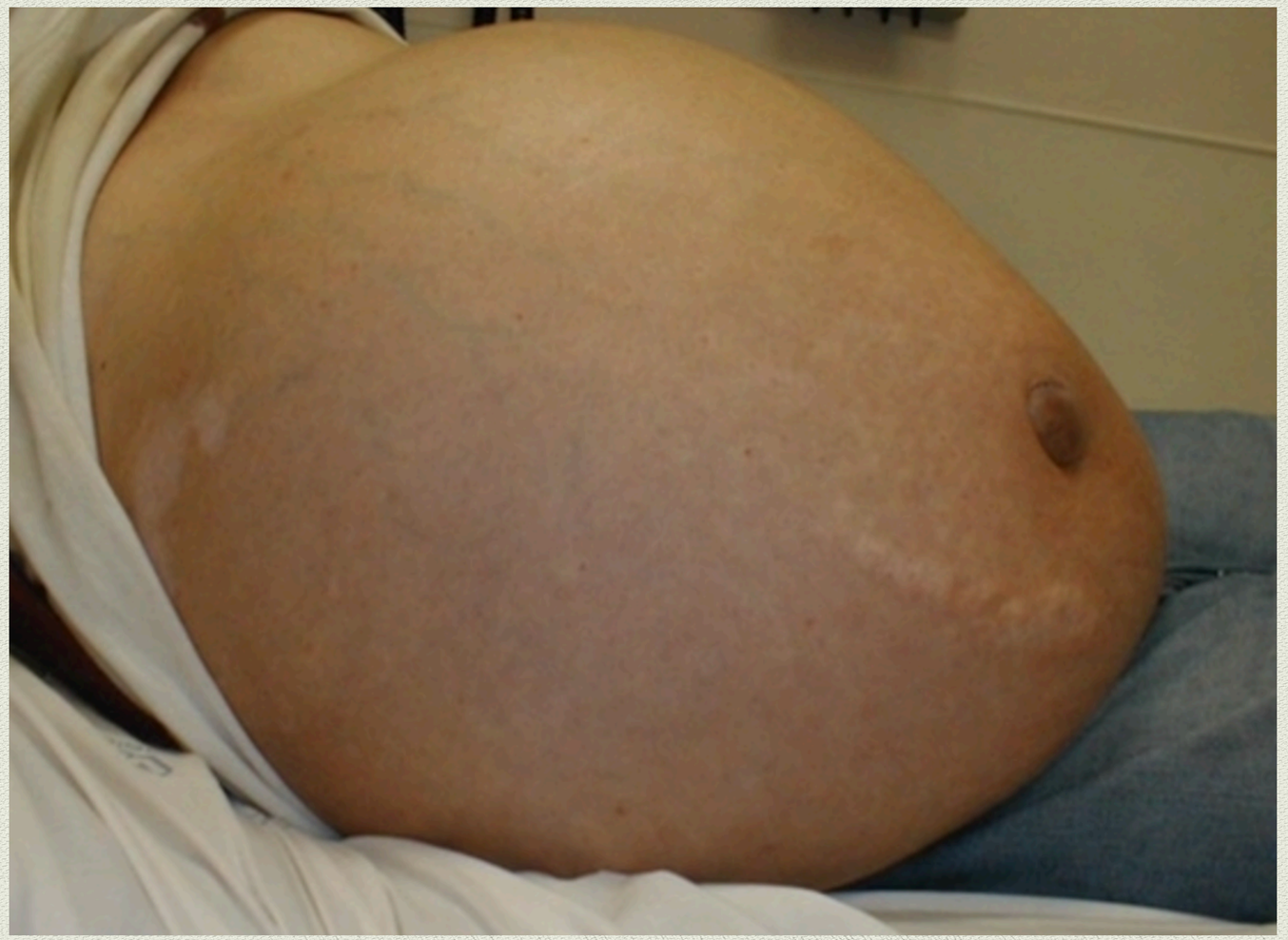
- No ha presentado edemas, fiebre ni exteriorización hemorrágica.
- **Tendencia al estreñimiento** que achaca a baja ingesta.
- Abandono del seguimiento por "encontrarse bien" y " miedo al bicho", abandono de diuréticos.
- Ha retomado el **consumo enólico** (problemas laborales). **Claudicación familiar.**

EXPLORACIÓN FÍSICA

Regular estado general. **Bradipsíquico . Desorientado en tiempo. Flapping +**. Eupneico. Afebril.

- ACR: Sin alteraciones
- ABD: Distendido con **ascitis a tensión.** Circulación colateral evidente. No doloroso. Sin masas palpables.
- MMII: Sin edemas
- Tacto rectal: Heces normales

EXPLORACIÓN FÍSICA



PRUEBAS COMPLEMENTARIAS

- Rx tórax y abdomen: Ascitis. Resto normal
- Analítica sanguínea: Hb 10.9 , PQ 90.000, L 4600. INR 1.9 Gluc 85, Urea 65, **Cr 2.4**, Na 130, K 4.4, AST 48, ALT 52, Bt 1.9 (1 directa) , Albúmina 2.5 , PCR 4
- Analítica orina: Sin alteraciones. Na 26
- EKG: Ritmo sinusal a 80lpm

PREGUNTA

¿Qué medidas llevarías a cabo?

1. Administraría 2 pool de plaquetas y plasma fresco antes de realizar paracentesis diagnóstica dada la plaquetopenia y coagulopatía.

2. Realizaría paracentesis diagnóstica y, una vez descartada PBE, llevaría a cabo PGV y reposición con albúmina.

3. No hay datos de infección en analítica ni el paciente presenta fiebre o dolor abdominal. Es seguro realizar paracentesis evacuadora directamente + reposición con albúmina.

PREGUNTA

¿Qué medidas llevarías a cabo?

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3. No hay datos de infección en analítica ni el paciente presenta fiebre o dolor abdominal. Es seguro realizar paracentesis evacuadora directamente + reposición con albúmina.

La **PGV extrae 12L** a pesar de ser incompleta ya que le paciente se retira el catéter por molestias.

- LA: Leuc 260 (85% L), Gluc 79, Prot 1.4 (0.8 albúmina), LDH y Amilasa normales. **GASA >1.1.** Celularidad negativa para malignidad.

PREGUNTA

Cirrosis descompensada CPT C11, MELD 24, MELD-Na 28.

Ascitis a tensión, EH-II y deterioro de la función renal

¿Qué actitud consideráis más acertada en el manejo del paciente?

1. Diuréticos a dosis plenas (400mg espironolactona + 160mg furosemida) para forzar diuresis. Enemas con lactulosa por turnos + oral . Profilaxis primaria PBE.

2. Es una ascitis refractaria. Paracentesis de gran volumen, repondría con albúmina y solicitaría TIPS. Añadiría lactulosa al tratamiento.

3. Paracentesis de gran volumen y reposición con albúmina a razón de 8gr/L drenado. Enemas con lactulosa por turnos + oral . Nada de diuréticos ni AINES. Profilaxis 1º PBE.

PREGUNTA

Cirrosis descompensada CPT C11, MELD 24, MELD-Na 28.

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Ascitis a tensión no complicada

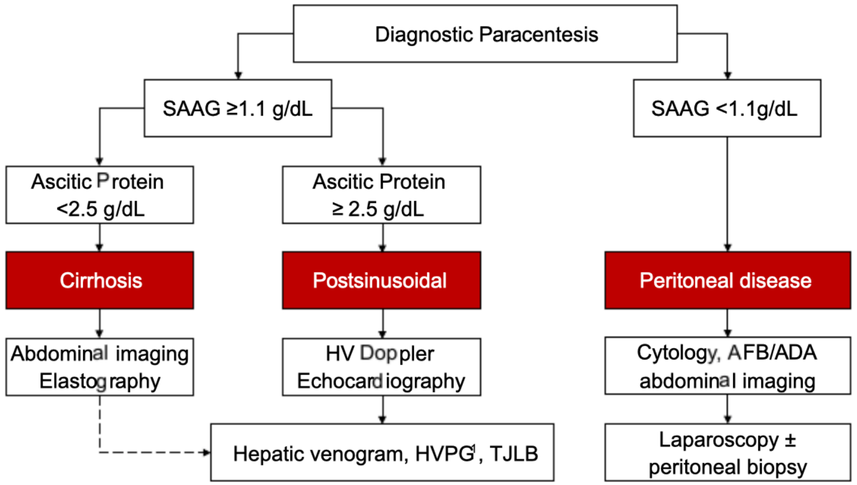

FIG. 2. Diagnostic approach to new-onset ascites. [1]May include right heart catheterization. Abbreviations: Abd, abdominal; ADA, adenosine deaminase; AFB, acid fast bacterium; HV, hepatic venous; SAAG, serum-ascites albumin gradient; TJLB, transjugular liver biopsy.

Diagnosis, Evaluation, and Management of Ascites, Spontaneous Bacterial Peritonitis and Hepatorenal Syndrome: 2021 Practice Guidance by the American Association for the Study of Liver Diseases

- LVP is the first-line therapy in patients with large ascites (grade 3 ascites), which should be completely removed in a single session **(I;1)**.
- LVP should be followed with plasma volume expansion to prevent PPCD **(I;1)**.
- In patients undergoing LVP of greater than 5 L of ascites, plasma volume expansion should be performed by infusing albumin (8 g/L of ascites removed), as it is more effective than other plasma expanders, which are not recommended for this setting **(I;1)**.
- In patients undergoing LVP of less than 5 L of ascites, the risk of developing PPCD is low. However, it is generally agreed that these patients should still be treated with albumin because of concerns about use of alternative plasma expanders **(III;1)**.

EASL Clinical Practice Guidelines for the management of patients with decompensated cirrhosis. J Hepatol. 2018

Ascitis no complicada

Diuréticos

Recommendations

- A moderate restriction of sodium intake (80–120 mmol/day, corresponding to 4.6–6.9 g of salt) is recommended in patients with moderate, uncomplicated ascites **(I;1)**. This is generally equivalent to a no added salt diet with avoidance of pre-prepared meals. Adequate nutritional education of patients on how to manage dietary sodium is also recommended **(II-2;1)**.
- Diets with a very low sodium content (<40 mmol/day) should be avoided, as they favour diuretic-induced complications and can endanger a patient's nutritional status **(II-2;1)**.
- Prolonged bed rest cannot be recommended because there is insufficient evidence that it is beneficial in the treatment of ascites **(III;1)**.

EASL Clinical Practice Guidelines for the management of patients with decompensated cirrhosis. J Hepatol. 2018

- **Anti-mineralcorticoide:** Espironolactona
  Inicio a 100mg/ día—> Incrementos cada 72h hasta 400mg/día. " Ojo-> **K**"
- **Diuréticos del asa: Furosemida.** Inicio a 40mg/día—-> 160mg/día. Útil en: Escasa respuesta diurética / hiperK
- **Respuesta adecuada**: Pérdida 0.5Kg/ día sin edemas o 1Kg/día con edemas

Ascitis no complicada

Recommendations

- A moderate restriction of sodium intake (80–120 mmol/day, corresponding to 4.6–6.9 g of salt) is recommended in patients with moderate, uncomplicated ascites **(I;1)**. This is generally equivalent to a no added salt diet with avoidance of pre-prepared meals. Adequate nutritional education of patients on how to manage dietary sodium is also recommended **(II-2;1)**.
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EASL Clinical Practice Guidelines for the management of patients with decompensated cirrhosis. J Hepatol. 2018

Diuréticos:

Contraindicaciones

Diuretics should be discontinued if severe hyponatraemia (serum sodium concentration <125 mmol/L), AKI, worsening hepatic encephalopathy, or incapacitating muscle cramps develop **(III;1)**.

Furosemide should be stopped if severe hypokalemia occurs (<3 mmol/L). Anti-mineralocorticoids should be stopped if severe hyperkalemia occurs (>6 mmol/L) **(III;1)**.

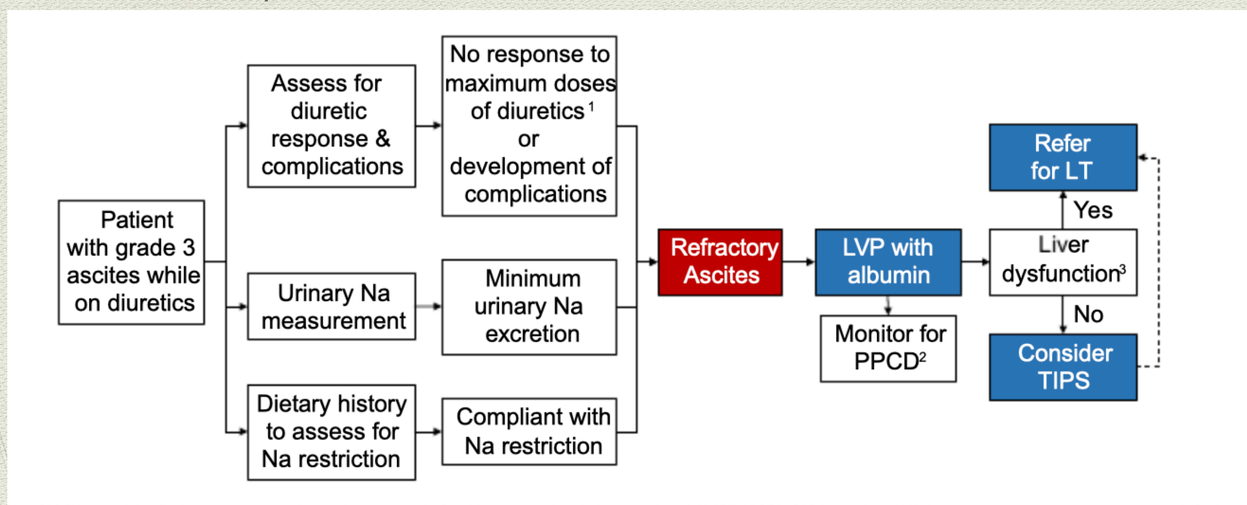
EASL Clinical Practice Guidelines for the management of patients with decompensated cirrhosis. J Hepatol. 2018

Ascitis refractaria

"Ascitis que no puede ser movilizada o que recurre rápidamente tras PGV a pesar de restricción de sodio y diuréticos"

Ascitis resistente a diuréticos: Persistencia de ascitis a pesar de diuréticos a dosis máximas y restricción salina

Ascitis intratable: Administrar /incrementar dosis de diuréticos induce efectos adversos



Diagnosis, Evaluation, and Management of Ascites, Spontaneous Bacterial Peritonitis and Hepatorenal Syndrome: 2021 Practice Guidance by the American Association for the Study of Liver Diseases

Ascitis refractaria

¿Infusiones de Albúmina?

Editorial > Liver Int. 2019 Jan;39(1):45-48. doi: 10.1111/liv.13996.

Long-term albumin administration in patients with decompensated cirrhosis. It is time for a reappraisal

Rafael Bañares [1] [2], Mauro Bernardi [3]

TABLE 1 Main features and results of controlled trials assessing the effects of long-term albumin administration in patients with decompensated cirrhosis

Author	Patient population	Study design/sample size	Primary end-point	MELD score (points)	Albumin dose	Mean/median follow-up	Main results	Comments
Di Pascoli et al[15]	Refractory ascites	Single centre Non-randomized consecutive series of patients N = 70 (45 albumin)	Mortality	14.9 (±) in albumin group 15.2 (±) in SMC group	20 g of albumin twice a week	Mean: 400.8 and 318.7 d in the albumin and SOC group	Increased survival in albumin-treated patients Decrease in hospitalization	No use of TIPS
Caraceni et al[16]	Cirrhosis and ascites requiring at least 200 mg/d of an antimineralocorticoid drug and 25 mg/d of furosemide	Multicentre open-label Pragmatic Clinical trial Randomized patients: N = 440 Modified ITT analysis: N = 431	Mortality	12 (10-15) in albumin group 13 (10-16) in SMT group	40 g twice a week for the initial 2 wk and 40 g weekly thereafter	Median[a]: 11.5 mo in the SMT group and 17.6 mo in the albumin group	Increased survival, reduced incidence of complications, amelioration of QoL in albumin-treated patients	
Solà et al[17]	Patients with decompensated cirrhosis included in the waitlist for liver transplantation	Double-blind placebo-controlled trial Randomized patients N = 196 Modified ITT analysis: N = 173	Incidence of complications of cirrhosis	17 (±) in albumin plus midodrine 16 (±) in SMC	40 g/15 d (plus midodrine adjusted to mean arterial pressure)	Median: 63 d in the albumin plus midodrine group and 105 d in the SMT group	No reduction in the incidence of cirrhosis complications No survival benefit	Rapid access to liver transplant in both arms

ITT, intention to treat; QoL, quality of life; SMT, standard medical treatment; SOC, standard of care.
[a]Calculated by reverse Kaplan-Meier method.

PREGUNTA

Cirrosis descompensada CPT C11, MELD 24, MELD-Na 28.

Ascitis a tensión, EH-II y **deterioro función renal**

¿Cómo definirías el deterioro de la función renal del paciente?

1. Seguramente ya llevaba varios meses deteriorada. Enfermedad renal crónica (CKD).
2. Daño renal agudo (AKI) estadio II
3. Estamos ante un Síndrome Hepatorrenal tipo I. Inicio terlipresina en perfusión y aviso a UCI.

PREGUNTA

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3. Estamos ante un Síndrome Hepatorrenal tipo I.

Definición de daño renal: KDIGO

Definition	Functional criteria	Structural criteria
AKI	Increase in sCr ≥50% within seven days, *or* increase in sCr ≥0.3 mg/dl within two days	No criteria
AKD	GFR <60 ml/min per 1.73 m^2 for <3 months, *or* decrease in GFR ≥35% for < 3 months, *or* increase in sCr ≥50 % for < 3 months	Kidney damage for <3 months
CKD	GFR <60 ml/min per 1.73 m^2 for ≥3 months	Kidney damage for ≥3 months

AKD, acute kidney disease; AKI, acute kidney injury; CKD, chronic kidney disease; GFR, glomerular filtration rate; sCr, serum creatinine.

Diagnosis and management of acute kidney injury in patients with cirrhosis: Revised consensus recommendations of the International Club of Ascites. Angeli P et. al. Journal of Hepatology 2015 vol: 62 (4) pp: 968-974.

Definición de daño renal: KDIGO

Table 7. International Club of Ascites (ICA-AKI) new definitions for the diagnosis and management of acute kidney injury in patients with cirrhosis.

Subject	Definition		
Baseline sCr	A value of sCr obtained in the previous three months, when available, can be used as baseline sCr. In patients with more than one value within the previous three months, the value closest to the admission time to the hospital should be used In patients without a previous sCr value, the sCr on admission should be used as baseline.		
Definition of AKI	- Increase in sCr ≥0.3 mg/dl (≥26.5 μmol/L) within 48 h; or, - A percentage increase sCr ≥50% which is known, or presumed, to have occurred within the prior seven days		
Staging of AKI	- Stage 1: increase in sCr ≥0.3 mg/dl (≥26.5 μmol/L) or an increase in sCr ≥1.5-fold to 2-fold from baseline; - Stage 2: increase in sCr >2-fold to 3-fold from baseline; - Stage 3: increase of sCr >3-fold from baseline or sCr ≥4.0 mg/dl (353.6 μmol/L) with an acute increase ≥0.3 mg/dl (≥26.5 μmol/L) or initiation of renal replacement therapy		
Progression of AKI	**Progression**	**Regression**	
	Progression of AKI to a higher stage and/or need for RRT	Regression of AKI to a lower stage	
Response to treatment	**No response**	**Partial response**	**Full response**
	No regression of AKI	Regression of AKI stage with a reduction of sCr to ≥0.3 mg/dl (≥26.5 μmol/L) above the baseline value	Return of sCr to a value within 0.3 mg/dl (≥26.5 μmol/L) of the baseline value

AKI, acute kidney injury; sCr, serum creatinine; RRT, renal replacement therapy.

- EASL Clinical Practice Guidelines for the management of patients with decompensated cirrhosis. Angeli P Bernardi M Villanueva C Francoz C Mookerjee R et. al. Journal of Hepatology2018 vol: 69 (2) pp: 406-460

Cirrosis descompensada CPT C11, MELD 24, MELD-Na 28.

Ascitis a tensión, EH-II y **AKI II**

¿Qué medidas llevarías a cabo?

1. Estudio de factores precipitantes y corrección de los mismos. Expansión con cristaloides y albúmina durante 24h. Estricto control de diuresis.

2. Hazme caso, es un Síndrome Hepatorrenal tipo I. Inicio terlipresina y aviso a UCI.

3. Estudio factores precipitantes y corrección de los mismos. Expansión solo con cristaloides.

4. Estudio de factores precipitantes y corrección de los mismos. Expansión con cristaloides y albúmina durante 48h. Estricto control de diuresis.

Cirrosis descompensada CPT C11, MELD 24, MELD-Na 28.

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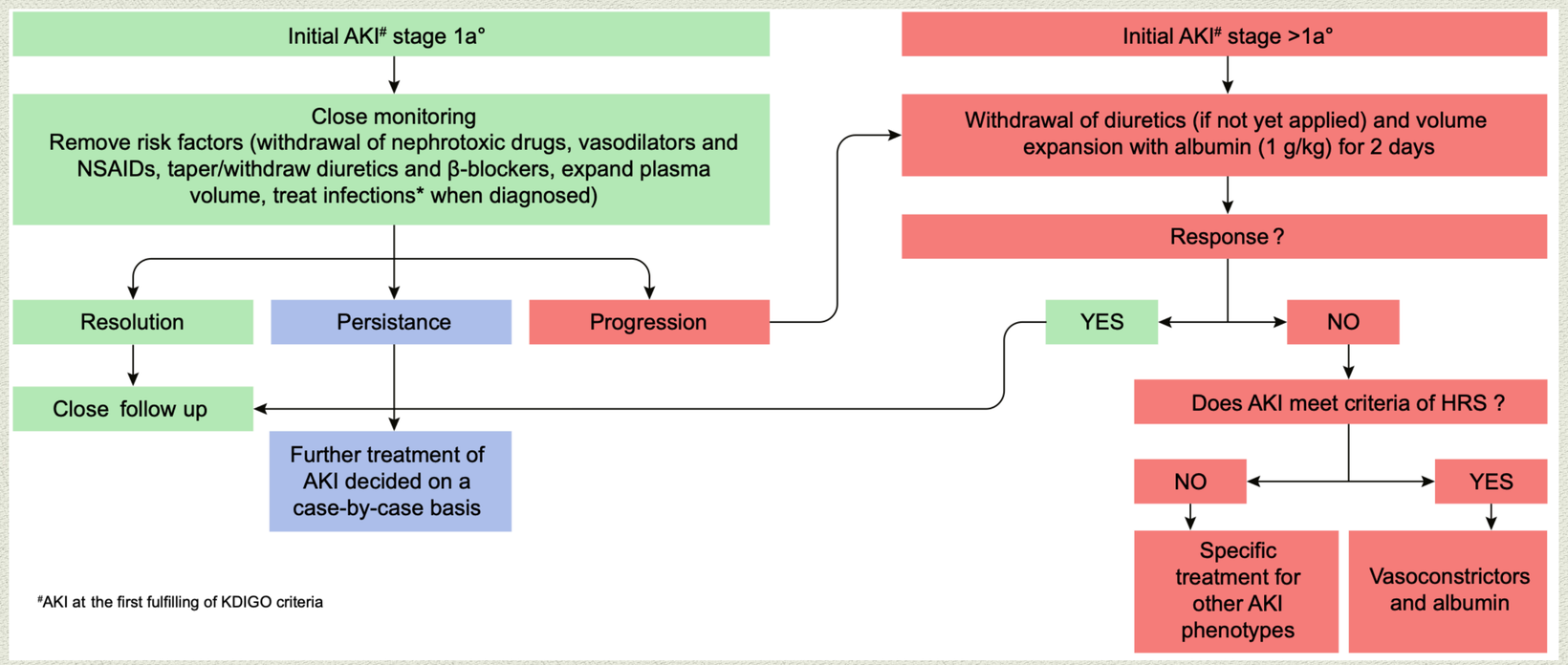
4. **Estudio de factores precipitantes y corrección de los mismos. Expansión con cristaloides y albúmina durante 48h. Estricto control de diuresis.**

Factores precipitantes

- Infecciones
- Diuresis excesivas por diuréticos
- Hemorragias GI
- Disfunción circulatoria post-paracentesis
- Fármacos nefrotóxicos



Adapted from Angeli P, et al. J Hepatol 2015;62:968–74; EASL CPG decompensated cirrhosis. J Hepatol 2018;doi: 10.1016/j.jhep.2018.0 3.024

EVOLUCIÓN DURANTE 48h

Resolución de la EH con lactulosa oral y enemas. Leve distensión abdominal a pesar de buenas diuresis (1500cc/día).

- **Analítica sanguínea control** a las 48h : Hb 10.7 (10.9), PQ 86.000, Leuc 7600(4600). INR 1.85 Gluc 93, Urea 52, **Cr 1.5 (2.4)**, Na 129, K 4.4, AST 51, ALT 52, Bt 1.9 (1.2 directa) , Albúmina 2.7 , **PCR 23**

- **EDA**: VVEE pequeñas sin estigmas de riesgo de sangrado. Gastropatía por HTP.

- **ECO abdomen:** Hígado, pequeño, abollonado, sin LOE´s. Porta permeable de 15mm con flujo medio 12cm/s. Bazo 170mm. **Moderada cuantía de líquido ascítico.**

4º Día ingreso

Avisa enfermería por fiebre de 37,8ºC, mayor sensación de distensión abdominal e indicando que el paciente se quiere quitar el sondaje vesical.

- A nuestra llegada el paciente se encuentra sin datos de EH refiriendo dolor en relación con la sonda y molestias abdominales leves desde esa mañana.
- Abdomen con ascitis moderada, no a tensión, molestias a la palpación sin datos de peritonismo.
- Orina clara en bolsa, mínimo resto hemático sin sedimento.

4º Día ingreso

Dada la exploración abdominal achaco la fiebre a un foco urinario y solicito analíticas de sangre y orina, hemocultivos/ urocultivos e inicio ceftriaxona empírica hasta ver el resultado del antibiograma…

4º Día ingreso

Dada la exploración abdominal achaco la fiebre a un foco urinario y anodina solicito analíticas de sangre y orina, hemocultivos/ urocultivos e inicio ceftriaxona empírica hasta ver el resultado del antibiograma…

¿Qué se ha hecho bien , qué se ha hecho mal y qué harías tú?

Fiebre en paciente cirrótico ingresado

Hacer **cribado completo** a pesar de creer tener foco localizado:

- Analítica completa con PCR y PCT / Hemocultivos
- Urianálisis /urocultivo
- Rx tórax y cultivo de esputo
- **Análisis de líquido ascítico y cultivo**

4º Día ingreso

Paracentesis diagnóstica

- LA: Leuc 832 (64% N), Gluc 65, Prot 1.3 (0.9 albúmina), LDH y Amilasa normales.

¿Cuál es la sospecha diagnóstica?

Peritonitis bacteriana espontánea

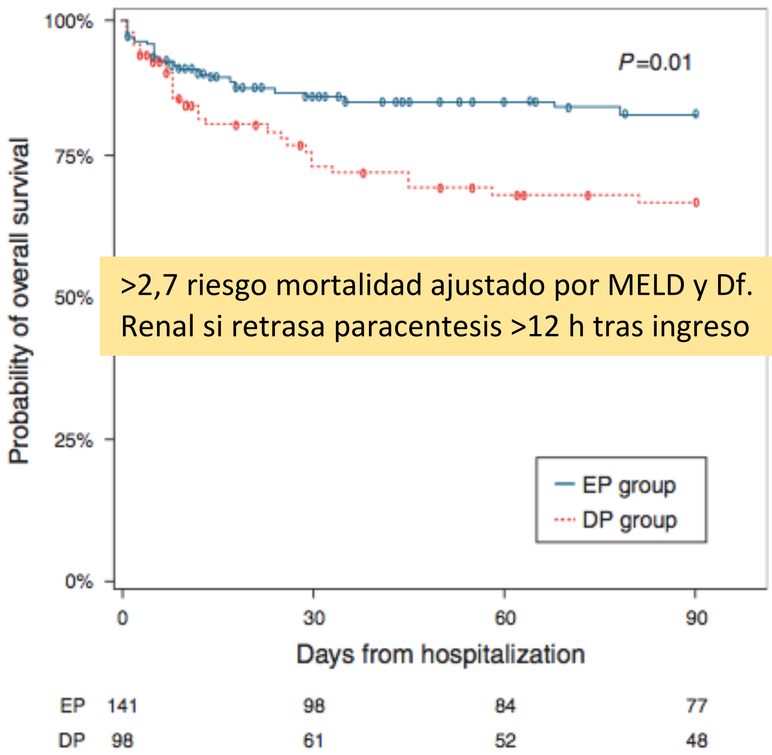
- Prevalencia 1,5 – 3,5% ambulatorios/ 10% hospitalizados.
- 50% en el momento ingreso.

Signos/síntomas peritonitis.
Datos respuesta inflamatoria sistémica.
Deterioro función hepática/ EH/HDA/shock/fracaso renal.

PBE suele ser ASINTOMÁTICA, sobre todo en pacientes ambulatorios

Peritonitis bacteriana espontánea

Recommendation		
Diagnostic paracentesis should be carried out in: • Patients with cirrhosis and ascites, **at admission**, to rule out SBP • Patients with GI bleeding, shock, fever or other signs of systemic inflammation, worsening liver and/or renal function, and HE	II-2	1
SBP diagnosed by a neutrophil count in ascitic fluid >250/mm^3 • Neutrophil count is determined by microscopy or flow cytometry • **No clear evidence** to support routine use of **reagent strips**	II-2	1
Ascitic fluid culture positivity is not a prerequisite for SBP diagnosis*	II-2	1



>2,7 riesgo mortalidad ajustado por MELD y Df. Renal si retrasa paracentesis >12 h tras ingreso

Figure 1. Interval to death in hospitalized patients with spontaneous bacterial peritonitis stratified by early (EP) vs. delayed paracentesis (DP).

EASL Clinical Practice Guidelines for the management of patients with decompensated cirrhosis. Angeli P Bernardi M Villanueva C Francoz C Mookerjee R et. al. Journal of Hepatology2018 vol: 69 (2) pp: 406-460
Delayed paracentesis is associated with increased in-hospital mortality in patients with spontaneous bacterial peritonitis.Kim et. al. The American journal of gastroenterology 2014 vol: 109 (9) pp: 1436-42.

PREGUNTA

¿Qué tratamiento antibiótico elegirías?

PREGUNTA

¿Qué tratamiento antibiótico elegirías?

1. Norfloxacino oral 400mg/12h durante 7 días
2. Ceftriaxona 1gr IV durante 7 días
3. Ciprofloxacino 400mg/12h + metronidazol 500mg/8h durante 7 días
4. Meropenem 1 gr/8h +/- Linezolid /daptomicina/vancomicina en función del patrón de resistencias del centro.

Peritonitis bacteriana espontánea

- Inicio inmediato desde el diagnóstico
- Evitar antibióticos potencialmente nefrotóxicos (aminoglucósidos)
- Adaptar el tratamiento a la prevalencia de bacterias **MDR/XDR** (**Nosocomial(>48))vs Asociada a cuidados sanitarios (<48h)**
- **Comprobación de la respuesta ATB mediante paracentesis a las 48h (día 3)**

Recommendation		
Third-generation cephalosporins are recommended as first-line antibiotic treatment for **community-acquired SBP** in countries with **low rates of antibiotic resistance**	I	1
• In countries with **high rates of antibiotic resistance** piperacillin/tazobactam or carbapenem should be considered	II-2	1
Antibiotic resistance is more likely in **healthcare-associated** and **nosocomial SBP** • Piperacillin/tazobactam: in areas with low prevalence of MDR bacteria • Carbapenem: in areas with high prevalence of ESBL-producing *Enterobacteriaceae* • Carbapenem + glycopeptides, daptomycin linezolid in areas with high prevalence of gram-positive MDR bacteria	I	1

EASL CPG decompensated cirrhosis. J Hepatol 2018;doi: 10.1016/j.jhep.2018.03.024

Peritonitis bacteriana espontánea

Table 4. Recommended empirical antibiotic treatment for community-acquired and nosocomial bacterial infections in cirrhosis.

Type of infection	Community-acquired infections	Nosocomial infections*
SBP, SBE and spontaneous bacteremia	Cefotaxime **or** ceftriaxone **or** amoxicillin/clavulanic acid	Piperacillin/tazobactam[Δ] **or** meropenem[§] ± glycopeptide[#]
Urinary infections	**Uncomplicated:** ciprofloxacin **or** cotrimoxazole **If sepsis:** cefotaxime **or** ceftriaxone **or** amoxicillin/clavulanic acid	**Uncomplicated:** nitrofurantoin or fosfomycin **If sepsis:** piperacillin/tazobactam[Δ] **or** meropenem[§] ± glycopeptide[#]
Pneumonia**	Amoxicillin/clavulanic acid **or** ceftriaxone + macrolide **or** levofloxacin **or** moxifloxacin	Piperacillin/tazobactam[Δ] **or** meropenem/ceftazidime + ciprofloxacin ± glycopeptide[#] should be added in patients with risk factors for MRSA[¶]
Cellulitis	Amoxicillin/clavulanic acid **or** ceftriaxone + oxacillin	Meropenem/ceftazidime[&] + oxacillin **or** glycopeptides[#]

SBP, spontaneous bacterial peritonitis; SBE, spontaneous bacterial empyema; MRSA, methicillin-resistant *Staphylococcus aureus*.
Dosages of antibiotics have not been formally investigated or defined in cirrhotic population and it is advisable to follow standard recommended dosages.
*Recommended empirical treatment also for health-care associate (HCA) urinary infections and pneumonia. Empirical antibiotic treatment of HCA spontaneous infections and cellulitis will be decided on the basis of the severity of infection (patients with severe sepsis should receive the schedule proposed for nosocomial infections) and on the local prevalence of multiresistant bacteria in HCA infections.
[Δ]In areas with a low prevalence of multiresistant bacteria.
[§]To cover extended-spectrum β-lactamase (ESBL)-producing *Enterobacteriaceae*.
[#]IV vancomycin or teicoplanin in areas with a high prevalence MRSA and vancomycin-susceptible enterococci (VSE). Glycopeptides must be replaced by IV linezolid in areas with a high prevalence of vancomycin-resistant enterococci (VRE).
**Liver disease is considered as severe comorbidity for community-acquired pneumonia in guidelines.
[&]Antibiotics active against *Pseudomonas aeruginosa*.
[¶]Ventilator-associated pneumonia, previous antibiotic therapy, nasal MRSA carriage.

Jalan, R et al. Bacterial infections in cirrhosis: A position statement based on the EASL Special Conference. J Hepatol. 2014 Jun;60(6):1310-24

Se inicia **Meropenem 1gr/8h mejorando el dolor** en 24h pero presentando **empeoramiento de la ascitis** y un importante **recorte de diuresis**

¿Qué ha podido pasar y por qué?

Peritonitis bacteriana espontánea

Uso de albúmina

Recommendation		
The administration of albumin is recommended in patients with SBP • 1.5 g/kg at diagnosis and • 1 g/kg on Day 3	I	1

¿Debe aplicarse a todos los pacientes con PBE o solo a los pacientes con alto riesgo de insuficiencia renal o mortalidad?

TABLE 2. CLINICAL OUTCOME ACCORDING TO THE ASSIGNED TREATMENT.*

OUTCOME VARIABLE	CEFOTAXIME (N=63)	CEFOTAXIME PLUS ALBUMIN (N=63)	P VALUE
Resolution of infection — no. (%)†	59 (94)	62 (98)	0.36
Duration of antibiotic therapy — days	6±1	5±1	0.48
Paracentesis for ascites after resolution of infection — no. (%)‡	16 (25)	14 (22)	0.83
Hospital stay — days	13±1	14±1	0.48
Renal impairment — no. (%)	21 (33)	6 (10)	0.002
Death — no. (%)			
In hospital§	18 (29)	6 (10)	0.01
At three months¶	26 (41)	14 (22)	0.03

*Plus–minus values are means ±SE.

†The infection resolved with the initial cefotaxime therapy in 53 of the 63 patients (84 percent) in the cefotaxime group and in 57 of the 63 patients (90 percent) in the cefotaxime-plus-albumin group. In the other patients, the infection resolved after modification of the antibiotic therapy.

‡These patients required at least one therapeutic paracentesis for the management of ascites.

§The causes of in-hospital death were combined liver and renal failure (13 patients in the cefotaxime group and 5 in the cefotaxime-plus-albumin group), massive gastrointestinal hemorrhage (2 patients in the cefotaxime group and 1 in the cefotaxime-plus-albumin group), septic shock (2 patients in the cefotaxime group), and liver failure (1 patient in the cefotaxime group).

¶Seven patients (four in the cefotaxime group and three in the cefotaxime-plus-albumin group) were lost to follow-up after discharge from the hospital. The three-month mortality rates were calculated as the number of known deaths at this time divided by the total number of enrolled patients in each group.

- Effect of Intravenous Albumin on Renal Impairment and Mortality in Patients with Cirrhosis and Spontaneous Bacterial Peritonitis
Sort P et. al. New England Journal of Medicine1999 vol: 341 (6) pp: 403-409
- EASL Clinical Practice Guidelines for the management of patients with decompensated cirrhosis. Angeli P Bernardi M Villanueva C Francoz C Mookerjee R et. al. Journal of Hepatology2018 vol: 69 (2) pp: 406-460

Peritonitis bacteriana espontánea

APT Alimentary Pharmacology and Therapeutics

Review article: spontaneous bacterial peritonitis – bacteriology, diagnosis, treatment, risk factors and prevention

J. B. Dever* & M. Y. Sheikh†

41%, $P = 0.03$).[124] Renal impairment occurs in one-third of patients with SBP, and albumin is not indicated for all patients. Patients should be carefully screened to receive albumin infusion, because those at risk for renal impairment [i.e. serum creatinine > (1 mg/dL = 88.4 μmol/L), bilirubin > (4 mg/dL = 68.4 μmol/L), BUN > 30 mg/dL] have clearly shown benefit.[125] Patients with chronic kidney

Dever, APT 2015

Se recomienda en pacientes con bilirrubina ≥68lmol/L (4mg/dl) o SCr ≥88 lmol/L (1 mg/dl) o urea ≥ 11 mmol/L.

Intravenous albumin in patients with spontaneous bacterial peritonitis. SBP without septic shock may precipitate deterioration of circulatory function with severe liver failure, hepatic encephalopathy, and type 1 HRS and has approximately 20% hospital mortality despite infection resolution.[272] A randomised, controlled study in patients with SBP treated with cefotaxime showed that albumin (1.5 g/kg body weight at diagnosis, followed by 1 g/kg on day three) significantly decreased the incidence of type 1 HRS (from 30% to 10%) and reduced mortality from 29% to 10% compared with cefotaxime alone. Treatment with albumin was particularly effective in patients with baseline serum bilirubin ≥68 μmol/L (4 mg/dl) or SCr ≥88 μmol/L (1 mg/dl). It is unclear whether i.v. albumin is useful in patients with baseline bilirubin <68 μmol/L and creatinine <88 μmol/L, as the incidence of type 1 HRS in patients meeting these criteria was very low in the two treatment groups (7% without albumin and 0% with albumin).[272] The application of the schedule of this therapeutic option should be implemented in clinical practice.[273] Non-randomised studies in patients with SBP also show that the incidence of renal failure and death are very low in patients with moderate liver failure and without renal dysfunction at diagnosis of SBP, so albumin is probably not necessary.[274]

PREGUNTA

¿Realizarías paracentesis de grandes volúmenes a un paciente con PBE?

PREGUNTA

¿Realizarías paracentesis de grandes volúmenes a un paciente con PBE?

Randomized Controlled Trial > J Gastroenterol Hepatol. 2005 Aug;20(8):1215-22.
doi: 10.1111/j.1440-1746.2005.03861.x.

Long-term clinical outcome of large volume paracentesis with intravenous albumin in patients with spontaneous bacterial peritonitis: a randomized prospective study

Chang Hwan Choi [1], Sang Hoon Ahn, Do Young Kim, Sang Kil Lee, Jeong Youp Park, Chae Yoon Chon, Young Myoung Moon, Kwang-Hyub Han

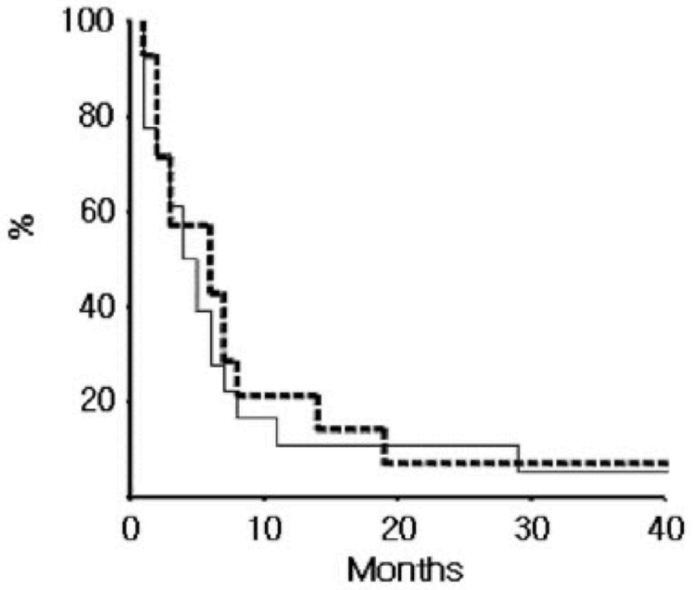


Figure 2 Cumulative probability of survival after entry to the study in patients with liver function of Child–Pugh class C. Eighteen patients treated with large volume paracentesis (Group 1, continuous line) and 14 patients treated with diuretics (Group 2, dotted line) were analyzed. Comparison with log-rank test showed no difference in cumulative survivals between the two groups ($P = 0.60$).

Mensajes para llevar a casa

- Los pacientes con cirrosis tienen un importante compromiso a múltiples niveles (hemodinámico, inmunitario…)
- Las complicaciones son múltiples y en muchas ocasiones superpuestas
- Prevenirlas es un elemento fundamental en el cuidado de estos paciente
- Alto índice de sospecha clínica cuando aparecen: Retrasar diagnóstico o tratamiento impacta en la supervivencia de estos pacientes.

MUCHAS GRACIAS