Candidiasis Invasiva en el neonato

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Candidiasis invasiva:

1). Escenario actual

CI global:

- 4ª causa infección torrente sanguíneo
- Incremento> 487% en incidencia en los últimos 20a
- Mortalidad:39%

CI en NN:

- 32,6 casos/100 000 RN/año
- 1,1 casos/100 altas UCI-N (24-40.5 sem gestación).
- Mortalidad cruda 21%.

RodriguezD. P Infect Dis J. 25(3):224-229, March 2006.

- Incidencia NN<1000g (N 1515): 9%.
- Mortalidad 20%

Bejamin DK. Pediatrics 2010, 126.

Candidemia global: 2) Puerta de entrada

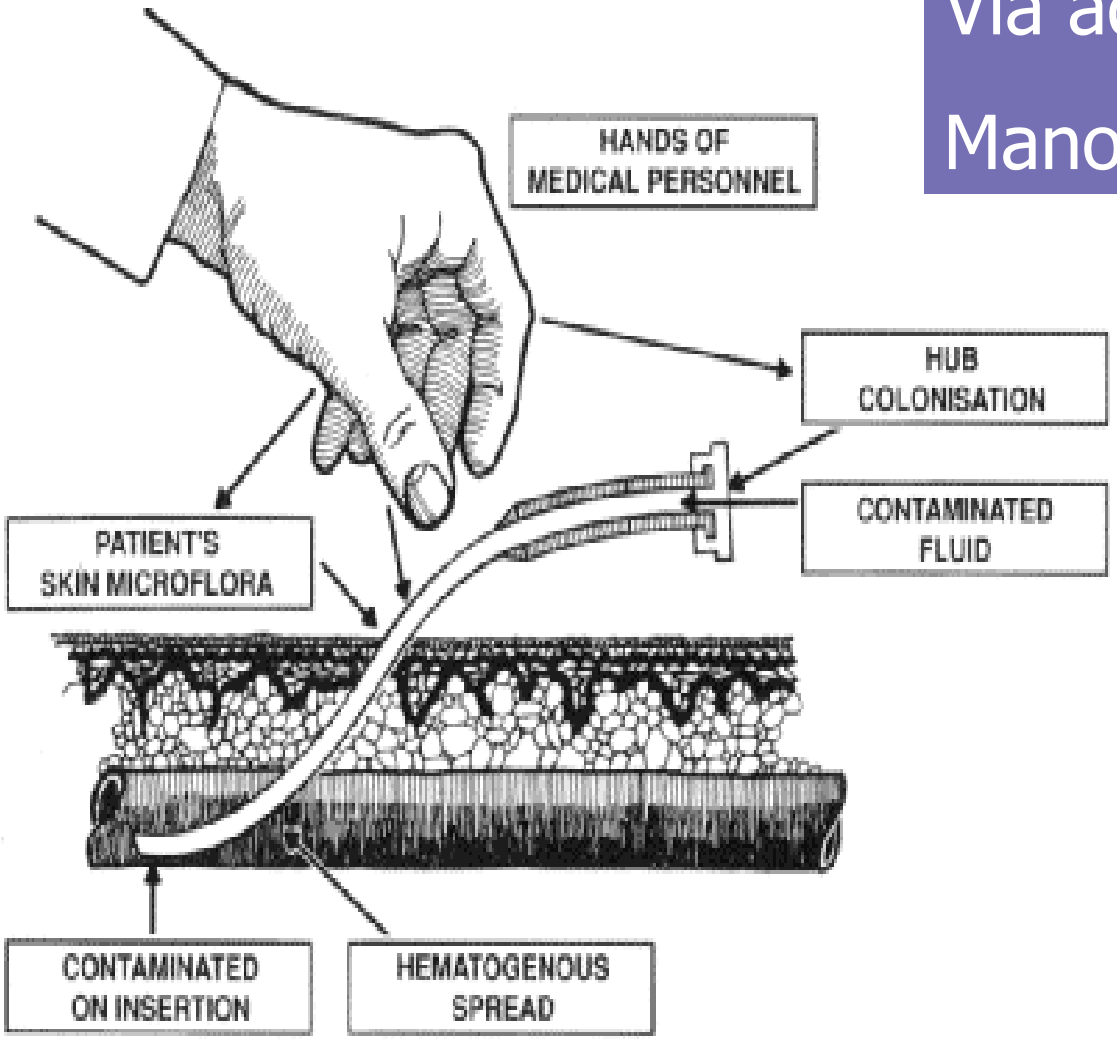
Table 7 Portal of entry of candidemia in patients hospitalized in French hospitals in 1995

Portal of entry	%
Venous catheter	25.8
Digestive tract	11.4
Respiratory tract	7.9
Urinary tract	6.0
Operation site	0.9
Other	5.0
Undetermined	43.1

Clin Microbiol Infect 2002; 8: 405-12

Vía adquisición UCI-N:

Manos personal sanitario



P. entrada NN: Candidiasis vaginal materna
Candidiasis Cutánea Congénita

- Adquisición vertical: c. vaginal materna+ c.extraño(DIU, suturas cervicales...)
- Cuadro no-invasivo en NN término
- CI grave en <27sem y < 1000 g

FIGURE 4: Congenital candidiasis

3). Fc de riesgo en UCI-N

Tabla 3
Factores de riesgo de candidiasis neonatal en unidad de cuidados intensivos neonatal

Peso muy bajo
Uso de cefalosporinas de tercera generación
Catéteres venosos centrales
Uso previo de intralípidos
Nutrición parenteral
Shock/coagulación intravascular diseminada
Fármacos anti-H_2
Corticosteroides
Puntuación < 5 en el test de Apgar
Intubación o estancia superior a 7 días en unidad de cuidados intensivos neonatal antecediendo a la candidiasis
Colonización gastrointestinal con *Candida*
Malformaciones congénitas

Modificada de Zaoutis et al[70].

4) Diagnóstico clínico

- Clínica inespecífica
- DD: contaminación/colonización/infección
- Ausencia de “scores” validados en pediatría

5) Diagnóstico micológico

- Métodos convencionales
  - Examen directo
  - Cultivo e identificación
- Métodos no convencionales
  - Detección de Ag
  - Detección de Ac
  - Técnicas genéticas

6) *Candida* spp. Epidemiología: Niños vs adultos

Table I. Recent epidemiology of *Candida* spp. isolated in blood cultures from immunocompromised adult and pediatric patients

Candida spp.	Percent of all *Candida* spp.	
	pediatrics[23-27]	adults[1,15-17,19,20]
C. albicans	40–48	28–67
C. parapsilosis	24–27	5–11
C. tropicalis	8–11	5–27
C. glabrata	2–12	5–26
C. krusei	3	1–13
C. lusitaniae	2–3	<1–2
C. guilliermondii	<1	<1–6

Brian T. Fisher and *Theoklis E. Zaoutis* Pediatr Drugs 2008; 10 (5)

Candida spp.: epidemiología en NN

Tabla 4
Porcentaje de *Candida* aisladas en unidad de cuidados intensivos neonatal

	Roilides et al[52] (%)	Levy et al[28] (%)
C. albicans	65	64,3
C. albicans y *C. parapsilosis*	0	7,1
C. parapsilosis	16	21,4
C. tropicalis	7	1,8
C. lusitaniae	5	0
C. guilliermondii	3	0
C. glabrata	2	5,4
C. intermedia	2	0

Modificada de Roilides et al[52] y Levy et al[28].

7) Presentación Clínica

TABLE 2. Lowest, Median, Highest, and Interquartile Range Reported Prevalence for End-Organ Damage After Neonatal Candidemia From 21 Studies

End-Organ	Lowest	25%	Median	75%	Highest
Attributed deaths	0%	12%	25%	33%	57%
Endophthalmitis	0%	0%	3%	17%	57%
Meningitis	0%	3%	15%	23%	67%
Brain abscess or ventriculitis	0%	3%	4%	21%	50%
Endocarditis	0%	0%	5%	13%	15%
Positive urine culture	0%	40%	61%	76%	100%
Renal ultrasound	0%	0%	5%	14%	50%
Liver/spleen ultrasound	0%	-	-	-	50%

PEDIATRICS
OFFICIAL JOURNAL OF THE AMERICAN ACADEMY OF PEDIATRICS

Neonatal Candidemia and End-Organ Damage: A Critical Appraisal of the Literature Using Meta-analytic Techniques
Daniel K. Benjamin, Jr, Charles Poole, William J. Steinbach, Judith L. Rowen and Thomas J. Walsh
Pediatrics 2003;112;634

Candidemia: Afectación cutánea

- Lesiones macronodulares
- Ectima gangrenoso
- Púrpura fulminans

**Diagnóstico: biopsia

Candidemia:Afectación ocular Coriorretinitis

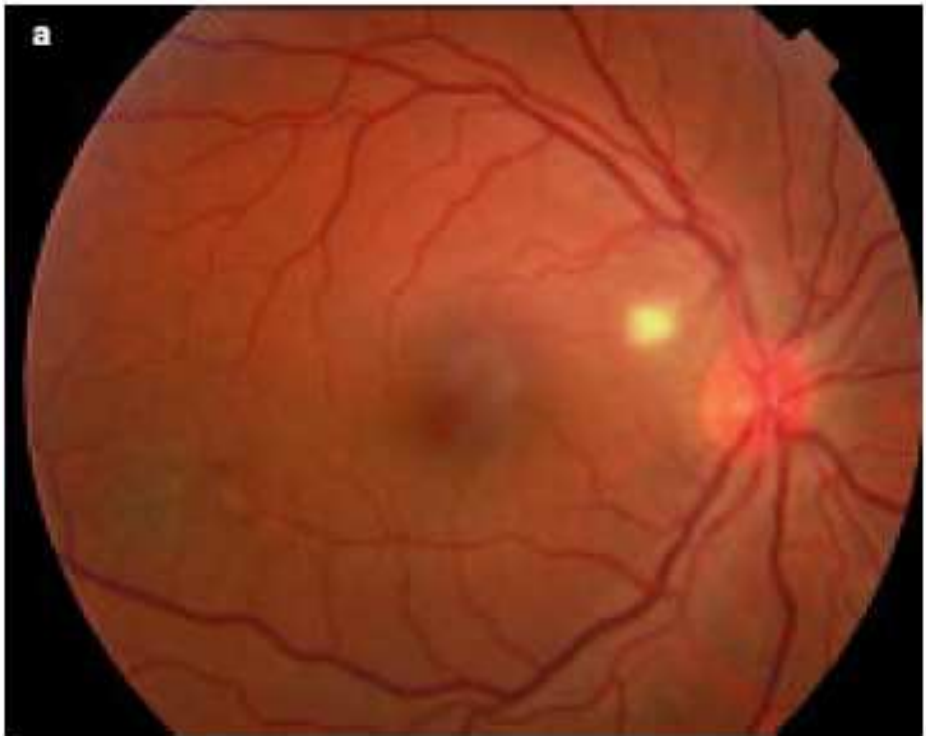


Figura 1. Fondo de ojo de un paciente con coriorretinitis por *C. albicans* tratado con fluconazol oral (200 mg/12 h) durante cinco semanas: a) Basal (previa al tratamiento): lesión coriorretiniana única, blanca, de bordes algodonosos peripapilar. b) A las dos semanas de tratamiento: crecimiento en tamaño

9-23 % de afectación ocular en candidemia.

Candidemia:Afectación ocular endoftalmitis por afectación vítreo

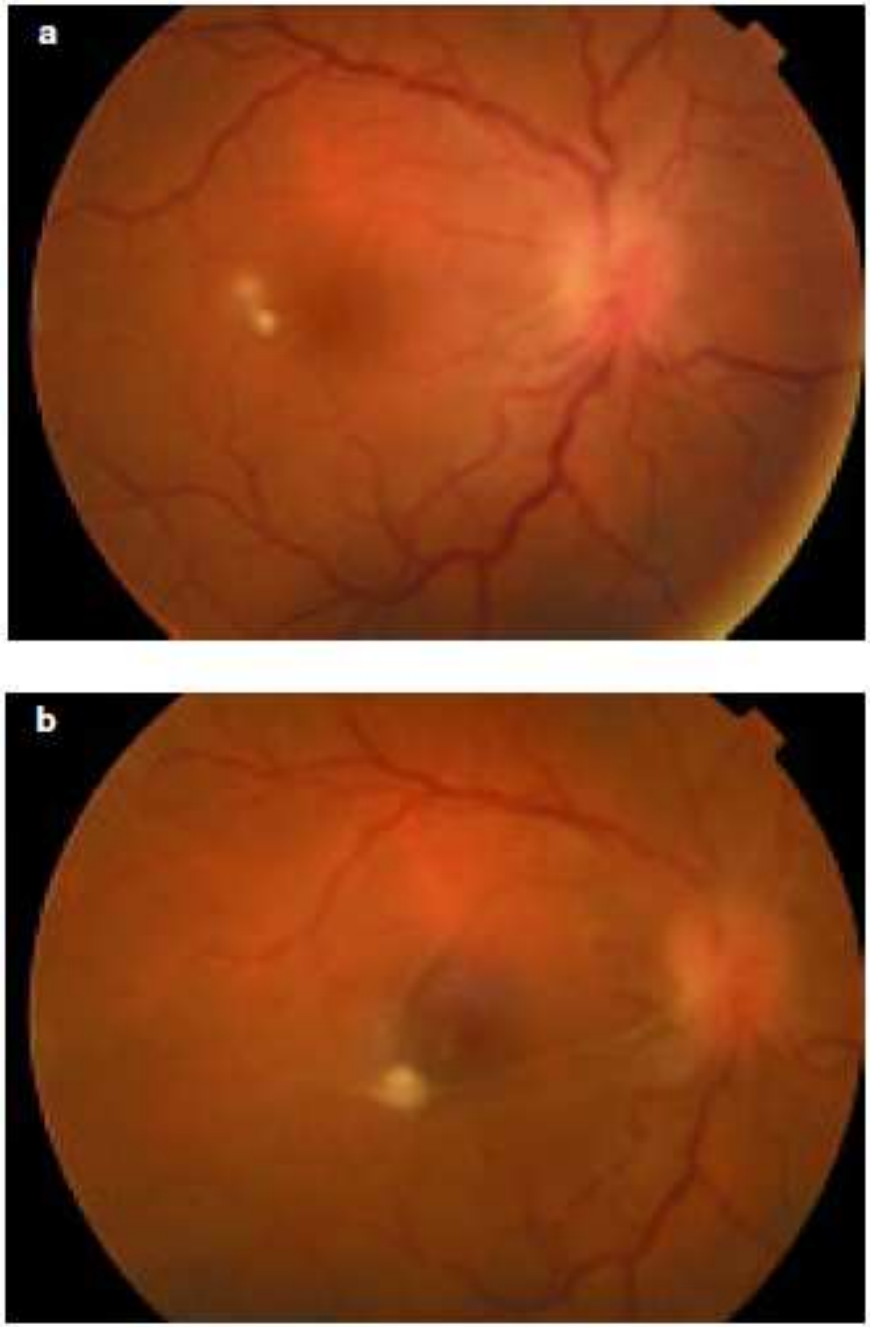
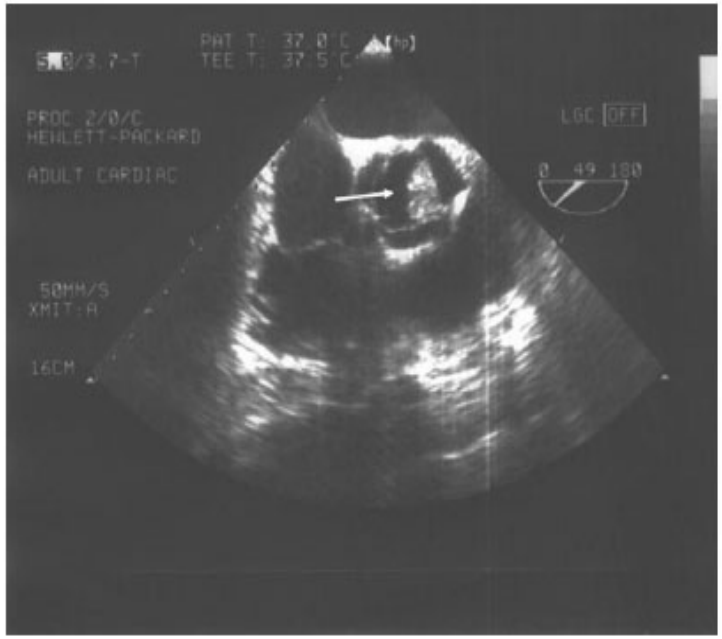
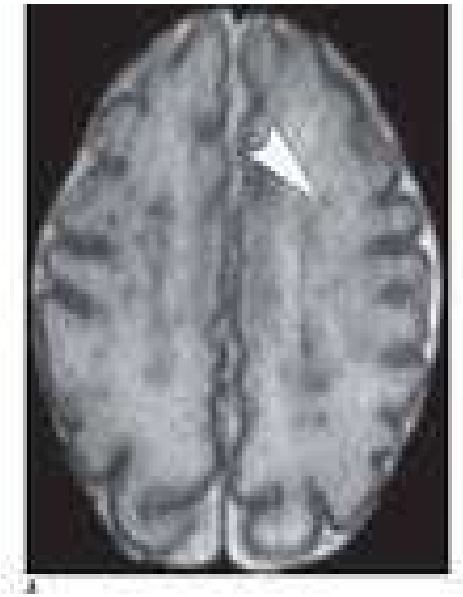


Figura 2. Fondo de ojo de una paciente con progresión a vítreo (endoftalmitis por *C. albicans)* a pesar del tratamiento inicial con fluconazol oral y respuesta parcial al tratamiento intravítreo con anfotericina B. Precisó vitrectomía para la resolución de la infección. a) Basal (previa al tratamiento): lesión en fóvea con invasión vítrea incipiente. Fluconazol oral (200 mg/12 h). b) A las dos semanas de tratamiento: progresión en forma de "perla vítrea".

Candidemia: endocarditis



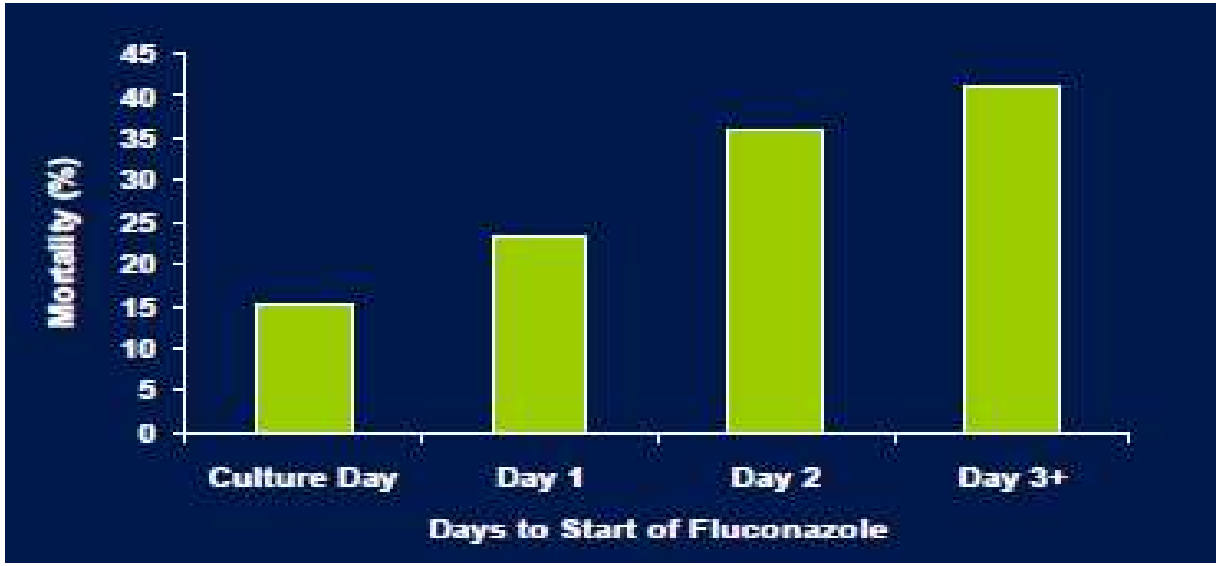


Candidemia:

Infecc. SNC

8) Tratamiento: Importancia del tratamiento precoz

- Mortalidad directamente asociada al retraso en el inicio del tratamiento



Garey KW, et al. *Clin Infect Dis* 2006;43:25-31.

Fc a valorar en el tratamiento empírico

- Epidemiología
- Condiciones del paciente:
  - Situación hemodinámica
  - Patología de base
  - Disfunción hepática/renal
  - Aislamientos previos/concomitantes
  - Otros fármacos. Interacciones
  - Antifúngicos previos
- Características del fármaco

Condiciones fármaco ideal

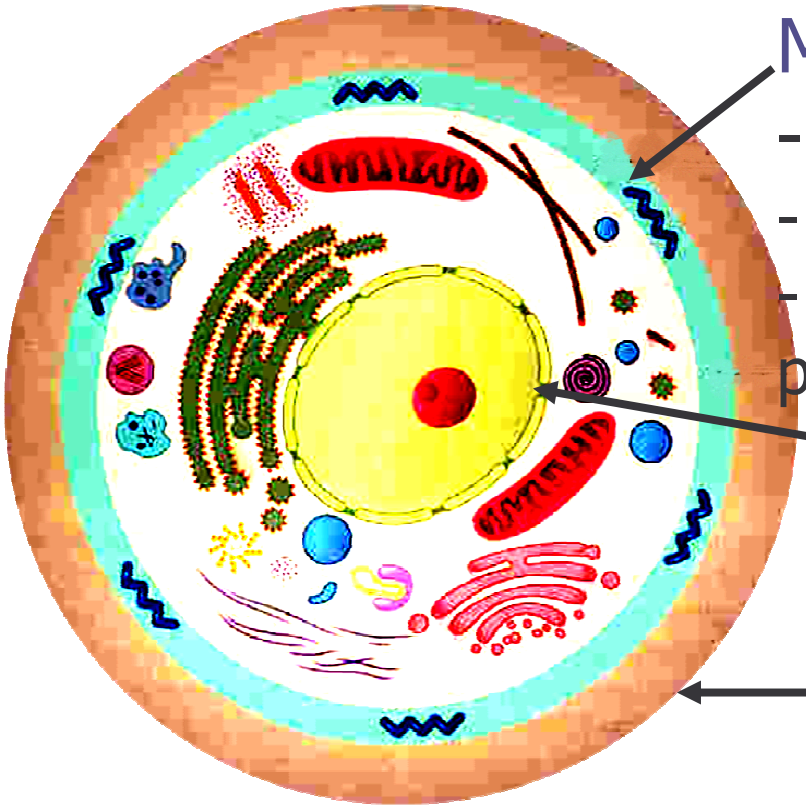
- Fungicida
- Amplio espectro
- Ausencia toxicidad
- Formulación IV y VO . Buena biodisponibilidad
- FK/FD favorable . Buena penetración tisular
- Ausencia interacciones
- Ausencia variabilidad genética en metabolización
- Estable a R
- Costoefectivo

⇩

Azoles – ABL/ABCL – Equinocandinas

Qué debemos saber acerca del fármaco

a. Dianas de actuación



Membrana celular

- Polienos: Unión ergosterol
- Azoles: inhib. síntesis ergosterol
- Alilaminas (terbinafina):inhiben paso de escualeno a lanosterol

Síntesis DNA

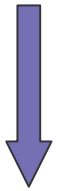
- 5-Fluocytosina

Pared celular

- Equinocandinas: inhib. Síntesis glucano

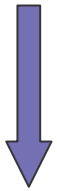
b) Mecanismo de actuación

- Fungicida: Anfotericinas
  Equinocandinas



Inestabilidad hemodinámica (1as 48-72h)

- Fungistático: fluconazol



Paciente estable

c) Espectro de actividad

Table 1. Antifungal spectrum of activity against common fungi.

Organism	Antifungal agent								
	AmB[a]	Flu	Itr	Vor	Pos	Anidulafungin	Caspofungin	Micafungin	Flucytosine
Aspergillus species	+	−	+	+	+	+	+	+	−
A. flavus	±	−	+	+	+	+	+	+	−
A. fumigatus	+	−	+	+	+	+	+	+	−
A. niger	+	−	±	+	+	+	+	+	−
A. terreus	−	−	+	+	+	+	+	+	−
Candida species	+	+	+	+	+	+	+	+	+
C. albicans	+	+	+	+	+	+	+	+	+
C. glabrata	+	±	±	+	+	+	+	+	+
C. krusei	+	−	±	+	+	+	+	+	±
C. lusitaniae	−	+	+	+	+	+	+	+	+
C. parapsilosis	+	+	+	+	+	±	±	±	+
C. tropicalis	+	+	+	+	+	+	+	+	+
Cryptococcus neoformans	+	+	+	+	+	−	−	−	+
Coccidioides species	+	+	+	+	+	±[b]	±[b]	±[b]	−
Blastomyces	+	+	+	+	+	±[b]	±[b]	±[b]	−
Histoplasma species	+	+	+	+	+	±[b]	±[b]	±[b]	−
Fusarium species	±	−	−	+	+	−	−	−	−
Scedosporium apiospermum	±	−	±	+	+	−	−	−	−
Scedosporium prolificans	−	−	−	±	±	−	−	−	−
Zygomycetes	±	−	−	−	+	−	−	−	−

d) Farmacocinética

Table 2. Comparative pharmacokinetics of the antifungal agents.

	Antifungal agent											
Pharmacokinetic parameter	AmB	ABCD	ABLC	LAB	Flu	Itr[a]	Vor	Pos	Anidulafungin	Caspofungin	Micafungin	Flucytosine
Oral bioavailability, %	<5	<5	<5	<5	95	50	96	ND	<5	<5	<5	80
Food effect	NA	NA	NA	NA	NE	ES	ES	Food	NA	NA	NA	NE
Distribution												
Total C_{max}, μg/mL	0.5–2	4	131	0.1	0.7	11	4.6	7.8	0.83	0.27	0.24	80
AUC, mg × h/L	17	43	14	555	400	29.2	20.3	8.9	99[b]	119	158[b]	62
Protein binding, %	>95	>95	>95	>95	10	99.8	58	99	84	97	99	4
CSF penetration, %	0–4	<5	<5	<5	>60	<10	60	NR	<5	<5	<5	75
Vitreal penetration, %	0–38[c,d]	0–38[c,d]	0–38[c,d]	0–38[c,d]	28–75[c,d]	10[c]	38[c]	26[c,d]	0[d]	0[c]	<1[d]	49[d]
Urine penetration, %[e]	6–20	<5	<5	4.5	90	1–10	<2	<2	<2	<2	<2	90
Metabolism	Minor Hep	Unk	Unk	Unk	Minor Hep	Hep	Hep	Hep	None	Hep	Hep	Minor intestinal
Elimination	Feces	Unk	Unk	Unk	Urine	Hep	Renal	Feces	Feces	Urine	Feces	Renal
Half-life, h	50	30	173	100–153	31	24	6	25	26	30	15	3–6

Dodds Ashley, CID 2006:43(suppl 1)

Farmacocinética equinocandinas

Table II. Comparison of major properties and key pharmacokinetic parameters of echinocandins in adults[a][17,20,51]

Variable	Caspofungin	Micafungin	Anidulafungin
C_{max} (mg/L) [50 mg single dose]	7.64	4.95	2.07–3.5
Bioavailability (%)	<10	<10	2–7
$t_{½}$ (h)	9–11	11–17	24–26
Vd (L/kg)	0.14	0.22–0.24	0.5
AUC (mg • h/L)	87.9–114.8	111.3	44.4–53
Protein binding (%)	96–97	99.8	>99
Metabolism	Slow peptide hydrolysis, N-acetylation and spontaneous degradation to inactive product	Catechol-*O*-methyltransferase pathway	Not metabolized Slow chemical degradation to inactive metabolites
Cl_T	0.15	0.185	0.26
Fraction excreted unchanged in urine (%)	1.4	0.7	<1
Elimination	35% faeces, 41% urine, 1.4% as unchanged drug	40% faeces, <15% urine	Primarily faeces (<10% as intact drug), 1% urine
CSF penetration (% plasma)	Low	Low	<0.1%
Dose adjustment in renal insufficiency	No dose adjustment needed	No dose adjustment needed	No dose adjustment needed
Dose adjustment in geriatric patients	No dose adjustment needed	No dose adjustment needed	No dose adjustment needed
Dose adjustment in hepatic insufficiency	Child-Pugh 5–6: none Child-Pugh 7–9: significant increase in AUC; reduce maintenance dose to 35 mg/day Child-Pugh >9: no data	Child-Pugh 7–9: C_{max} not altered, AUC significantly decreased compared with healthy subjects	No dose adjustment needed in patients with mild, moderate or severe hepatic dysfunction

a Data are from multiple sources and are not always comparable.

AUC = area under the plasma concentration-time curve; **Cl_T** = total clearance; **C_{max}** = maximum concentration; **CSF** = cerebrospinal fluid; **$t_{½}$** = elimination half-life; **Vd** = volume of distribution.

e) Toxicidad

Table 6. Comparative toxicities of antifungal agents.

	Antifungal agent											
Type of toxicity	AmB	ABCD	ABLC	LAB	Flu	Itr	Vor	Pos	Anidulafungin	Caspofungin	Micafungin	Flucytosine
Hepatic	++	++	++	++	+	+	+	+	+	+	+	++
Nephrotic	++++	+++	+++	++	–	–	–	–	–	–	–	–
Hematologic	+	+	+	+	NR	NR	NR	NR	NR	+	+	+++
Infusion-related	+++	+++	+++	++	–	–	–	NA	+	+	+	NA
Electrolyte abnormalities[a]	+++	++	++	++	NR	+	+	NR	+	+	NR	+

NOTE. Plus signs indicate degree of toxicity: +, mild; ++, moderate; and +++, severe. ABCD, amphotericin B colloidal dispersion; ABLC, amphotericin B lipid complex; AmB, amphotericin B; Flu, fluconazole; Itr, itraconazole; LAB, liposomal amphotericin B; NA, data not available because of a lack of formulation; NR, not reported; Pos, posaconazole; Vor, voriconazole. Data are derived from [5, 37, 41, 45, 47, 51, 52, 54, 58, 70, 111–118].

[a] Includes hypokalemia and hypomagnesemia.

Dodds Ashley, CID 2006:43(suppl 1)

f) Requerimiento ajuste dosis por disfunción orgánica

Table 3. Suggested dose modifications for antifungal agents, by type of organ dysfunction.

Type of organ dysfunction	Antifungal agent											
	AmB	ABCD	ABLC	LAB	Flu	Itr	Vor	Pos	Anidulafungin	Caspofungin	Micafungin	Flucytosine
Hepatic	None	None	None	None	None	None	Decrease dose for mild-to-moderate cirrhosis	None	None	Decrease dose for moderate insufficiency	None	None
Renal	None	None	None	None	Decrease dose	Caution with iv preparation for ClCr <30 mL/min	Caution with iv preparation for ClCr <50 mL/min	None	None	None	None	Decrease dose

NOTE. ABCD, amphotericin B colloidal dispersion; ABLC, amphotericin B lipid complex; AmB, amphotericin B; ClCr, creatinine clearance; Flu, fluconazole; Itr, itraconazole; LAB, liposomal amphotericin B; Pos, posaconazole; Vor, voriconazole. Data are derived from [37, 41, 45, 47, 54, 80, 81, 90, 92].

Dodds Ashley, CID 2006:43(suppl 1)

g) Interacciones: Azoles

Table 4. Summary of azole-mediated cytochrome P450 drug-drug interactions.

Drug mechanism	Drug			
	Flu	Itr	Pos	Vor
Inhibitor				
2C19	+			+++
2C9	++	+		++
3A4	++	+++	+++	++
Substrate				
2C19				+++
2C9				+
3A4		+++		+

NOTE. Plus signs indicate degree of activity: +, minimal activity; ++, moderate activity; and +++, strong activity. Flu, fluconazole; Itr, itraconazole; Pos, posaconazole; Vor, voriconazole. Data are derived from [37, 41, 45, 95, 96].

Dodds Ashley, CID 2006:43(suppl 1)

Voriconazol: interacciones

Tabla 3. Principales interacciones medicamentosas de voriconazol

Tipo de interacción	Recomendación
El fármaco disminuye los niveles de voriconazol	
Carbamazepina	Contraindicado
Rifampicina	Contraindicado
Barbitúricos de acción prolongada	Contraindicado
Voriconazol incrementa las concentraciones del fármaco	
Astemizol	Contraindicado
Cisaprida	Contraindicado
Ciclosporina	Reducir dosis a la mitad y monitorizar niveles
Alcaloides ergotamínicos	Contraindicado
Omeprazol	Reducir dosis a la mitad
Quinidina	Contraindicado
Sirolimus	Contraindicado
Tacrolimus	Reducir la dosis dos tercios y monitorizar las concentraciones
Terfenadina	Contraindicado
Warfarina	Monitorizar el tiempo de protrombina
El fármaco disminuye los valores de voriconazol y voriconazol incrementa concentraciones del otro fármaco	
Rifabutina	Contraindicado
Fenitoína	Doblar dosis de voriconazol y monitorizar niveles de fenitoína
Voriconazol probablemente incrementa los valores del fármaco	
Estatinas	Monitorizar los efectos y considerar una reducción en las dosis cuando se añade voriconazol
Alcaloides de la VINCA	
Sulfonilureas	
Bloquedores de los canales del calcio	
Benziodiacepinas	

Adaptado de ref. 4

Interacciones: equinocandinas

Table IV. Drug interactions with the echinocandins[11-13,138-142]

Drug	Caspofungin	Micafungin	Anidulafungin
CYP/P-glycoprotein interactions	Poor substrate for CYP Not an inhibitor of CYP Not a substrate/inhibitor of P-glycoprotein	Substrate for CYP3A4 Weak inhibitor CYP3A4 Not a substrate/inhibitor of P-glycoprotein	Not a substrate, inducer or inhibitor of CYP
Tacrolimus	AUC, peak and 12-hour concentrations of tacrolimus are decreased by ~20%	No significant effect on tacrolimus	No significant effect on tacrolimus
Sirolimus	No data	Increases AUC of sirolimus by 12%	No data
Ciclosporin	35% increase in the AUC of caspofungin	Decreases clearance of ciclosporin by 16%	22% increase in AUC of anidulafungin; dose adjustment not required
Rifampicin	Decreases steady-state plasma caspofungin concentrations	No significant effect on micafungin	No significant effect on anidulafungin
Voriconazole	No data	No significant effect on micafungin	No significant effect on anidulafungin
Nefidipine	No data	Increases the AUC and C_{max} of nifedipine by 18% and 43%, respectively	No data

AUC = area under the plasma concentration-time curve; $\mathbf{C_{max}}$ = maximum concentration; **CYP** = cytochrome P450.

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h) Monitorización C plasmáticas

Table 5. Serum drug concentration monitoring for antifungal agents.

Antifungal agent	Serum drug concentration monitoring[a]	Target range	Timing of sample
Amphotericin B[b]	No	NA	NA
Flucytosine	Yes (because of toxicity)	<100 μg/mL	Peak 2 h after dose
Fluconazole	No	NA	NA
Itraconazole	Yes (to ensure absorption and efficacy)	>0.5 μg/mL	Trough after 7 days of therapy
Voriconazole	Yes (because of variable metabolism, toxicity and drug interactions, and potentially pediatrics)	2–6 μg/mL	Trough after 7 days of therapy
Echinocandins	No	NA	NA

NOTE. The recommendations in this table represent expert opinion and have not been validated in prospective clinical trials. NA, not applicable. Data are derived from [109, 110].

[a] Serum drug concentration monitoring may be considered to monitor for efficacy and/or toxicity.

[b] Includes lipid preparations.

Dodds Ashley, CID 2006:43(suppl 1)

Recomendaciones Terapéuticas

Clinical Practice Guidelines for the Management of Candidiasis: 2009 Update by the Infectious Diseases Society of America

Peter G. Pappas,[1] Carol A. Kauffman,[2] David Andes,[4] Daniel K. Benjamin, Jr.,[5] Thierry F. Calandra,[11] John E. Edwards, Jr.,[6] Scott G. Filler,[6] John F. Fisher,[7] Bart-Jan Kullberg,[12] Luis Ostrosky-Zeichner,[8] Annette C. Reboli,[9] John H. Rex,[13] Thomas J. Walsh,[10] and Jack D. Sobel[3]

Neonatal candidiasis	AmB-d 1 mg/kg daily (A-II); or fluconazole 12 mg/kg daily (B-II) for 3 weeks	LFAmB 3–5 mg/kg daily (B-III)	A lumbar puncture and dilated retinal examination should be performed on all neonates with suspected invasive candidiasis. Intravascular catheter removal is strongly recommended. Duration of therapy is at least 3 weeks. LFAmB used only if there is no renal involvement. Echinocandins should be used with caution when other agents cannot be used.

Treatment for Neonatal Candidiasis

- AmB-d (1 mg/kg daily) is recommended for neonates with disseminated candidiasis (A-II). If urinary tract involvement is excluded, LFAmB (3–5 mg/kg daily) can be used (B-II). Fluconazole (12 mg/kg daily) is a reasonable alternative (B-II). The recommended length of therapy is 3 weeks (B-II).
- A lumbar puncture and a dilated retinal examination, preferably by an ophthalmologist, are recommended in neonates with sterile body fluid and/or urine cultures positive for *Candida* species (B-III). Imaging of the genitourinary tract, liver, and spleen should be performed if the results of sterile body fluid cultures are persistently positive (B-III).
- Echinocandins should be used with caution and are generally limited to situations in which resistance or toxicity precludes the use of fluconazole or AmB-d (B-III).
- Intravascular catheter removal is strongly recommended (A-II).
- In nurseries with high rates of invasive candidiasis, fluconazole prophylaxis may be considered in neonates whose birth weight is <1000 g (A-I). Antifungal drug resistance, drug-related toxicity, and neurodevelopmental outcomes should be observed (A-III).

An Pediatr (Barc). 2011;xxx(xx):xxx.e1–xxx.e17

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ASOCIACIÓN ESPAÑOLA DE PEDIATRÍA

Recomendaciones de la Sociedad Española de Infectología Pediátrica sobre diagnóstico y tratamiento de la candidiasis invasiva

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Tratamiento: Pautas literatura

Not Just Little Adults: Candidemia Epidemiology, Molecular Characterization, and Antifungal Susceptibility in Neonatal and Pediatric Patients
Christopher C. Blyth, Sharon C. A. Chen, Monica A. Slavin, Carol Serena, Quoc Nguyen, Deborah Marriott, David Ellis, Wieland Meyer and Tania C. Sorrell
Pediatrics 2009;123;1360

TABLE 3 Antifungal Therapy Administered to Neonates, Children, and Adults With Candidemia

Antifungal Therapy	Neonates, *n* (%)	Children, *n* (%)	Adults, *n* (%)	Significant Results
Amphotericin B monotherapy[a]	7 (31.2)	21 (28.4)	46 (7.1)	N vs A: $P < .001$ C vs A: $P < .001$
Fluconazole monotherapy	3 (13.6)	27 (36.5)	368 (56.7)	N vs C: $P = .024$ N vs A: $P < .001$ C vs A: $P < .002$
Amphotericin B then fluconazole[a]	8 (36.4)	13 (17.6)	54 (8.3)	N vs A: $P < .001$ C vs A: $P = .018$
Fluconazole then Amphotericin B[a]	2 (9.1)	5 (6.8)	56 (8.6)	All not significant
Combination antifungal therapy	2 (9.1)	3 (4.1)	18 (2.8)	All not significant
Amphotericin B and azole[a]	2	3	2	
Amphotericin B and echinocandin[b]	0	0	12[b]	
Azole and echinocandin	0	0	3	
Other combination	0	0	1	
Other antifungal agents used either alone or sequentially	0	5	107	N vs A: $P = .035$ C vs A: $P = .027$

50%

N indicates neonates; C, children; A, adults.

[a] Data include all of the amphotericin preparations: deoxycholate and lipid formulations.

[b] Eleven patients were included in a clinical trial using the combination of micafungin and liposomal amphotericin B.

9). Consideraciones sobre Profilaxis

- *Kaufman (2001) y Manzoni (2007)*: Fluconazol (3 mg/kg o 6 mg/kg, 2 veces/sem) reduce CI en NICUS con tasas de 20% (RN<1000g) y de 13% (RN1000–1500g).

- Incidencia CI casi 10 veces < en la mayoría de NICUS: *Smith PB, Jr. Neonatal candidiasis. Infect Dis Clin North Am 2005; 19:603–15.: Benjamin, Pediatrics 2006; 117:84–92*.

- No existen datos de seguridad sobre fluconazol en prematuros: profilaxis indiscriminada no recomendable

- Guía IDSA: se recomienda profilaxis rutinaria con fluconazol sólo en prematuros con pesos muy bajos en NICUS con elevada incidencia de CI

10). Conclusiones

- Incidencia variable en NN de riesgo
- Importancia prevención: lavado manos
- Importancia del diagnóstico precoz
- Necesidad retirada catéter
- Necesidad estudio de extensión
- Tratamiento antifúngico eficaz y adaptado a la presentación de la infección en el NN
- Profilaxis según incidencia

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Gracias !

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