

DIPLOMADO EN NUTRICIÓN CLÍNICA

TEMA: INTERVENCIÓN NUTRICIONAL EN PATOLOGÍA
HEPÁTICA

MNA MARIANA ARÉCHIGA CARRILLO ARELLANO

22 DE JUNIO DE 2024



Intervención nutricional en hígado graso

Intervención nutricional en cirrosis

Intervención nutricional en hepatitis

Caso clínico



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Enfermedad de hígado graso no alcohólico (EHGNA)



Esteatosis Hepática Metabólica (MASLD)



Incluye a pacientes que tienen esteatosis hepática y al menos uno de 5 factores de riesgo metabólico



Exceso de peso

IMC ≥ 25 kg /m² o
Circ. Cint
 ≥ 94 cm (H) o ≥ 80 cm (M)



Alteración en glucosa

Ayuno >100 mg/dL
2 horas después de comer >200 mg/dL
HbA1c >5.7 %
Diagnóstico o tratamiento para diabetes



Presión arterial

$>130/85$ o
Tratamiento para hipertensión

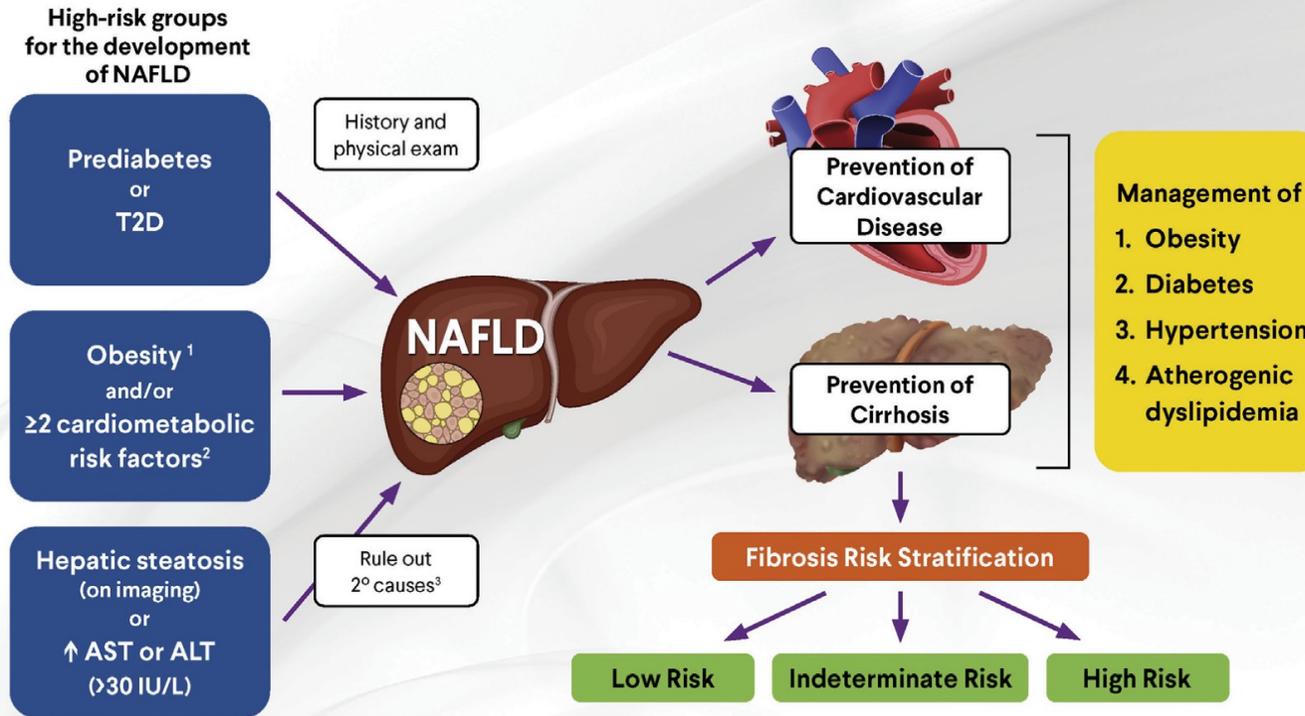


Lípidos

TG > 150 mg/dL o tratamiento
HDL < 40 mg/dL (H / <50 mg/dL (M))



Management Algorithm for NAFLD – Overview



Abbreviations: ALT = Alanine aminotransferase, AST = Aspartate aminotransferase, T2D = Type 2 diabetes mellitus

1. Adiposity-based chronic disease (ABCD) is a diagnostic term proposed by AACE to better describe the disease of obesity in a complication-centric manner of abnormal adipose tissue mass, distribution, function and resulting morbidity that can be ameliorated with weight loss.

2. Cardiometabolic risk factors of the metabolic syndrome are waist circumference >40 inches men >35 inches women, triglycerides ≥150 mg/dL, HDL-C <40 mg/dL men, <50 mg/dL women, BP ≥130/≥85 mm Hg, fasting plasma glucose ≥100 mg/dL (NCEP ATP III)

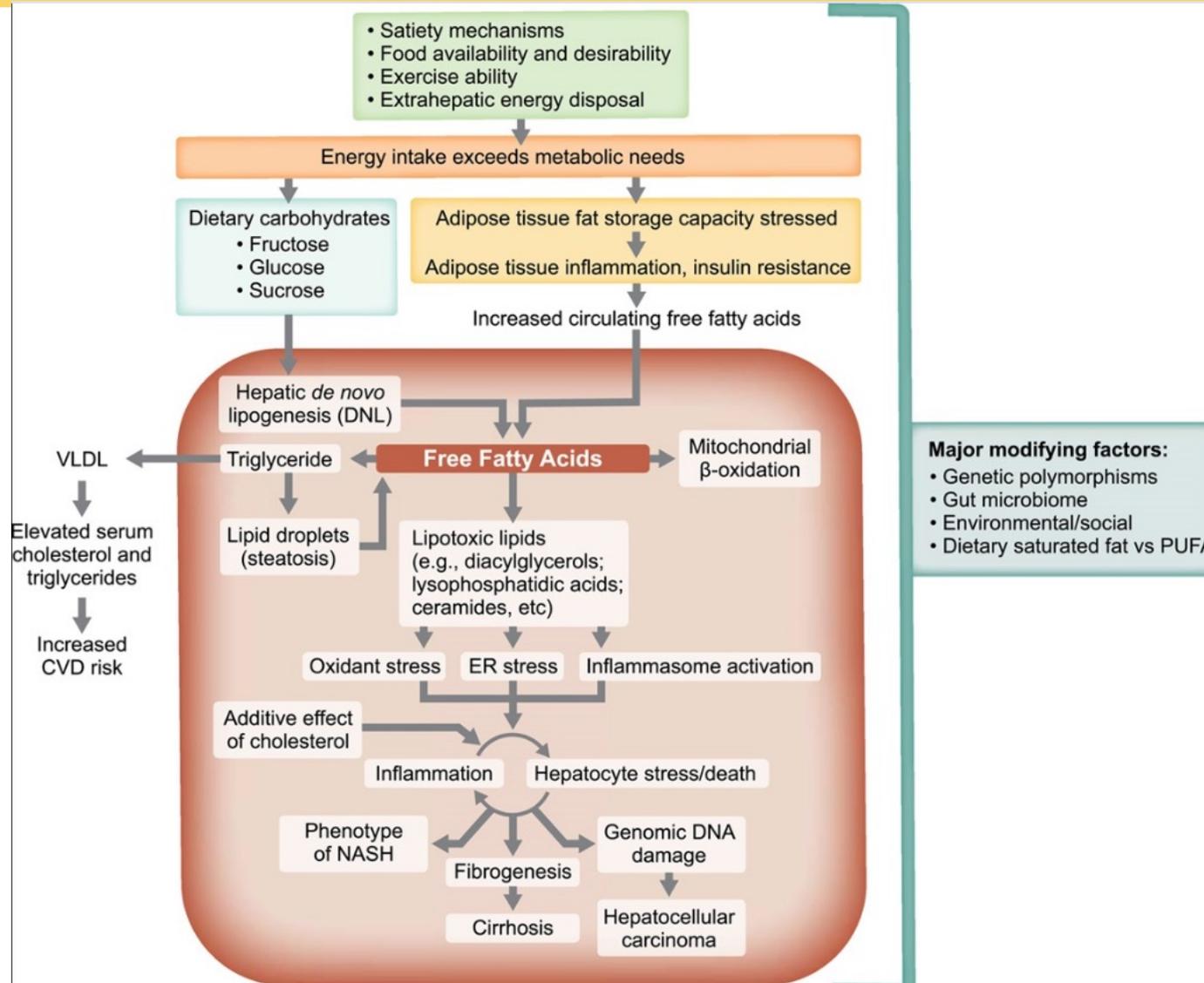
3. Secondary causes of liver steatosis or elevated transaminases (AST or ALT) are excessive alcohol consumption (≥14 drinks/week for women or ≥21 drinks/week for men), hepatitis B, hepatitis C (genotype 3), Wilson's disease, alpha 1 antitrypsin deficiency, lipodystrophy, starvation, parenteral nutrition, abetalipoproteinemia, hemochromatosis, mass lesions, medications and other causes.

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Algorithm Figure 1



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Intervención nutricional *Pérdida de peso*

5%

- Sensibilidad a la insulina
- Transaminasas
- Perfil de lípidos

≥7%

- Cambios histológicos
- Grado de esteatosis
- Balonización
- Inflamación

10%

- Remisión de la esteatohepatitis hasta en el 90% de los pacientes
- Reducción de fibrosis en el 45%



Intervención nutricional

Tipo de dieta

Energía

- Reducción del 25% de la energía habitual.

Distribución

- Baja en Hidratos de carbono (50-60%) en Diabetes
- Baja en lípidos (25-25%) en dislipidemia

Patrones

- Dieta keto: Reducción en la grasa del hígado, inflamación y fibrosis en el 80%
- Dieta mediterránea: Reducción en grasa hepática



Tratamiento de pérdida de peso en la NAFLD

Fibrosis Risk Stratification

<p>Low Risk</p> <p>FIB-4: <1.3 LSM <8 kPa ELF <7.7</p>	<p>Indeterminate Risk</p> <p>FIB-4: 1.3 - 2.67 LSM 8 - 12 kPa ELF 7.7 - 9.8</p>	<p>High Risk</p> <p>FIB-4: >2.67 LSM >12 kPa ELF >9.8</p>
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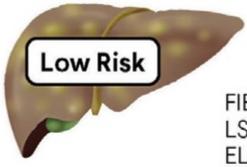
General lifestyle changes	Decrease sedentary time and increase daily movement. Stress reduction through exercise and other methods.		
Dietary recommendations	Creating an energy deficit is the priority with reduction of saturated fat, starch, & added sugars. Persons with cirrhosis need an individualized nutritional assessment and treatment plan.		
Exercise	To improve cardiometabolic health, support weight loss and mitigate sarcopenia. Aerobic exercise for 30-60 min (3-5 days/week) + resistance training 20-30 min (2-3 times/week).		
Alcohol intake	Minimize	Minimize	Avoid if F3 or cirrhosis (F4) ¹
Weight loss goal to treat NAFLD (if overweight or obesity) ²	Greater weight loss associated with greater liver and cardiometabolic benefit.		
Weight loss tools	Behavioral modification counseling. In person or remote programs.	Greater intensity of weight loss to reverse steatohepatitis and fibrosis.	Specialized obesity management, with a structured program, anti-obesity medications, bariatric surgery.
Medical therapy to treat obesity	Phentermine, phentermine/topiramate ER, naltrexone/bupropion, orlistat, liraglutide 3 mg/d, semaglutide 2.4 mg/wk	GLP-1 RA preferred for NASH. ^{3,4}	GLP-1 RA preferred for NASH. ^{3,4}
Bariatric surgery	Consider to treat obesity and comorbidities.	Strong consideration to treat steatohepatitis and fibrosis.	Stronger consideration to treat steatohepatitis and fibrosis. Avoid in decompensated cirrhosis.

Abbreviations: GLP-1 RA = Glucagon-like peptide-1 receptor agonists, HCC = Hepatocellular carcinoma, NASH = Nonalcoholic steatohepatitis

- Persons with confirmed cirrhosis based on biopsy or high likelihood based on LSM >13.6kPa from vibration controlled transient elastography (FibroScan®), ELF ≥9.8 or >5.0 kPa on MRE) should undergo HCC surveillance. Varices screening is recommended if LSM >20 kPa or platelet count of <150,000/mm³.
- These goals should only be taken as a broad guidance. NAFLD/NASH may also improve by changes in macronutrient content, exercise and other factors beyond magnitude of weight loss. All high-quality studies available limited to a maximum of 12 month duration.
- No high-quality evidence for pharmacotherapy in persons with NASH cirrhosis. Treatment should be individualized and used with caution only by liver specialists.
- Among GLP-1 RAs, semaglutide has the best evidence of benefit in persons with steatohepatitis and fibrosis.

Tratamiento de la diabetes en la NAFLD

Fibrosis Risk Stratification

	 <p>Low Risk</p> <p>FIB-4: <1.3 LSM <8 kPa ELF <7.7</p>	 <p>Indeterminate Risk</p> <p>FIB-4: 1.3 - 2.67 LSM 8 - 12 kPa ELF 7.7 - 9.8</p>	 <p>High Risk¹</p> <p>FIB-4: >2.67 LSM >12 kPa ELF >9.8</p>
General goal	Optimize glycemic control using preferred agents that reverse steatohepatitis, whenever possible. Prefer GLP-1 RA and SGLT2i in CVD. Prefer SGLT2i in CKD and HF.		
Dietary recommendations	Glycemic load reduction via emphasis on whole food carbohydrates (vegetables, legumes, fruit) versus sugar/processed carbohydrates.		
Individualize A1c target	≤6.5% for persons without concurrent serious illness and at low hypoglycemic risk (≥6.5% otherwise).		In advanced cirrhosis ¹ , caution with risk of hypoglycemia and avoid oral agents ²
Preferred diabetes pharmacotherapy	Consider agents that reduce liver fat (pioglitazone, GLP-1 RA, SGLT2i).	Strongly consider agents with efficacy in NASH: Pioglitazone and/or GLP-1 RA ³ . No evidence that SGLT2i improve steatohepatitis.	Strongly consider agents with efficacy in NASH: Pioglitazone and/or GLP-1 RA ³ . No efficacy data in cirrhosis.
Metformin, sulfonyleurea, DPP-4i, acarbose and insulin	May continue but limited benefit on liver histology in NAFLD.	May continue but limited benefit on liver histology in NAFLD.	May continue (F2-F3) but avoid oral agents if advanced cirrhosis present. Cannot avoid insulin in patients with advanced liver cirrhosis – often only option

Abbreviations: CKD = Chronic kidney disease, CVD = Cardiovascular disease, DPP-4i = Dipeptidyl peptidase 4, GLP-1 RA = Glucagon-like peptide-1 receptor agonists, HF = Heart failure, NASH = Nonalcoholic steatohepatitis, SGLT2i = Sodium-glucose cotransporter-2 inhibitors.

1. Advanced cirrhosis is defined as persons with cirrhosis based on biopsy and Child class B or C with clinical evidence of comorbidities (varices, portal hypertension, ascites, etc.).

2. Limited data on oral diabetes medications and GLP-1 RA in persons with cirrhosis. Avoid metformin, GLP-1 RA appear safe, insulin preferred. Avoid oral agents in advanced cirrhosis.

3. Among GLP-1 RAs, semaglutide has the best evidence of benefit in persons with steatohepatitis and fibrosis.

Tratamiento de la hipertensión en la NAFLD

Fibrosis Risk Stratification

	 <p>Low Risk</p> <p>FIB-4: <1.3 LSM <8 kPa ELF <7.7</p>	 <p>Indeterminate Risk</p> <p>FIB-4: 1.3 – 2.67 LSM 8 – 12 kPa ELF 7.7 – 9.8</p>	 <p>High Risk¹</p> <p>FIB-4: >2.67 LSM >12 kPa ELF >9.8</p>
General goal	Optimize BP control and improve cardiovascular health using preferred agents, whenever possible. Assess every 3 months and intensify therapy until goal achieved.		
Goal (individualize) ^{2,3,4}	Systolic < 130 mm Hg / Diastolic < 80 mm Hg	Systolic < 130 mm Hg / Diastolic < 80 mm Hg	Systolic < 130 mm Hg / Diastolic < 80 mm Hg; individualize if decompensated cirrhosis
Dietary recommendations	In addition to general dietary recommendations, reduce sodium & increase high potassium foods (e.g., DASH diet).		
Pharmacotherapy for hypertension ⁵	First-line therapy: ACEIs and ARBs.	First-line therapy: ACEIs and ARBs.	Same but avoid ACEI or ARB if decompensated cirrhosis.
Intensification of therapy	Second agent: CCB, BB ⁶ or thiazide diuretic (as additional agents as needed).		Same but individualize if decompensated cirrhosis. Use diuretics with caution (risk of excessive diuresis).
Additional options	Additional BP medication choices: alpha blockers, central agents, vasodilators, aldosterone antagonist.		Same but individualize if decompensated cirrhosis.

Abbreviations: ACEIs = Angiotensin-converting enzyme inhibitors, ARBs = angiotensin II receptor blockers, BB = beta blockers, CCB = calcium channel blockers.
 1. Advanced cirrhosis defined as persons with cirrhosis based on biopsy and Child class B or C and clinical evidence of comorbidities (varices, portal hypertension, ascitis, etc.).
 2. AACE recommends that BP control be individualized, but that a target of <130/80 mm Hg is appropriate for most persons.
 3. Less-stringent goals may be considered for frail persons with complicated comorbidities or those who have adverse medication effects.
 4. A more intensive goal (e.g., <120/80 mm Hg) should be considered for some persons if this target can be reached safely without adverse effects from medication.
 5. If initial BP > 150/100 mm Hg start with dual therapy. (ACEI or ARB + CCB, BB or thiazide diuretic).
 6. Prefer weight neutral beta-blockers: carvedilol, nebivolol.

Tratamiento de la dislipidemia aterogénica en la NAFLD

Lipid risk levels are similar in the presence of NAFLD or NASH

General goal	Early intensive management of dyslipidemia needed to reduce cardiovascular risk. Intensify therapy until lipid goal is reached.		
Dietary recommendations	Increase fiber intake (>25 g/d), prioritize vegetables, fruits whole grains, nuts, reduce saturated fat & added sugars (e.g., Mediterranean diet).		
Lipid risk levels	High CV Risk¹ ≥2 risk factors and 10-year risk 10–20% Diabetes or CKD ≥3 with no other risk factors	Very high CV Risk¹ Established CVD or 10-year risk >20% Diabetes with >1 risk factor, CKD ≥3, HeFH	Extreme CV Risk¹ Progressive CVD CVD + diabetes or CKD ≥3 or HeFH FHx premature CVD (<55 yrs male <65 yrs female)
LDL-C goal (mg/dL)	<100	<70	<55
Non-HDL-C goal (mg/dL)	<130	<100	<80
Triglycerides goal (mg/dL)	<150	<150	<150
Apo B goal (mg/dL)	<90	<80	<70
First line pharmacotherapy: Statins	Use a moderate-to-high intensity statin ² , unless contraindicated. Statins are safe in NAFLD or NASH but do not use in decompensated cirrhosis (Child C).		
If LDL-C not at goal ³ : Intensify statin therapy	Use higher dose or higher potency statin.		
If LDL-C not at goal (or statin intolerant) ⁴ : add 2nd agent, then add 3rd agent	Ezetemibe, PCSK9 inhibitor, bempedoic acid, colesevelam, inclisiran.		
If triglycerides > 500 mg/dL	Fibrates, Rx grade omega 3 FA, icosapent ethyl (if diabetes, optimize glycemic control and consider pioglitazone). ⁵		
If TG 135–499 mg/dL on max statin dose	Emphasize diet (as above).	Add icosapent ethyl. ⁶	Add icosapent ethyl. ⁶

Adapted from Handelsman Y, et al. Endocr Pract. 2020;26:1196-1224.

Abbreviations: CKD = Chronic kidney disease, CVD = cardiovascular disease, FA = Fatty acids, HeFH = Heterozygous familial hypercholesterolemia, HTN = Hypertension, Rx = Prescription

1. Major risk factors: age >40, DM, HTN, FHx of early CVD, low HDL C, elevated LDL, Smoking, CKD 3,4
2. High intensity statin therapy: rosuvastatin 20, 40 mg/d, atorvastatin 40, 80 mg/d.
3. Other lipid modifying agents should be used in combination with maximally tolerated statins if goals not reached: ezetimibe, PCSK9 inhibitor, bempedoic acid, colesevelam, or inclisiran.
4. Assess adequacy and tolerance of therapy with focused laboratory evaluations and patient follow up.
5. Niacin may lower triglycerides but does not reduce CVD and worsens insulin resistance. It may promote hyperglycemia in a population at high-risk of diabetes.
6. Icosapent ethyl 4g/d is recommended as an adjunct to maximally tolerated statin therapy to reduce risk of cardiovascular disease in high-risk persons.

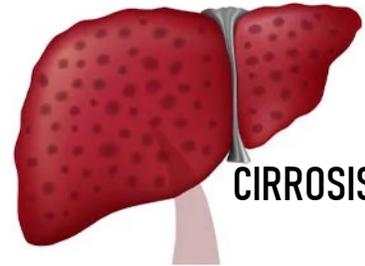
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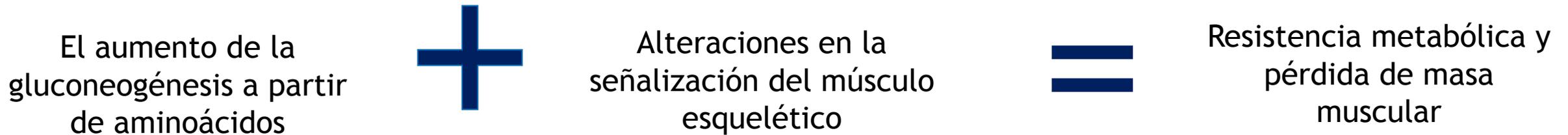
22 DE JUNIO DE 2024





Compensada: asintomática o síntomas específicos

Estado de inanición acelerada con alteración de la síntesis proteica



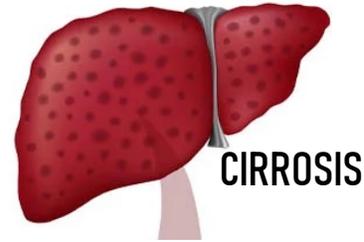
Depleción de masa grasa y magra

Retención de líquidos y sodio

Depleción de potasio, magnesio, zinc

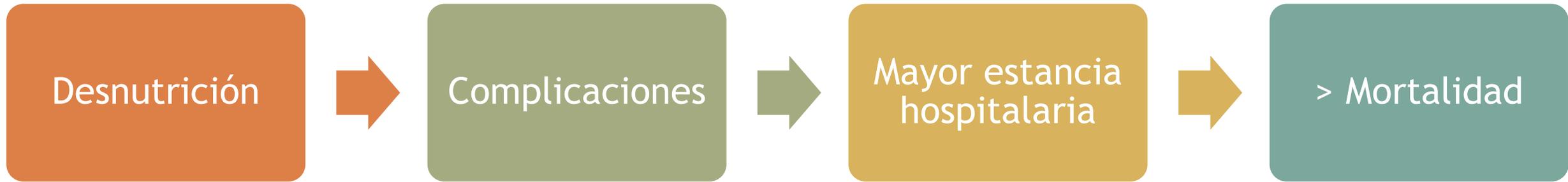
Deficiencia de vitaminas hidrosolubles y liposolubles





Compensada:
Asintomática o síntomas específicos
Prevalencia de desnutrición 20%

Descompensada
Complicaciones derivadas de la hipertensión portal (Ascitis, sangrado variceal, encefalopatía hepática)
Prevalencia de desnutrición 60%



Objetivos de la terapia nutricional:

Compensada:
Mejorar el estado nutricional

Descompensada:
Mantener la masa muscular
Disminuir la tasa de desnutrición

GASTO ENERGÉTICO

Cirrosis compensada:
Igual al de adultos
sanos

Cirrosis descompensada
o pacientes
desnutridos:
Gasto energético
incrementado

Pacientes con obesidad:
No se recomienda
incrementar ingesta

35-40 kcal/ kg / día
Sin ascitis: peso actual
Con ascitis: peso ideal

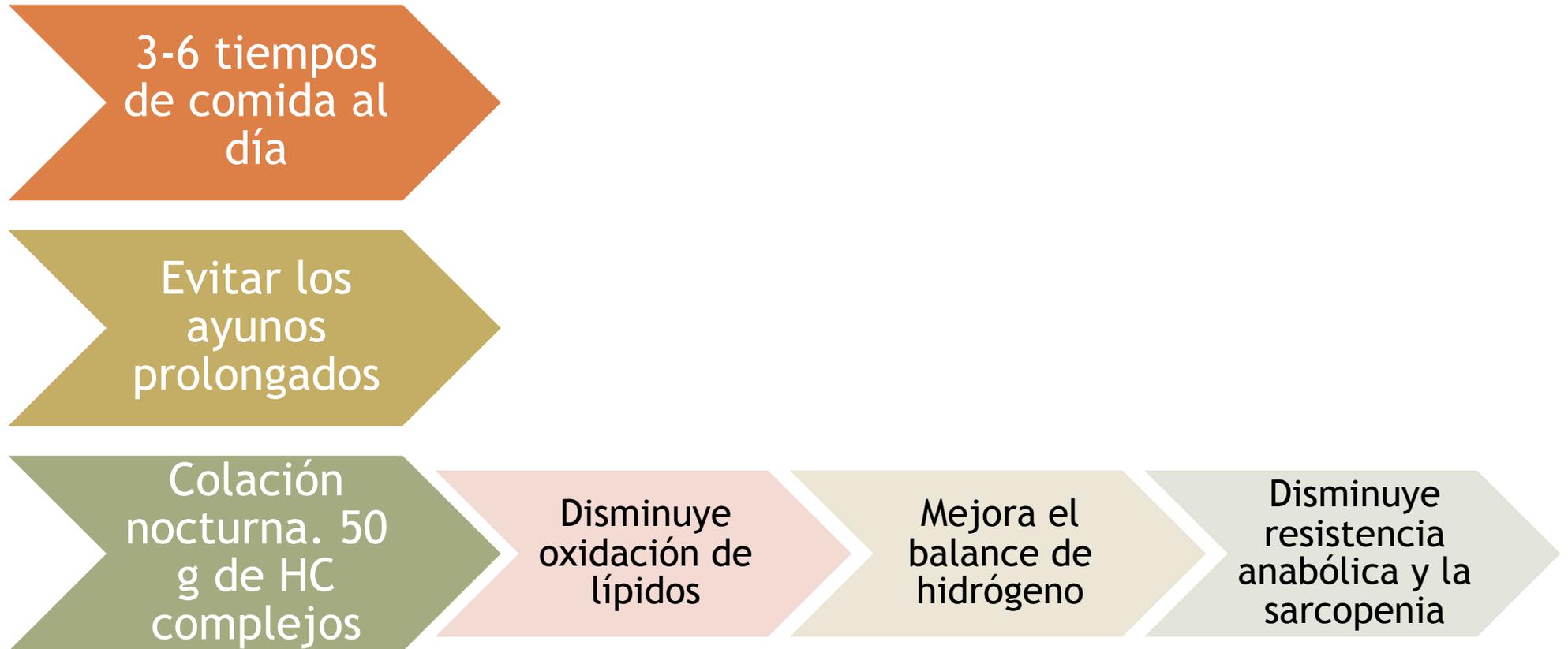
DISTRIBUCIÓN DE MACRONUTRIMENTOS EN COMPLICACIONES

ENCEFALOPATÍA HEPÁTICA	
Proteína	1.2-1.5g/kg/día 30-40% origen animal 60-70% origen vegetal
Aminoácidos de cadena ramificada	0.25g /kg / d
Fibra 25-25g	Con lactulosa 25 g Sin lactulosa 45 g

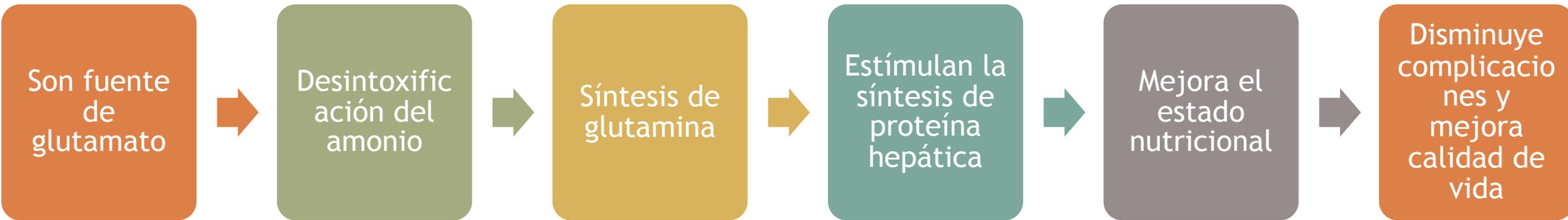
ASCITIS	
SODIO	1.2-1.5g/kg/día 30-40% origen animal 60-70% origen vegetal
Aminoácidos de cadena ramificada	0.25g /kg / d
Fibra 25-25g	Con lactulosa 25 g Sin lactulosa 45 g



de tiempos de comida



Aminoácidos ramificados Valina, leucina e isoleucina



Suplementación 0.25 g/ kg / día
Cirrosis avanzada

Incremento de aminoácidos aromáticos como consecuencia del hipercatabolismo y la detoxificación del músculo esquelético.



Otros nutrimentos

Vitamina D

- Suplementar en paciente con valores $<20\text{mg/ml}$
- $>30\text{ mg/ml}$

B1, B6, B9 y B12

- Podría considerarse en pacientes descompensados

Zinc y vit A

- Disminuye disgeusia
- Mejora la ingesta de alimentos



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Aumento de la ingesta energética a través de comida pequeñas y frecuentes

Restricción de la ingesta de sodio

Restricción de líquidos

Regímenes alimenticios con cantidades controladas de hidratos de carbono

Complementos vitamínicos y minerales

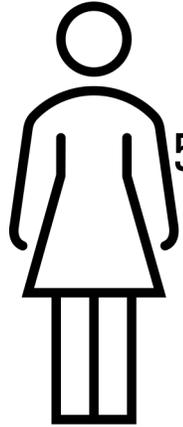
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51 AÑOS DE EDAD
AMA DE CASA

ANTECEDENTES PATOLÓGICOS

Dx de Hipotiroidismo desde
hace 3 años

Esteatosis hepática desde
hace 6 meses

Cirugías: apendicectomía,
hernia hiatal, vesícula.

Sin alergias alimentarias

CLÍNICOS

Estreñimiento

Inflamación intestinal

Levotidoxina ½ tableta

I ANTROPOMÉTRICOS

Estatura: 1.64m

Peso: 120.4kg

%Grasa 55%

Cintura 120cm

Cadera 138 cm

Peso mínimo: 90kg

Peso máximo: 123

I BIOQUÍMICOS

Glucosa 115 mg/dL

TG 150 mg/dL

Colesterol total 201 mg/dL

Colesterol HDL 35 mg/dL

Bilirubina D 0.3

Bilirubina I 0.9

AST 40

ALT 50

I ESTILO DE VIDA

Poco movimiento, dolor de
rodilla

I DIETÉTICOS

Desayuno 12:00

1 taza de café + 1 cda de
azúcar + 1 pieza de pan dulce

Almuerzo 1:00

½ taco de guisado (2 tortillas
de maíz + ½ taza de arroz rojo
+ pechuga empanizada (250 g)
+ 600ml de coca cola

Comida 18:00

1 hamburguesa de res con
queso amarillo + 1 orden de
papas + 600 ml de coca cola

Merienda 21:00

5 taquitos de chuleta con 2
tortillas + 600ml de cocoa cola

INSTRUCCIONES:

Revisa con detenimiento la información presentada sobre el paciente, desarrolla la evaluación /interpretación del estado de nutrición y responde:

¿Cuál es el principal problema nutricional de la paciente?

¿Qué abordaje nutricional tendrías con la paciente?

¿Calcula el requerimiento total de la paciente?

¿Qué tipo de patrón alimentario indicarías a la paciente?

¿Agregarías algún suplemento?

¿Qué consejo(s) nutricios darías a la paciente?

¿Qué resultados esperarías ver en tu siguiente consulta con la paciente?



CONCLUSIONES

El tratamiento nutricional participará de manera activa para disminuir el riesgo de desarrollar complicaciones.

El tratamiento nutricional debe adaptarse de acuerdo con el estadio de la condición y con la presencia de complicaciones

Satisfacer el requerimiento energético será un pilar fundamental. Distribuir la energía en más de 3 tiempos de comida y una colación nocturna reducirá el riesgo de hipoglucemias.

Preferir proteínas con aminoácidos ramificados será la mejor selección para prevenir encefalopatías.

