

**RICORS-ICTUS**



Instituto de Salud Carlos III

# INFECCION/INFLAMACION como un factor de riesgo para ICTUS ISQUEMICO

**Webinar Formativa Mensual RICORS**

*T. Segura*

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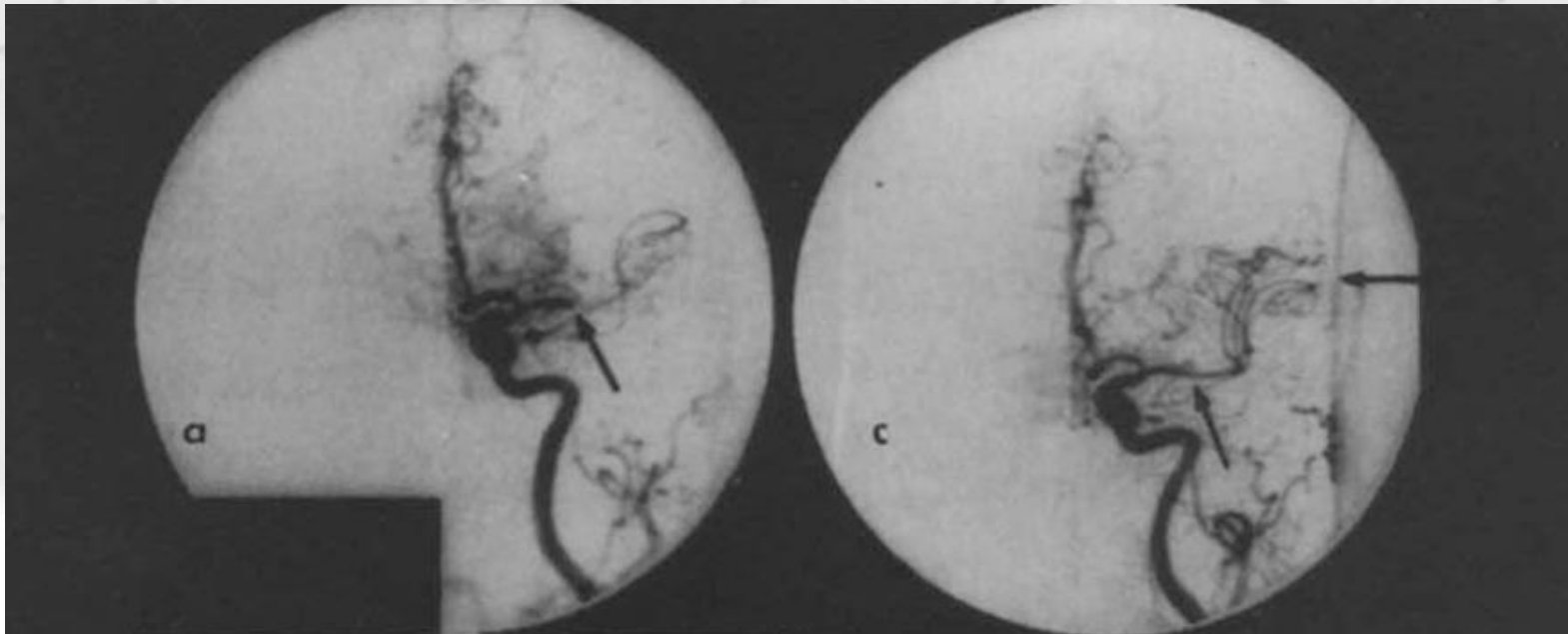


DOI: 10.1016/0022-510X(89)90060-9 • Corpus ID: 20276600

## Clinical and instrumental evaluation of patients with ischemic stroke within the first six hours

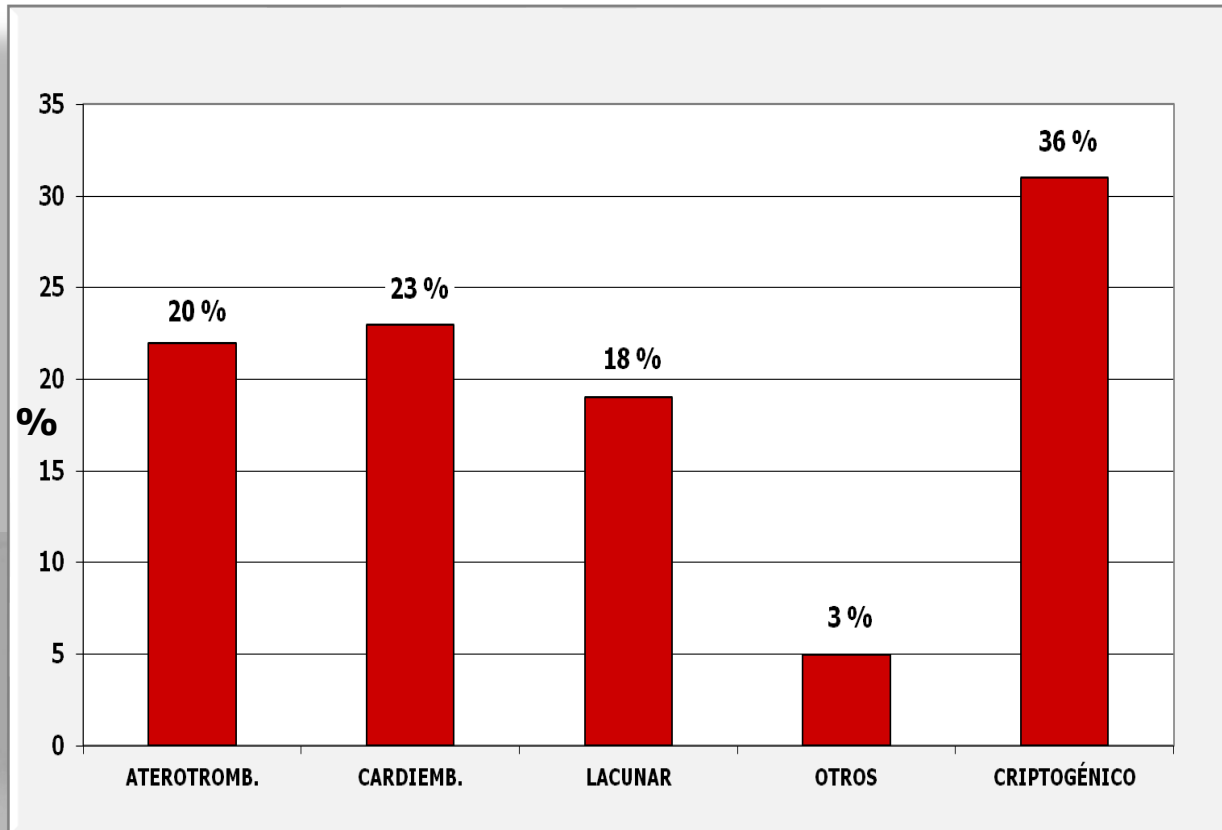
C. Fieschi, C. Argentino, +3 authors L. Bozzao • Published 1 July 1989 • Medicine • Journal of the Neurological Sciences

The development of fibrinolytic agents such as streptokinase and recombinant tissue type plasminogen activator (r-TPA) and other modalities of treatment in acute ischemic stroke, has raised the need for a more precise knowledge of the pathophysiology of the acute phases of ischemic stroke as it pertains to prediction of clinical outcome. In a prospective analysis, 80 patients were studied within less than 6 h from the onset of symptoms by means of a detailed protocol including clinical... [Expand](#)



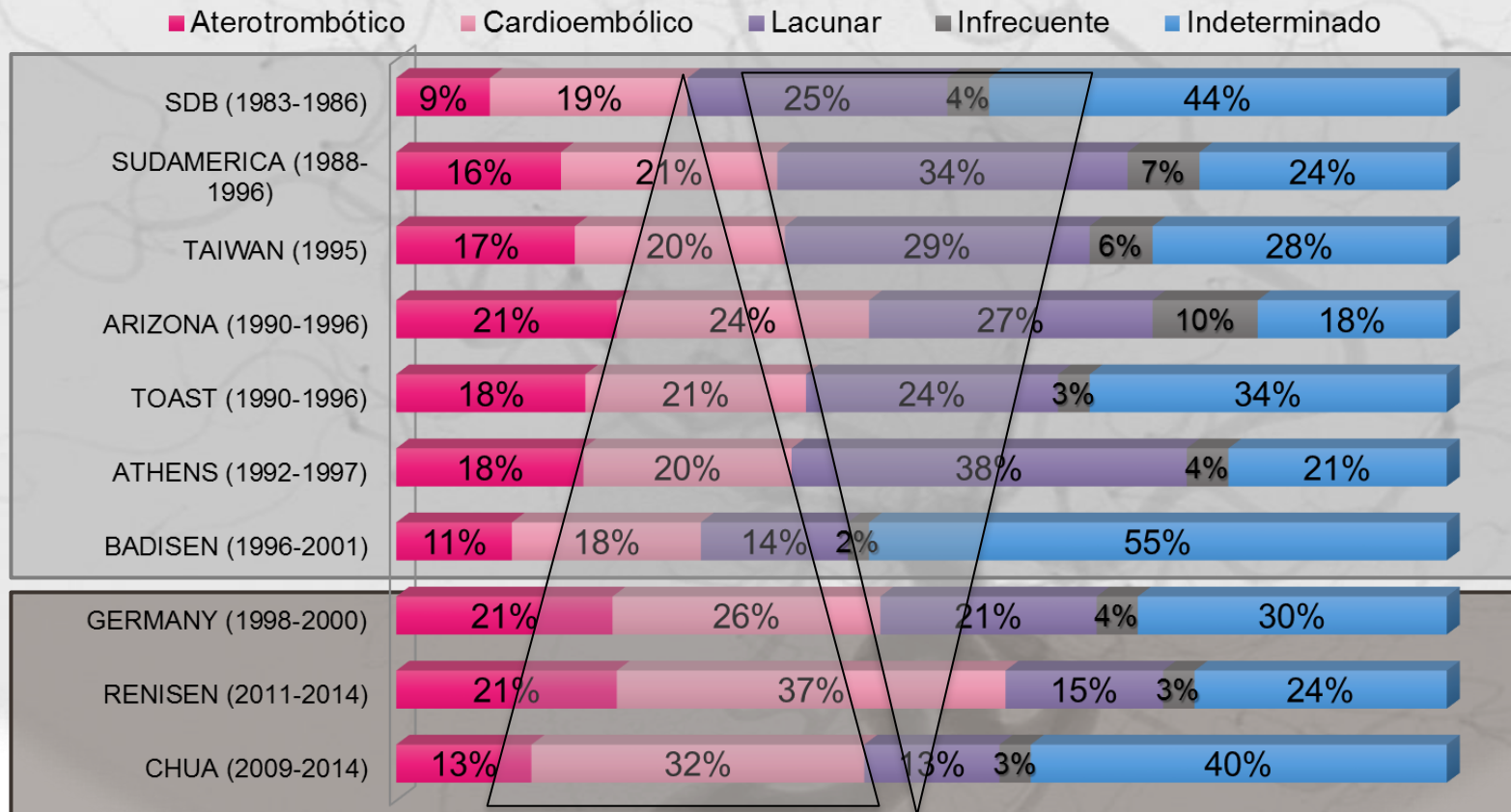
# Etiología del ictus isquémico

Registro BADISEN  
(N ≈ 5.603 PACIENTES)



Cortesía Dr. Serena

# Etiología del ictus isquémico



## **Etiología del ictus isquémico**

- ❑ La existencia de una alta proporción de ictus isquémicos en los que no se halla causa para el fenómeno embólico**
- ❑ La presencia de ictus en pacientes sin factores de riesgo vascular, sobre todo en los más jóvenes**
- ❑ La confirmación de que hay más incidencia de ictus en los meses invernales**
- ❑ La existencia de varios estudios caso-control que muestran mayor frecuencia de ictus si ha existido una infección previa**

**Sugieren la posibilidad de un papel relevante de los procesos infecciosos en la generación de la **isquemia cerebral****

## Frecuencia del ICTUS asociado a COVID-19

**Table 1.** Cohort studies reporting cerebrovascular events in COVID-19 patients during general hospital admission.

Cohort study	Date	COVID-19 hospital admissions	Cerebrovascular disease (%)*	IS (%)*	ICH (%)*	CVT (%)*
Yaghi <i>et al.</i> <sup>48</sup>	15 March–19 April 2020	3556		32 (0.9)	–	–
Merkler <i>et al.</i> <sup>47</sup>	4 March–2 May 2020	2132	–	31 (1.5)	–	–
Lodigiani <i>et al.</i> <sup>49</sup>	13 February–10 April 2020	388	–	9 (2.3)	–	–
Jain <i>et al.</i> <sup>50</sup>	1 March–13 April 2020	3218	35 (1.1)	26 (0.8)	9 (0.3)	–
Cantador <i>et al.</i> <sup>51</sup>	1 February–21 April 2020	1419	8 (0.6)	6 (0.4)	–	–
Shahjouei <i>et al.</i> <sup>46</sup>	27 March–1 May 2020	17,799	156 (0.9)	123 (0.7)	25 (0.1)	6 (<0.1)
Rothstein <i>et al.</i> <sup>52</sup>	15 March–3 May 2020	844	28 (3.3)	20 (2.4)	8 (0.9)	–
Li <i>et al.</i> <sup>53</sup>	16 January–29 February 2020	219	11 (5)	10 (4.6)	1 (0.5)	–
Romero-Sanchez <i>et al.</i> <sup>54</sup>	1 March–1 April 2020	841	14 (1.7)	11 (1.3)	3 (0.4)	–
Katz <i>et al.</i> <sup>55</sup>	14 March– 26 April 2020	10,596	86 (0.8)	72 (0.7)	14 (0.1)	–
Siegler <i>et al.</i> <sup>56</sup>	1 February–16 June 2020	14,483	172 (1.2)	156 (1.1)	28 (0.2)	3 (<0.1)
Requena <i>et al.</i> <sup>57</sup>	2 March–30 April 2020	2050	25 (1.2)	21 (1)	4 (0.2)	–
Kvernland <i>et al.</i> <sup>58</sup>	1 March–15 May 2020	4071	–	–	19 (0.5)	–
Altschul <i>et al.</i> <sup>59</sup>	1 March–1 May 2020	5227	–	–	35 (0.7)	–

\*Percentages correspond to the number of events out of the total COVID-19 general hospital admissions. COVID-19, coronavirus disease 2019; CVT, cerebral venous thrombosis; ICH, intracranial hemorrhage; IS, ischemic stroke.



# Frecuencia del ICTUS asociado a COVID-19

**ORIGINAL CONTRIBUTION** *Stroke* 2021;52:00–00. DOI: 10.1161/STROKEAHA.121.034883

## Impact of COVID-19 Infection on the Outcome of Patients With Ischemic Stroke

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13%


Martí-Fàbregas et al

Impact of COVID-19 in Outcome of Ischemic Stroke

**Table 1. Demographics, Vascular Risk Factors, and Clinical Data of the Patients**

	All patients (n=701)	COVID-19 (n=91)	Non-COVID-19 (n=610)	P value
Demographics				
Age, mean (SD)	72.3 (13.3)	71.6 (12.3)	72.4 (13.5)	0.603
Sex (men), n (%)	424 (60.5)	58 (63.7)	366 (60.0)	0.496

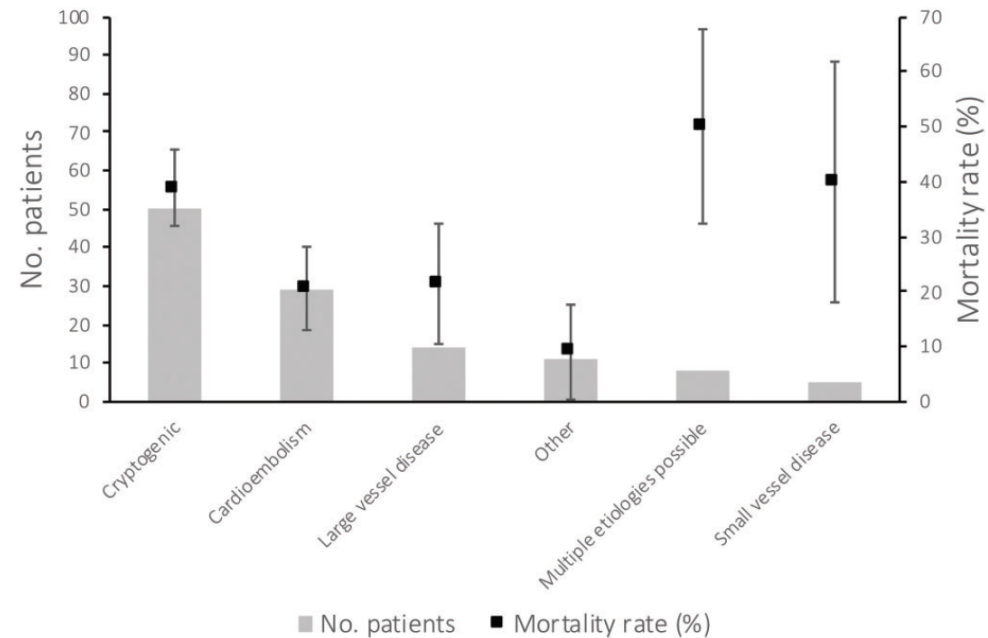
## Cerebrovascular events and outcomes in hospitalized patients with COVID-19: The SVIN COVID-19 Multinational Registry

International Journal of Stroke  
0(0) 1–11  
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DOI: [10.1177/1747493020959216](https://doi.org/10.1177/1747493020959216)  
[journals.sagepub.com/home/wso](http://journals.sagepub.com/home/wso)

Extraído de SieglerJ.E. 2020

### In-hospital mortality rate according to stroke etiology

The distribution of stroke etiologies is plotted using vertical bars (primary y-axis), and mortality rate according to stroke mechanism is plotted as squares with standard error bars (secondary y-axis).









# Infecciones que provocan ictus

## Box 1 | Infectious diseases associated with stroke

The following infectious diseases have been shown to be associated with stroke, and may elicit their effects through the proposed mechanisms:

### Bacterial diseases

- Infective endocarditis can result in infective and noninfective brain emboli, and arteritis
- Bacterial meningitis can lead to vasculitis and thrombosis of intracerebral arteries
- Tuberculous meningitis involves arterial invasion of mycobacteria, which may result in vasculitis
- Neurosyphilis involves arterial invasion by *Treponema pallidum*, which may lead to panarteritis and periarteritis as well as pro-atherosclerotic effects
- Neuroborreliosis can cause intracranial vasculitis
- Rickettsial diseases involve microbial invasion into the endothelium of small arteries

### Viral diseases

- HIV infection can lead to vasculitis or vasculopathy, and may increase the probability of opportunistic infections
- Herpes zoster involves viral invasion of the arterial walls, and can lead to granulomatous and necrotizing vasculitis
- Hepatitis B or C infection can cause vasculitis and cryoglobulinemia

### Fungal diseases

- Fungal meningitis, which may be caused by *Candida albicans*, *Coccidioides immitis*, *Aspergillus fumigatus* or *Cryptococcus neoformans*, among others, involves invasion of fungi into cerebral arteries
- Mucormycosis directly affects arteries, and can lead to necrotizing vasculitis

# Infecciones como factor de riesgo de ictus

Study*	Age of study participants	Prestroke intervals assessed	Number of patients with stroke, and prestroke infection rate	Number of controls and infection rate	Risk of stroke with acute infection	Comments
Syrjänen <i>et al.</i> (1988) <sup>7</sup>	<50 years	1 month	54 (35.2%)	54 (5.6%)	OR 14.5, 95% CI 1.5–112	Mainly respiratory infections; population controls
Grau <i>et al.</i> (1995) <sup>9</sup>	<80 years	1 week	197 (19.3%)	197 (5.1%)	OR 4.6, 95% CI 1.9–11.3	Bacterial infections; mainly respiratory infections; population controls
Bova <i>et al.</i> (1996) <sup>10</sup>	73 years (mean age)	2 months	182 (24.2%)	194 (9.7%)	OR 2.9, 95% CI 1.6–5.3	Mainly respiratory and urinary tract infections; controls were patients with previous stroke
Macko <i>et al.</i> (1996) <sup>11</sup>	59 years (mean age)	1 week	37 (35.1%)	81 (11.1%)	Risk not calculated; $P < 0.02$ for difference in infection rates	Febrile and nonfebrile infections, and other inflammatory events; population and hospital controls
Grau <i>et al.</i> (1998) <sup>8</sup>	<85 years	1 week	166 (22.3%)	166 (8.4%)	OR 2.9, 95% CI 1.3–6.4	Mainly respiratory tract infections; bacterial and viral infections; hospital controls
Nagaraja <i>et al.</i> (1999) <sup>12</sup>	<40 years	2 weeks	60 (33.3%)	60 (no data provided for time interval)	Risk not calculated; $P < 0.01$ for difference in infection rates	Mainly respiratory infections; bacterial and viral infections; population controls
Nencini <i>et al.</i> (2003) <sup>13</sup>	72 years (mean age)	1 week	93 (18.3%)	200 (8%)	OR 2.5, 95% CI 1.1–5.4	Mainly respiratory tract infection; noninfective inflammatory events were included; population and hospital controls
Paganini-Hill <i>et al.</i> (2003) <sup>14</sup>	>21 years	1 month	233 (43%)	362 (56%)	No significant difference in the infection rates <sup>†</sup>	Noninfective inflammatory events were included; outpatient controls
Clayton <i>et al.</i> (2008) <sup>15</sup>	>18 years	1–3 days <sup>§</sup>	9,208 (0.4%)	9,208 (0.1%)	OR 4.1, 95% CI 2.0–8.3	Respiratory and urinary tract infections analyzed (no further information on infections provided); controls from primary care database
		1–7 days <sup>§</sup>	9,208 (0.7%)	9,208 (0.4%)	OR 1.9, 95% CI 1.2–3.0	
		8–28 days <sup>§</sup>	9,208 (1.1%)	9,208 (0.7%)	OR 1.8, 95% CI 1.3–2.5	
		29–91 days <sup>§</sup>	9,208 (2.0%)	9,208 (1.9%)	OR 1.1, 95% CI 0.9–1.4	
		92–365 days <sup>§</sup>	9,208 (5.5%)	9,208 (5.0%)	OR 1.1, 95% CI 0.9–1.1	
		1–28 days <sup>  </sup>	9,208 (0.3%)	9,208 (0.1%)	OR 2.7, 95% CI 1.3–5.5	
Zurú <i>et al.</i> (2009) <sup>16†</sup>	73 years (mean age)	1 year	105 (29%)	354 (13%)	OR 4.9, 95% CI 2.3–10.2	Mainly respiratory tract infections; controls from outpatient database

# Infecciones como factor de riesgo de ictus

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

## Risk of Myocardial Infarction and Stroke after Acute Infection or Vaccination

### BACKGROUND

There is evidence that chronic inflammation may promote atherosclerotic disease. We tested the hypothesis that acute infection and vaccination increase the short-term risk of vascular events.

### METHODS

We undertook within-person comparisons, using the case-series method, to study the risks of myocardial infarction and stroke after common vaccinations and naturally occurring infections. The study was based on the United Kingdom General Practice Research Database, which contains computerized medical records of more than 5 million patients.

N Engl J Med 2004;351:2611-8.  
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# Infecciones como factor de riesgo de ictus

	Systemic Respiratory Tract Infection (N=22,400)		Urinary Tract Infection (N=14,603)	
	No. of Cases	IR (95% CI)	No. of Cases	IR (95% CI)
<b>Stroke</b>				
1–3 days	244	3.19 (2.81–3.62)	152	2.72 (2.32–3.20)
4–7 days	237	2.34 (2.05–2.66)	158	2.12 (1.81–2.48)
8–14 days	368	2.09 (1.89–2.32)	245	1.89 (1.65–2.13)
15–28 days	561	1.68 (1.54–1.82)	445	1.71 (1.55–1.88)
29–91 days	1,650	1.33 (1.26–1.40)	1,250	1.22 (1.15–1.30)
Baseline period	18,056	1.00	12,164	1.00



# Infecciones como factor de riesgo de ictus

Cardiovasc Intervent Radiol (2017) 40:1851–1860  
DOI 10.1007/s00270-017-1718-x

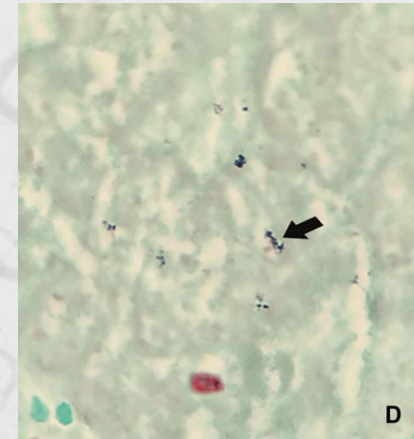


CLINICAL INVESTIGATION

ARTERIAL INTERVENTIONS

## Histopathological and Bacteriological Analysis of Thrombus Material Extracted During Mechanical Thrombectomy in Acute Stroke Patients

Francisco Hernández-Fernández<sup>1</sup> · Laura Rojas-Bartolomé<sup>1</sup> · Jorge García-García<sup>1</sup> · Oscar Ayo-Martín<sup>1</sup> · Juan David Molina-Nuevo<sup>2</sup> · Rosa Angélica Barbella-Aponte<sup>3</sup> · Gemma Serrano-Heras<sup>4</sup> · Enrique Juliá-Molla<sup>2</sup> · María José Pedrosa-Jiménez<sup>2</sup> · Tomás Segura<sup>1</sup>



**Table 2** Summary of the main pre-procedure characteristics of the series of patients with SE

Case number	Sex/age	Arterial occlusion	Leukocytes count (WBC per microliter)	Pre-procedural NIHSS	SE source
1	M, 57	RICA	14,180	13	Prosthetic valve endocarditis <i>S. sanguinis</i>
2	M, 75	LICA	14,960	26	UTI <i>E. Faecalis</i>
3	M, 66	LICA	9600	15	Native valve endocarditis <i>S. bovis</i>
4	F, 76	RICA	8980	19	Staphylococcal pneumonia <i>S. Aureus</i>

*F* female, *M* male, *LICA* left internal carotid artery, *RICA* right internal carotid artery, *SE* septic embolisms, *UTI* urinary tract infection, *WBC* white blood cell

**4/65: 6,2%**

# Infecciones como factor de riesgo de ictus criptogénico

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## OBJETIVO PRINCIPAL

Analizar si en el grupo de ictus embólicos de **origen indeterminado** existe un porcentaje relevante de pacientes con **antecedentes de infección/inflamación** sistémica o local.

## OBJETIVOS SECUNDARIOS

Conocer la relación existente entre tener **antecedentes de infección o inflamación** sistémica o local previa al ictus con:

- Presencia de ictus de una u otra etiología
- **El análisis histopatológico y microbiológico de los trombos**
- Situación funcional al alta hospitalaria

# Infecciones como factor de riesgo de ictus criptogénico

## BASE DATOS DE TROMBECTOMIAS

Recogida de **dos nuevas variables**:

1. Temperatura máxima alcanzada durante el ingreso hospitalario.
2. Si el paciente presentaba antecedente de infección/inflamación sistémica o local previa al ictus isquémico agudo (IPI).

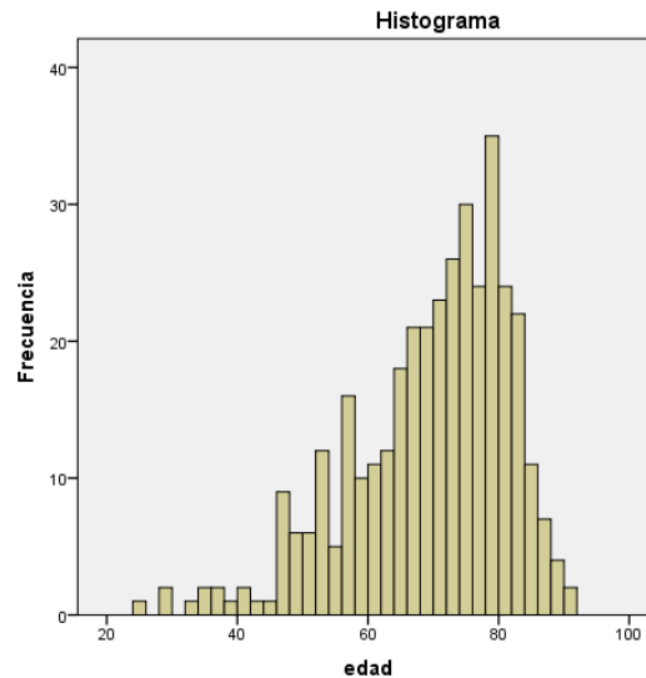
HISTORIAL MÉDICO:  
Infección/inflamación  
mes previo

URGENCIAS:  
Se demuestra  
infección en 1<sup>as</sup>  
24h

URGENCIAS:  
Fiebre 1<sup>as</sup> 12h

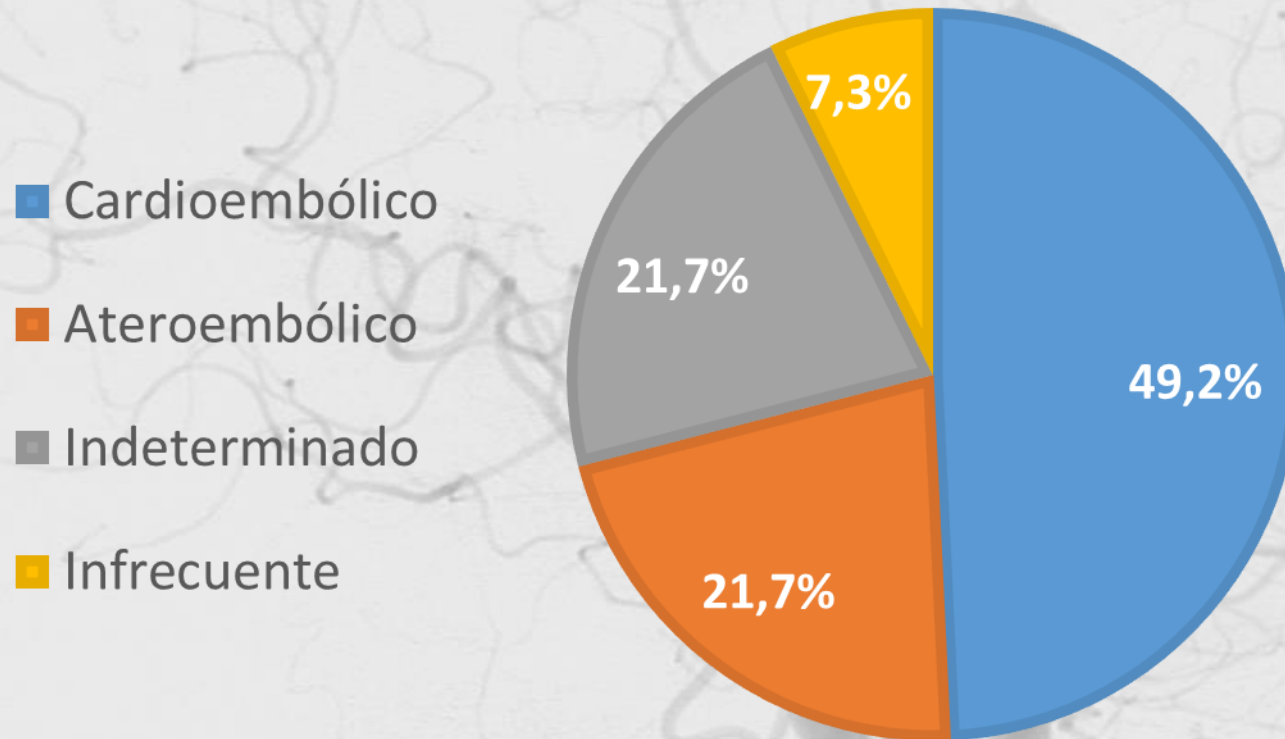
# Infecciones como factor de riesgo de ictus criptogénico

Durante el periodo de junio de 2014 a mayo de 2020, se incluyeron 368 pacientes con ictus isquémico agudo sometidos a trombectomía mecánica (TM). En el total de la muestra el número de hombres fue 222 (60,3%) y de mujeres 146 (39,7%). Los pacientes tenían edades comprendidas entre 25 y 91 años, siendo la edad media 69,1 años, la mediana 72, y el RIQ 62-78 (figura 1).



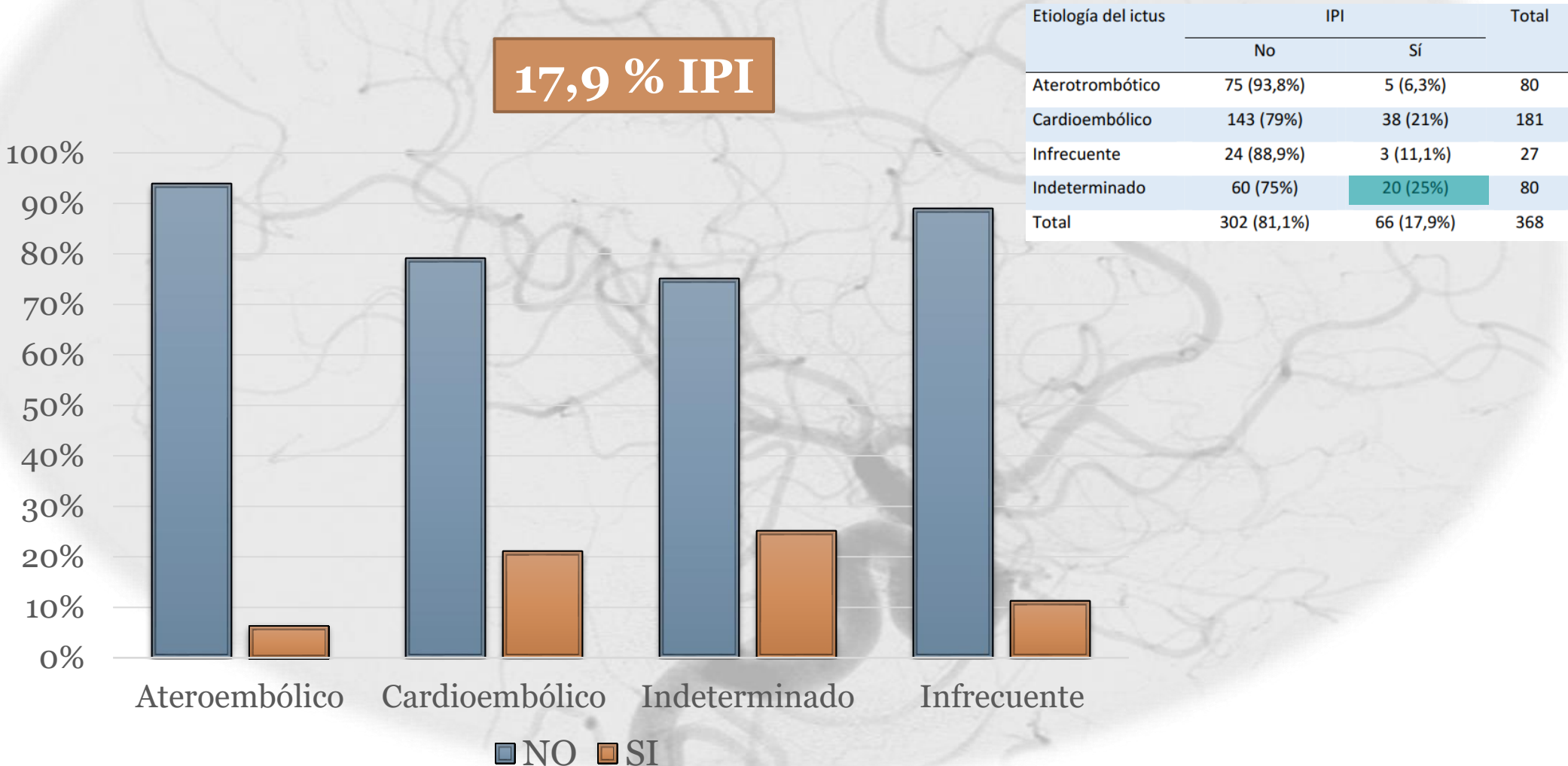
# Infecciones como factor de riesgo de ictus criptogénico

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# Infecciones como factor de riesgo de ictus criptogénico



# Infecciones como factor de riesgo de ictus criptogénico

**Tabla 5:** Relación IPI y PCR al ingreso ( $p=0,044$ ).

PCR al ingreso	IPI	
	No	Sí
n	205	43
Media ( $\pm$ desviación estándar)	44,9 ( $\pm 59,3$ )	74,0 ( $\pm 88,4$ )

# Infecciones como factor de riesgo de ictus criptogénico

## 3.1 Ictus de etiología indeterminada.

Pretendemos explorar qué covariables pueden explicar mejor que al final del proceso hospitalario un ictus sea catalogado como de origen indeterminado (es decir, sin causa conocida). La variable dependiente utilizada fue la categorización al alta del ictus como de origen indeterminado, y las covariables incluidas fueron las siguientes: sexo, edad, glucemia en urgencias, NIHSS inicial en urgencias, IPI y PCR. La única variable que se asociaba de forma independiente con la calificación de “ictus de etiología indeterminada” fue, precisamente, el antecedente de IPI

**Variables en la ecuación**

	B	Error estándar	Wald	gl	Sig.	Exp(B)
Paso 1 <sup>a</sup>						
EDAD_MAS65	-,527	,341	2,389	1	,122	,590
SEXO	-,086	,342	,063	1	,802	,918
GLUCURG_ELEVADA	-,504	,402	1,574	1	,210	,604
IPI	,832	,400	4,320	1	,038	2,297
PCR_ELEVADA	-,236	,343	,473	1	,492	,790
Constante	-,808	,380	4,518	1	,034	,446

a. Variables especificadas en el paso 1: EDAD\_MAS65, SEXO, GLUCURG\_ELEVADA, IPI, PCR\_ELEVADA.

# Infecciones como factor de riesgo de ictus criptogénico

Estudio  
histopatológico y  
microbiológico de  
261 trombos.

3,8% trombos  
sépticos

50% IPI  
( $p=0,0016$ )

Mayor dificultad  
técnica en la  
extracción del trombo

Trombo séptico

Antecedente IPI

# Infecciones como factor de riesgo de ictus criptogénico

**Tabla 6:** Relación entre trombos “rojos” e IPI (p=0,29).

	IPI		Total
	No	Sí	
Trombos no “rojos”	167 (81,5%)	38 (18,5%)	205
Trombos “rojos”	49 (87,5%)	7 (12,5%)	56
Total	216	45	261

**Tabla 7:** Relación entre trombos “blancos” e IPI (p=0,89).

	IPI		Total
	No	Sí	
Trombos no “blancos”	108 (82,4%)	23 (17,6%)	131
Trombos “blancos”	108 (83,1%)	22 (16,9%)	130
Total	216	45	261

**Tabla 8:** Relación entre trombos sépticos e IPI (p=0,016).

	IPI		Total
	No	Sí	
Trombos no sépticos	211 (84,1%)	40 (15,9%)	251
Trombos sépticos	5 (50%)	5 (50%)	10
Total	216	45	261



# Infecciones como factor de riesgo de ictus criptogénico

Ictus embólicos  
de origen  
indeterminado

Porcentaje relevante de  
pacientes con IPI

Asociación  
independiente

Antecedente de  
IPI

Ictus cardioembólicos e indeterminados



Histopatología de los trombos o con la dificultad técnica  
en su extracción mediante TM

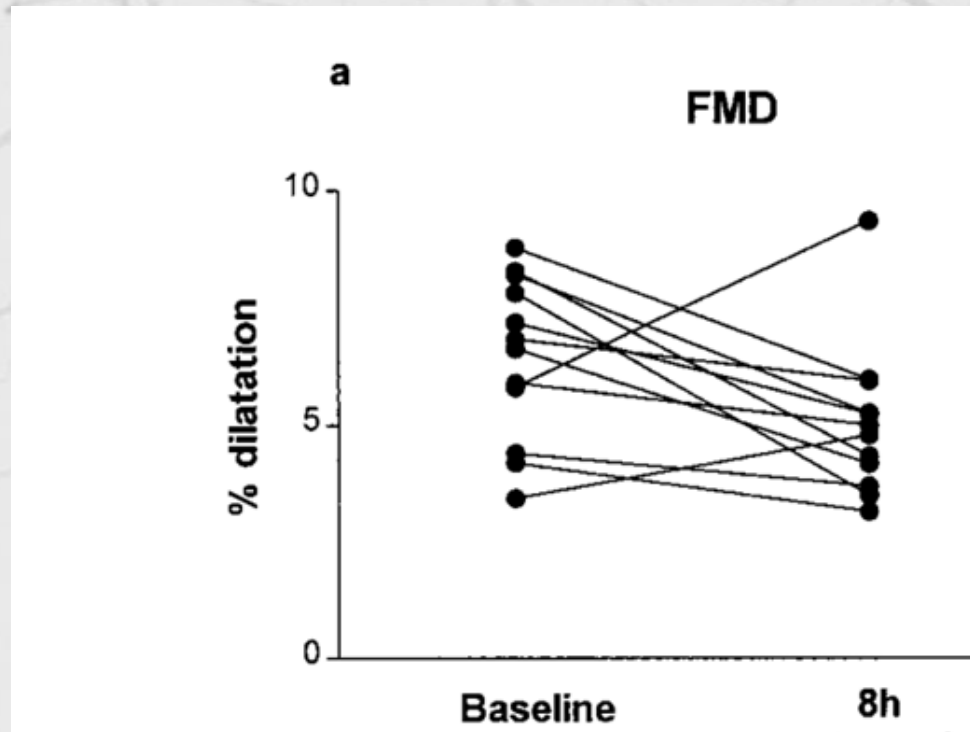
Trombos sépticos

# Mecanismos potenciales de asociación Ictus In/If

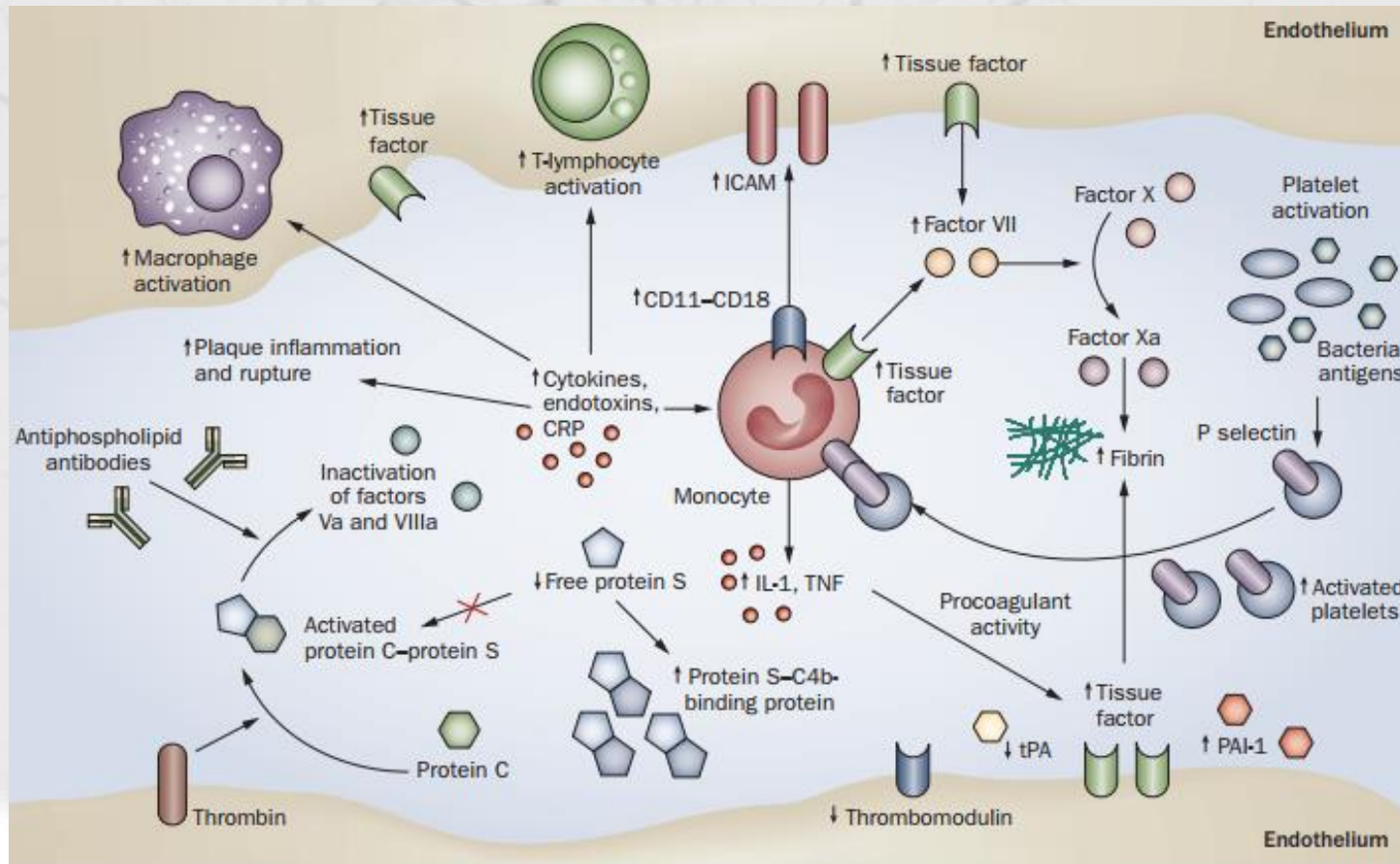
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- ❑ **La existencia de una reacción sistémica infecciosa o inflamatoria conlleva un estado protrombótico más o menos intenso**
- ❑ **Son condicionantes genéticos (del individuo) y antigénicos (del microorganismo) los que provocarán una respuesta más o menos intensa – y por tanto potencialmente patogénica- de la respuesta procoagulante**
- ❑ **No parece tan importante como se pensó la existencia de patología arterial previa (pero sí quizá otros cofactores: deshidratación, inmovilidad, fármacos)**

# Mecanismos potenciales de asociación Ictus In/If



# Mecanismos potenciales de asociación Ictus In/If





# Mecanismos potenciales de asociación Ictus In/If

## Acute respiratory tract infection leads to procoagulant changes in human subjects

*J Thromb Haemost* 2011; **9**: 1432–4.

M. VAN WISSEN,\* T. T. KELLER,† E. C. M. VAN GORP,\*‡ V. E. A. GERDES,\*† J. C. M. MEIJERS,† G. J. J. VAN DOORNUM,‡ H. R. BÜLLER† and D. P. M. BRANDJES\*

\*Department of Internal Medicine, Slotervaart Hospital, Amsterdam; †Department of Vascular Medicine, Academic Medical Center, University of Amsterdam, Amst

Amsterdam, Amst

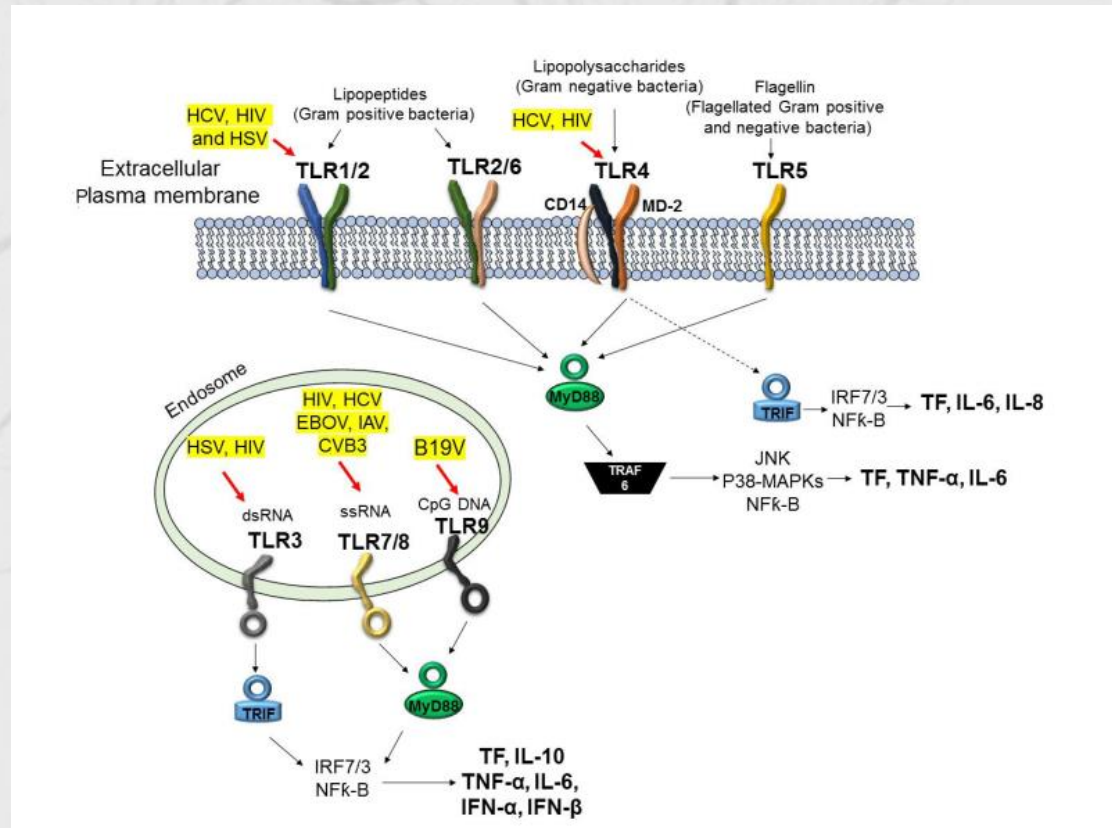
To cite this article  
respiratory tract in

	Median levels of hs-CRP and hemostatic proteins			
	T = 0	T = 1	T = 2	T = 3
hs-CRP (mg L <sup>-1</sup> )	2.6 (1.3–3.8)	46.8 (21.2–114)*	13.9 (5.0–47.7)*	2.6 (2.2–8.0)
vWF antigen (%)	123 (82–166)	211 (157–247)*	196 (148–237)*	129 (102–195)†
F1 + 2 (pmol L <sup>-1</sup> )‡	247 (206–326)	200 (170–317)	235 (186–273)	218 (187–300)
PAP (µg L <sup>-1</sup> )	458 (347–605)	673 (438–1041)*	778 (602–1029)*	516 (404–647)
D-Dimer (mg L <sup>-1</sup> FEU)‡	0.32 (0.24–0.56)	0.53 (0.37–0.70)*	0.59 (0.45–0.80)*	0.55 (0.44–0.98)*
PAI-1 (IU mL <sup>-1</sup> )	4.4 (3.1–5.2)	4.2 (3.6–5.5)	4.5 (4.0–5.3)	5.2 (4.4–5.9)†
Peak thrombin (nm) <sup>‡§</sup>	311 (269–356)	376 (335–414)*	401 (367–447)*	404 (359–432)*
Vel. Index (nm min <sup>-1</sup> ) <sup>‡§</sup>	121 (73–138)	168 (141–187)*	180 (161–212)*	160 (141–202)*
ETP (nm min <sup>-1</sup> ) <sup>‡§</sup>	1803 (1660–2067)	2009 (1661–2298)*	2041 (1851–2485)*	2155 (1846–2456)*
APC-sr <sup>‡</sup>				
Men (n = 6)	0.73 (0.59–1.78)	1.62 (0.99–3.65)	1.70 (1.21–3.95)	2.07 (1.40–4.60)†
Women (n = 8)	0.37 (0.05–0.67)	0.90 (0.35–1.32)*	1.26 (0.46–2.39)*	0.93 (0.43–1.60)*
Men + women	0.65 (0.26–0.78)	1.05 (0.63–2.12)*	1.60 (0.97–2.63)*	1.29 (0.74–1.98)*

Number of subjects: 15, with 11 confirmed infections in 10 subjects; infection could not be proven in 5 subjects. None of the female subjects were using hormone replacement therapy.

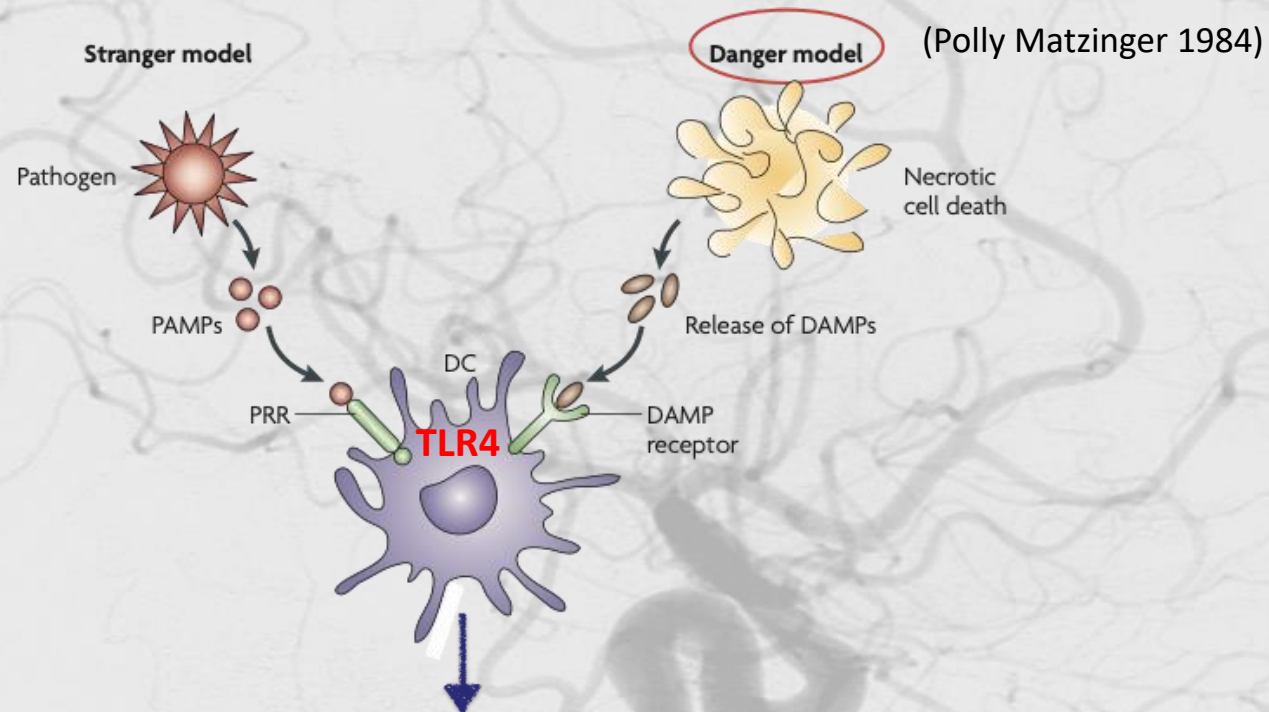
Numbers are medians with interquartile range (IQR) in parentheses. T = 0: baseline; T = 1: acute phase (influenza-like illness); T = 2: 2–3 days after T = 1; T = 3: 14 days after T = 1.

# Mecanismos potenciales de asociación Ictus In/If





# Mecanismos potenciales de asociación Ictus In/If

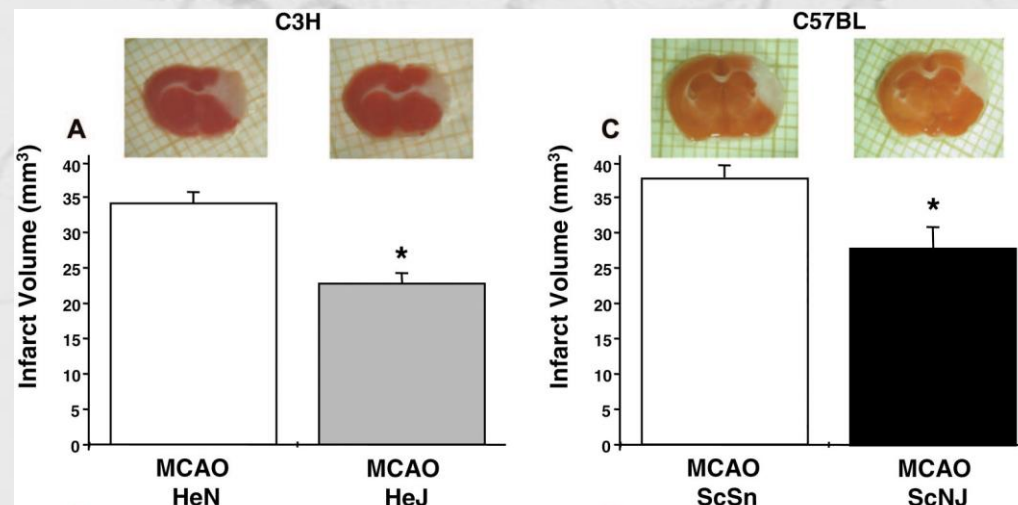


# Mecanismos potenciales de asociación Ictus In/If

## Toll-Like Receptor 4 Is Involved in Brain Damage and Inflammation After Experimental Stroke

Javier R. Caso, Jesús M. Pradillo, Olivia Hurtado, Pedro Lorenzo, María A. Moro and Ignacio Lizasoain

*Circulation.* 2007;115:1599-1608.

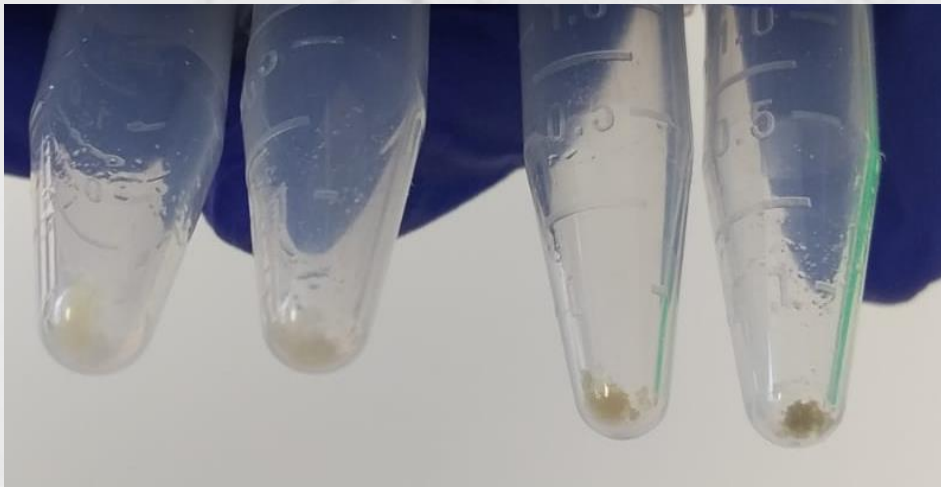


# Resumen

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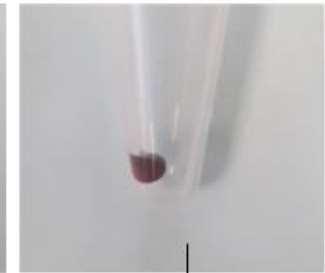
- ❑ La existencia de una reacción sistémica infecciosa o inflamatoria es un factor de riesgo de ictus isquémico posiblemente por su capacidad para generar un estado protrombótico (que variará en función del microorganismo y del individuo)
- ❑ El actor principal de esa respuesta es la Célula Endotelial, y en un lugar relevante pero secundario se situarían neutrófilos y plaquetas
- ❑ El hecho de que la actividad procoagulante esté fuertemente ligada a la inmunidad innata demuestra que se trata de una respuesta filogenéticamente muy antigua
- ❑ Sin embargo es diferente para cada especie, para cada tipo celular y para cada microorganismo, lo que complica mucho su estudio

# Mecanismos potenciales de asociación Ictus In/If

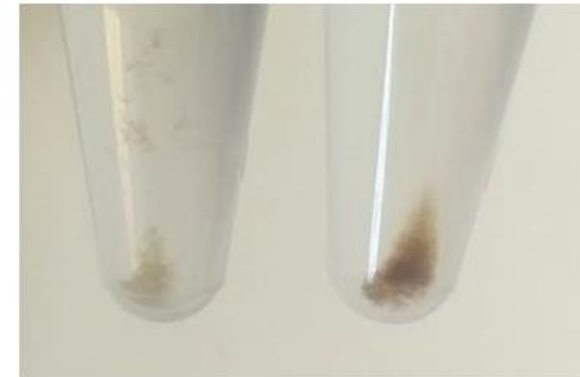


Trombo-control 3  
(material sin procesar)

Trombo-COVID 3  
(material sin procesar)



Extracción de proteínas  
para proteómica



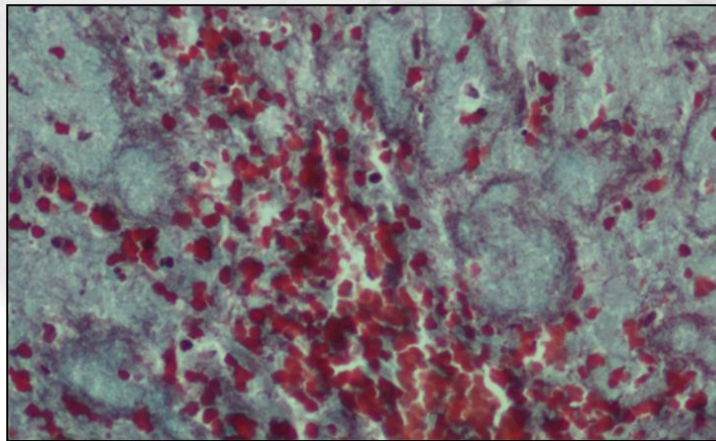
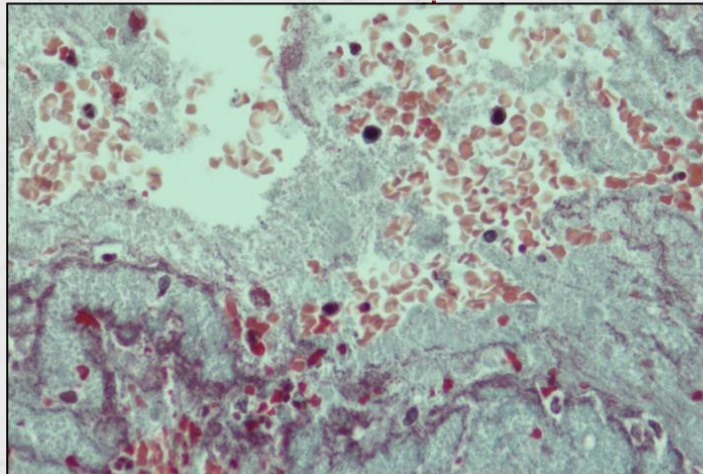
Trombo-control 3  
(Homogenizado)

Trombo-COVID 3  
(Homogenizado)

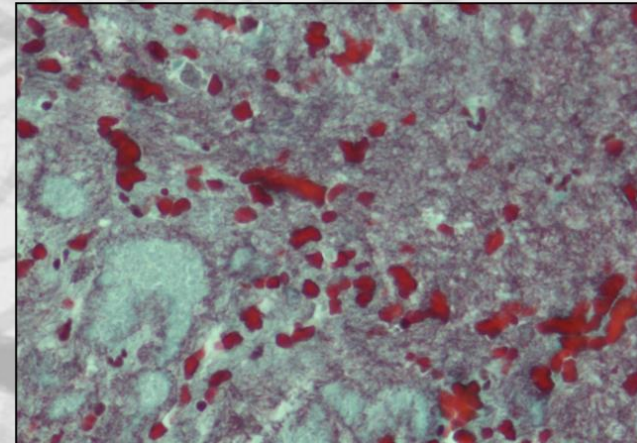
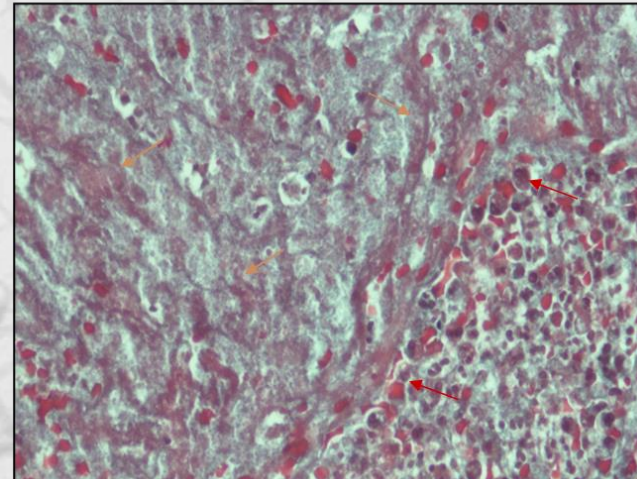


# Mecanismos potenciales de asociación Ictus In/If

NON-COVID



COVID



teru Kulo ederim Tinotenda Multumesc N'éá'eshe Càám dзиакује Gunasakulila Webale Imela maith Fafetai asko Kiitos mamexes blu Puno todá Ah hvala Ashi Uzbezco Gratias Netjer Rahmet Danke Dios raibh

olun rhat Paldies Moltes Dankon Maketai Xié Barka Maraba Tánan Mantióx Murakoze Tack leibh qui Pai Matóndo Tsín'aen Merçi Takk Matu

Grazzie Ntyox dimo Kommol Dekoju Ka Khawp Shokrán Arigato Sag aghabh Shukuriyyaa Emitekatí Tesekkür Trugarez so Ashoge

Fa'afetai gracies Fa'afetai gracies Aalghístapcham Marahaba òn Faleminderit Dyuspagrasunki Shterakravetsun Tashakkur Bulgaro Rakhmat Go Obridado suksama

chawe Mercí Hvala magah Dyakooyu Kili maluhlap Mahalo

Yuspagara Ngeyabonga

Dannaba Mwebare Tánan

Ngiiyabonga

# Gracias



