Quoi de neuf en 2020 sur les SHE ?

3ème journée du CEREO
Maison de l'Amérique Latine
25 sept 2020

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Plan

1. **Leucémie chronique à éosinophiles liée à FIP1L1-PDGFRA**

2. **Autres SHE myéloïdes (non liés à FIP1L1-PDGFRA)**

3. **SHE lymphoïde 3-4+**

4. **Formes de chevauchement**
   - ***Gastroentérites à éosinophiles***
   - ***Pneumopathies chroniques à éosinophiles (Carrington)***
   - ***Granulomatose éosinophilique avec polyangéite (Churg-Strauss)***

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Leucémie chronique à éosinophiles FIP1L1-PDGFRA

Frequent false-negative *FIP1L1-PDGFRA* FISH analyses of bone marrow samples from clonal eosinophilia at diagnosis

Linda Olsson-Arvidsson[1]
Anna Norberg[2]
Helene Sjögren[3]
Bertil Johansson[1,4]

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Leucémie chronique à éosinophiles FIP1L1-PDGFRA

Frequent false-negative *FIP1L1-PDGFRA* FISH analyses of bone marrow samples from clonal eosinophilia at diagnosis

Table I. *FIP1L1-PDGFRA*/del(4)(q12q12)-positive cases analysed by FISH, RT-PCR, SNP-A, and RNA-Seq.

			FIP1L1-PDGFRA/del(4)(q12q12)-positive					
Case No.	Sex/age (years)	Sample analysed	FISH (Lund)	FISH (Umeå)	FISH (Gothen-burg)	RT-PCR (Lund)	SNP-A (Lund)	RNA-Seq (Lund)
1	M/49	BM/PB	ND	ND	ND	Yes*	ND	ND
2	M/57	PB	ND	ND	ND	Yes	ND	ND
3	M/60	BM	Yes	ND	ND	ND	ND	ND
4	M/67	BM	Yes	ND	ND	Yes†	ND	ND
5	M/61	BM	**No‡**	**No**	**No**	Yes*	ND	No
6	M/46	BM	Yes	ND	ND	ND	ND	ND
7	F/28	BM	Yes	Yes	Yes	Yes*	Yes	Yes
8	M/72	BM	No	ND	ND	Yes†	No	No
9	M/57	BM	No	ND	ND	Yes§	No	ND

BM, bone marrow; F, female; FISH, fluorescence *in situ* hybridisation; M, male; ND, not done; PB, peripheral blood; RNA-Seq, whole transcriptome sequencing; RT-PCR, reverse transcription polymerase chain reaction; SNP-A, single nucleotide polymorphism array.

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7	F/28	BM	Yes	Yes	Yes	Yes*	Yes	Yes
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Leucémie chronique à éosinophiles FIP1L1-PDGFRA

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RESEARCH ARTICLE

AJH WILEY

Epidemiology, clinical picture and long-term outcomes of FIP1L1-PDGFRA-positive myeloid neoplasm with eosinophilia: Data from 151 patients

Julien Rohmer[1,2] | Amélie Couteau-Chardon[1,3] | Julie Trichereau[1,4] | Kewin Panel[1,4] | Cyrielle Gesquiere[1] | Raouf Ben Abdelali[5] | Audrey Bidet[6] | Jean-Sébastien Bladé[7] | Jean-Michel Cayuela[8] | Pascale Cony-Makhoul[1,9] | Vincent Cottin[10,11] | Eric Delabesse[12] | Mikaël Ebbo[1,13] | Olivier Fain[14] | Pascale Flandrin[15] | Lionel Galicier[16] | Catherine Godon[17] | Nathalie Grardel[18] | Aurélien Guffroy[1,19] | Mohamed Hamidou[1,20] | Mathilde Hunault[21] | Etienne Lengline[22] | Faustine Lhomme[23] | Ludovic Lhermitte[24] | Irène Machelart[1,25] | Laurent Mauvieux[26] | Catherine Mohr[27] | Marie-Joelle Mozicconacci[28] | Dina Naguib[29] | Franck E. Nicolini[30] | Jerome Rey[31] | Philippe Rousselot[32] | Suzanne Tavitian[33] | Louis Terriou[1,34] | Guillaume Lefèvre[1,34] | Claude Preudhomme[18] | Jean-Emmanuel Kahn[1,35] | Matthieu Groh[1,2] | CEREO and GBMHM collaborators

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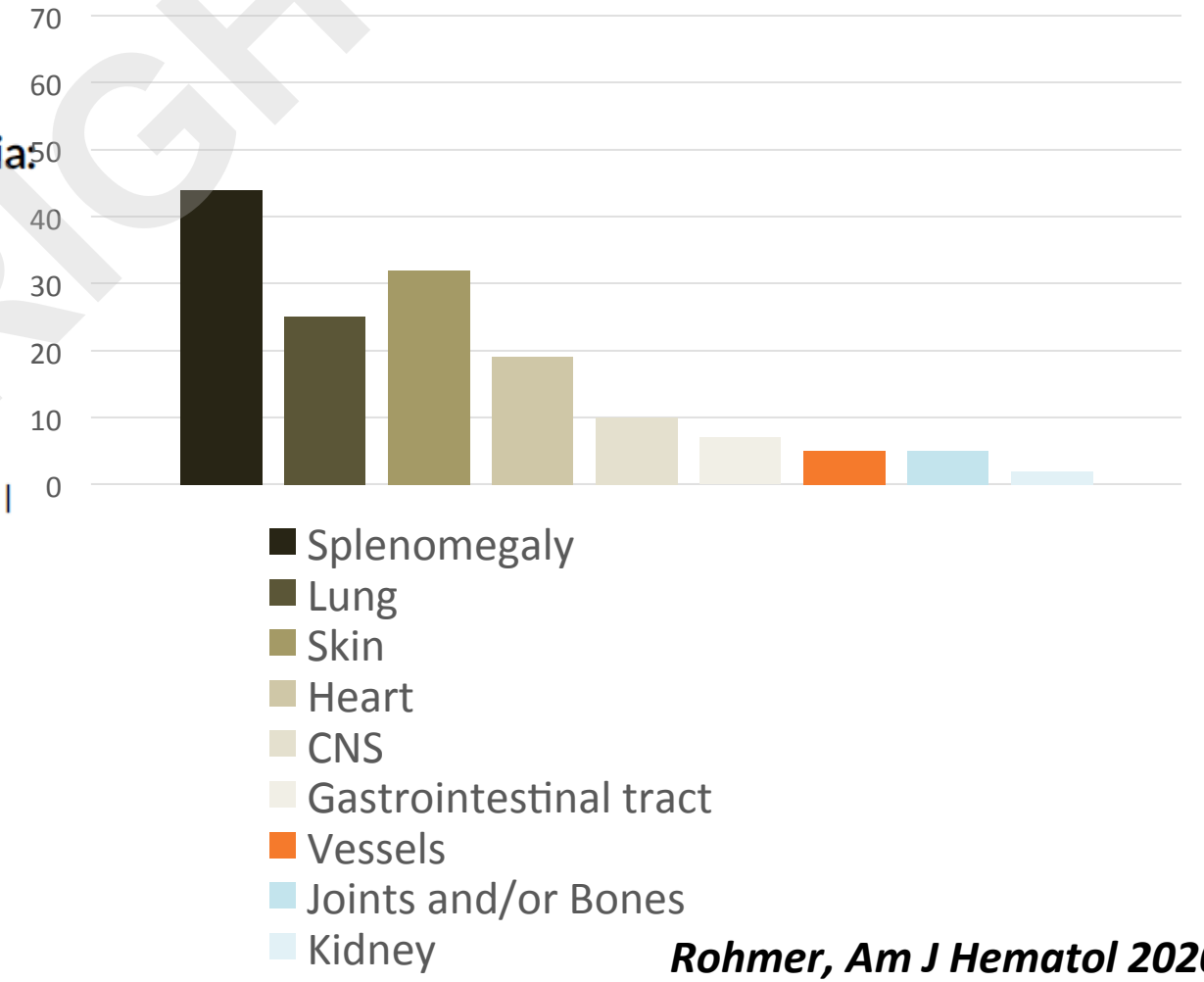
- ✓ Incidence moyenne: 0.18 cas/million/an
- ✓ 143 hommes (sexe ratio 18/1)



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Proportion de patients avec 0,1,2, 3 ou 4 paramètres:

< 10%
10-30%
>30%

Paramètres :

- Vitamine B12 > 700 pg/ml
- Tryptase > 15 ng/ml
- Sexe masculin
- Splénomégalie

	n	n=44
0	0	0%
1	1	2%
2	5	11%
3	22	50%
4	16	36%

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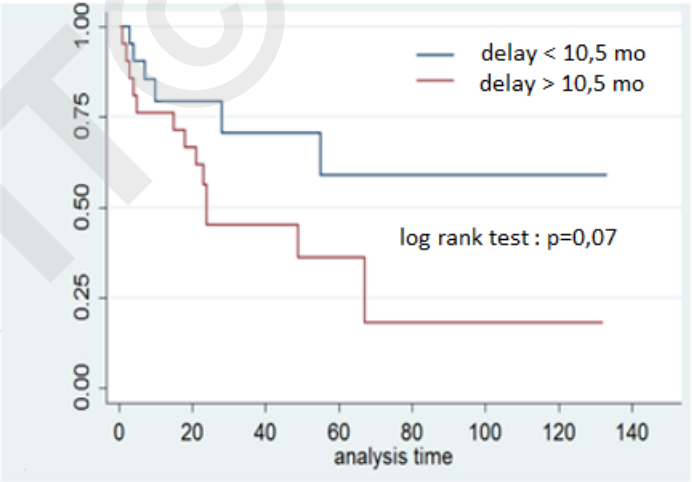

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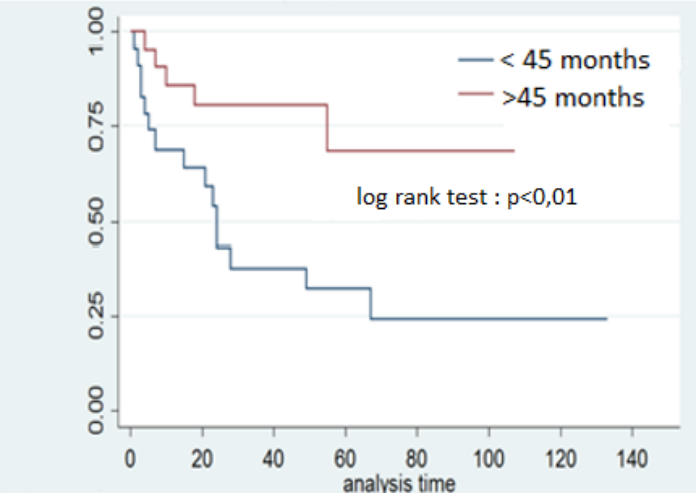

Durée de traitement par IM

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Leucémie chronique à éosinophiles FIP1L1-PDGFRA

Treatment-free remission in *FIP1L1-PDGFRA*–positive myeloid/lymphoid neoplasms with eosinophilia after imatinib discontinuation

Georgia Metzgeroth,[1] Juliana Schwaab,[1] Nicole Naumann,[1] Mohamad Jawhar,[1] Torsten Haferlach,[2] Alice Fabarius,[1] Andreas Hochhaus,[3] Wolf-Karsten Hofmann,[1] Nicholas C. P. Cross,[4,5] and Andreas Reiter[1]

[1]Department of Hematology and Oncology, University Hospital Mannheim, Heidelberg University, Mannheim, Germany; [2]Munich Leukemia Laboratory, Munich, Germany; [3]Klinik für Innere Medizin II, Universitätsklinikum Jena, Jena, Germany; [4]Wessex Regional Genetics Laboratory, Salisbury, United Kingdom; and [5]Faculty of Medicine, University of Southampton, Southampton, United Kingdom

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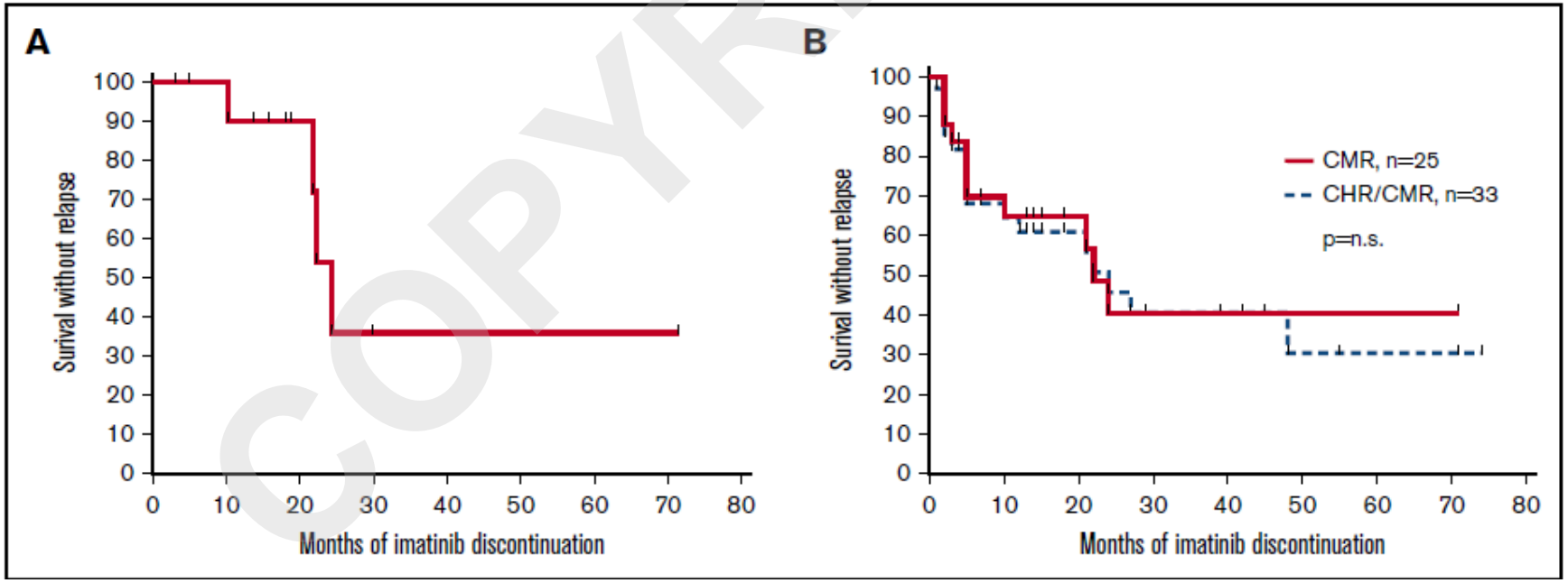

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Néoplasies myéloïdes liées à PCM1-JAK2, BCR-JAK2 et ETV6-ABL1

RESEARCH ARTICLE

AJH WILEY

Response to tyrosine kinase inhibitors in myeloid neoplasms associated with *PCM1-JAK2*, *BCR-JAK2* and *ETV6-ABL1* fusion genes

Juliana Schwaab[1] | Nicole Naumann[1] | Johannes Luebke[1] | Mohamad Jawhar[1] | Tim C.P. Somervaille[2,3] | Mark S. Williams[2,3] | Rebecca Frewin[4] | Philipp J. Jost[5] | Felix S. Lichtenegger[6] | Paul La Rosée[7] | Nicola Storch[8] | Torsten Haferlach[9] | Hans-Peter Horny[10] | Alice Fabarius[1] | Claudia Haferlach[9] | Andreas Burchert[11] | Wolf-Karsten Hofmann[1] | Nicholas C.P. Cross[12,13] | Andreas Hochhaus[14] | Andreas Reiter[1] | Georgia Metzgeroth[1]

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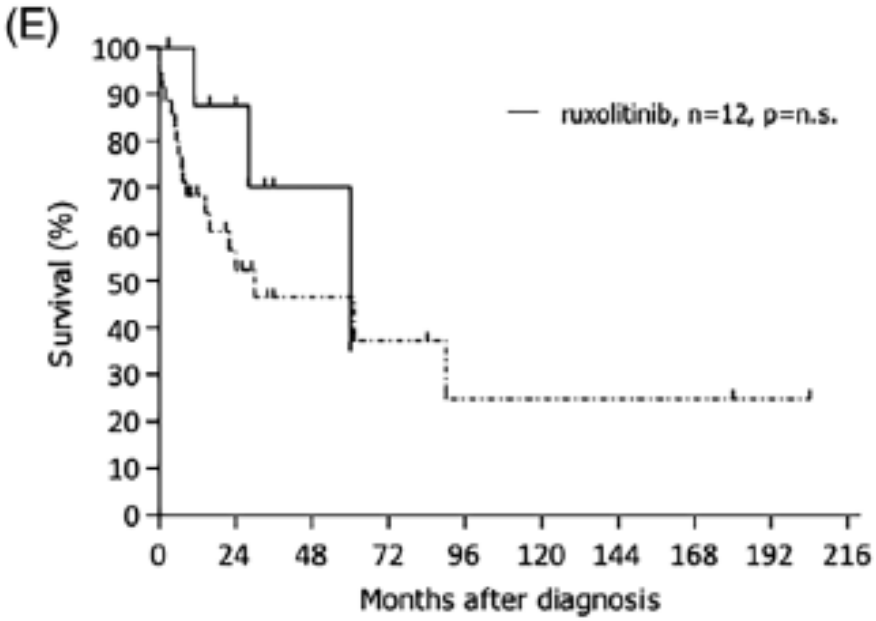

Réarrangements JAK2

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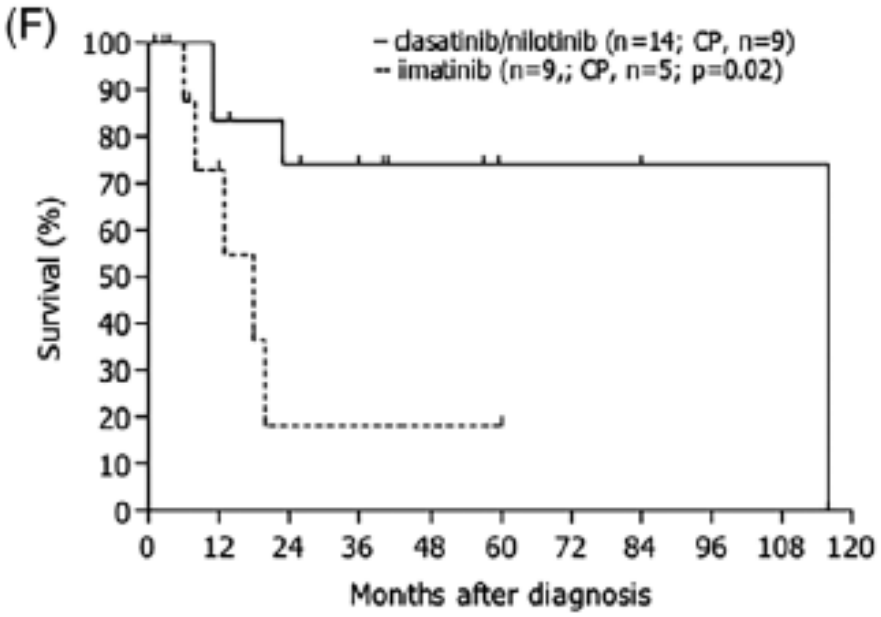

ETV6-ABL1

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Leucémie chronique à éosinophiles NOS

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WHO defined chronic eosinophilic leukemia, not otherwise specified (CEL, NOS): A contemporary series from the Mayo Clinic

Erika Morsia[1], Kaaren Reichard[2], Animesh Pardanani[1], Ayalew Tefferi[1], Naseema Gangat[1]

[1]Division of Hematology, Mayo Clinic, Rochester, Minnesota
[2]Division of Hematopathology, Mayo Clinic, Rochester, Minnesota

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[1]Division of Hematology, Mayo Clinic, Rochester, Minnesota
[2]Division of Hematopathology, Mayo Clinic, Rochester, Minnesota

- ✓ N=17 patients
  - ➢ Anomalies cytogénétiques: 15/17 patients (trisomie 8, caryotype complexe…)
  - ➢ 7/7 patients avec anomalies moléculaires NGS (*ASXL1*, *IDH1*, *TP53*…) mais pas de VAF
- ✓ 1/3 de RHC sous corticothérapie
- ✓ Médiane de suivi 13mois
- ✓ 9 décès (infections, n=3; LAM, n=3)
- ✓ F prédictifs de transformation (univariée)
  - ➢ PNE atypiques (PB ou BM)
  - ➢ Myélofibrose marquée
  - ➢ Caryotype complexe
  - ➢ Absence de RHC

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SHE lymphoïde 3-4+

frontiers in Immunology

Eosinophilia Associated With $CD3^{-}CD4^{+}$ T Cells: Characterization and Outcome of a Single-Center Cohort of 26 Patients

Caroline Carpentier[1†], Sylvain Verbanck[1†], Liliane Schandené[2], Pierre Heimann[3], Anne-Laure Trépant[4], Elie Cogan[1] and Florence Roufosse[1,5]*

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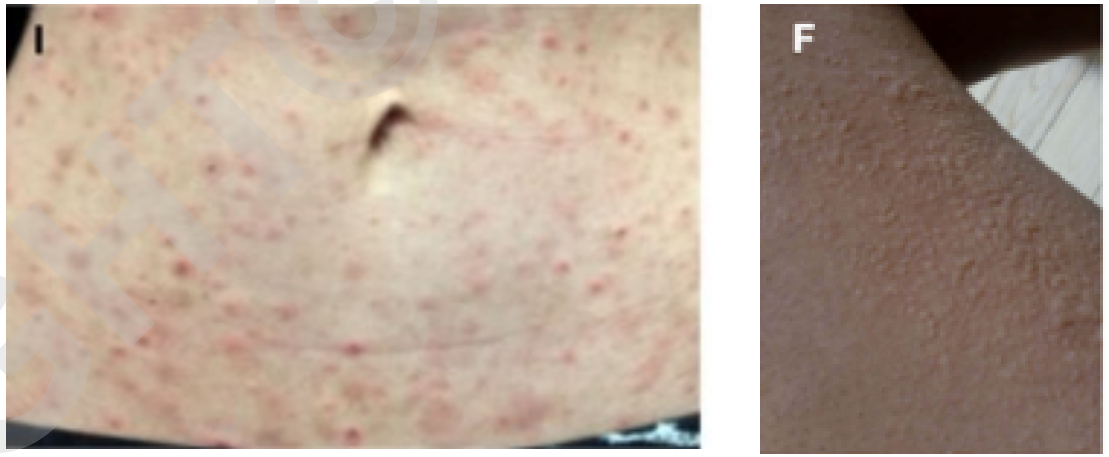

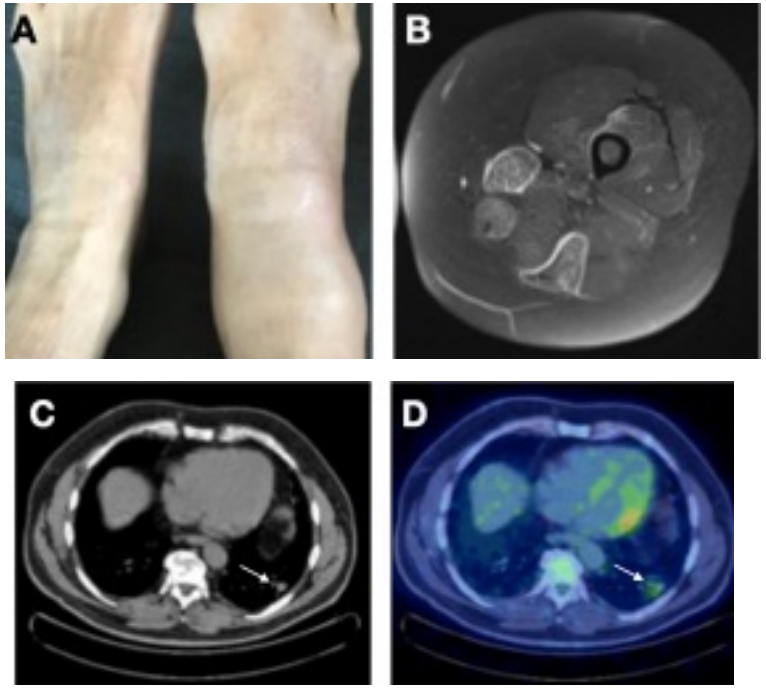

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- ✓ Tryptase <15 ng/mL chez tous patients
- ✓ IgE totales ↑: 14/25 (56%)
- ✓ CCL17/TARC ↑: 22/23 (96%)
- ✓ Hyperγ polyclonale: 13/26 (50%)
- ✓ CRP ↑: 2/23 (9%)
- ✓ 3/26 (12%) progressent vers LNH-T

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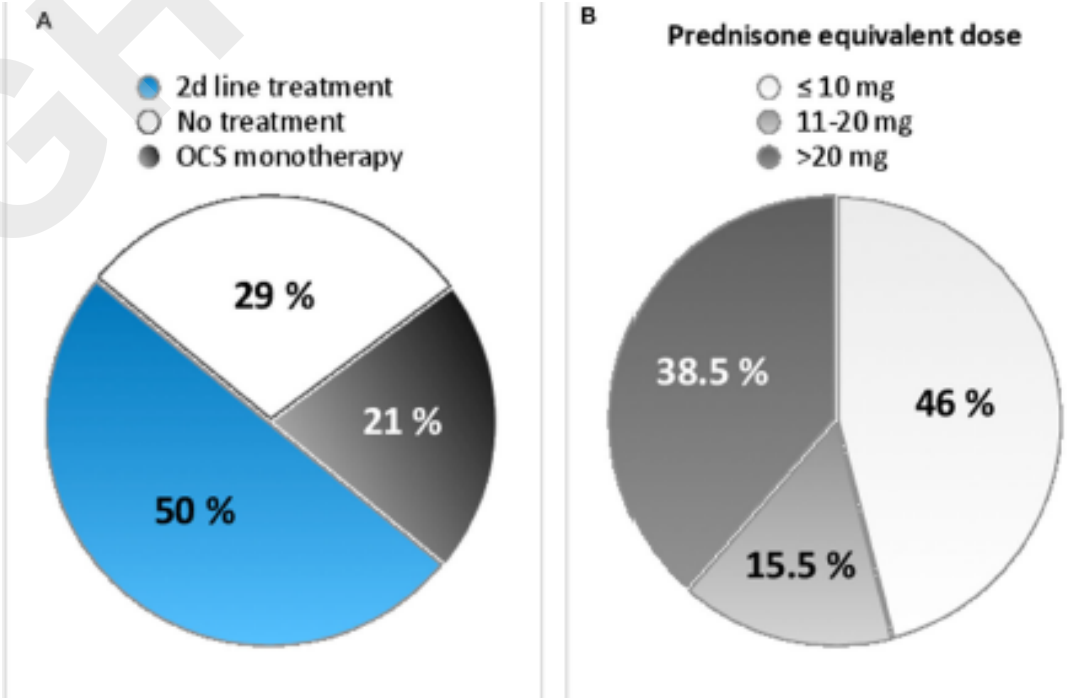

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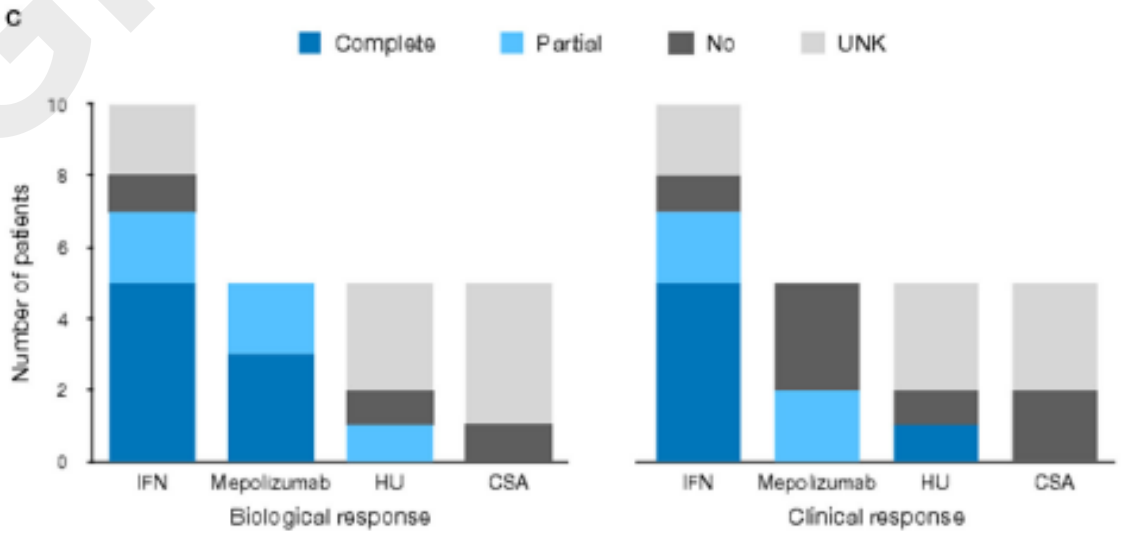

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SHE lymphoïde 3-4+: vers de nouveaux TTT ?

bjh correspondence

Pegylated interferon alpha 2a is an effective and well-tolerated treatment option for lymphocyte-variant hypereosinophilic syndrome

Choi, Br J Hematol 2020

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Choi, Br J Hematol 2020

Dramatic and Durable Responses to Romidepsin in Two Patients With the Lymphocytic Variant of Hypereosinophilic Syndrome

Aleeza Abbasi,[1] Catherine Chung,[2] Basem M. William[1]

Abbasi, Clin Lymphoma Myeloma Leuk 2020

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Aleeza Abbasi,[1] Catherine Chung,[2] Basem M. William[1]

Abbasi, Clin Lymphoma Myeloma Leuk 2020

Clinical Communications

Assessment of T-cell polarization on the basis of surface marker expression: Diagnosis and potential therapeutic implications in lymphocytic variant hypereosinophilic syndrome

Ledoult, JACI In Pract 2020

Clinical Implications

- Lymphocytic variant hypereosinophilic syndrome ($_L$HES)-associated $CD3^-CD4^+$ T cells in blood homogeneously express chemokine receptors associated with T_H2 polarization ($CCR_6^-CCR_4^+$)—even if the patients are treated with steroids. New treatment strategies such as $CRTh_2$ antagonists and anti-CCR_4 antibody could be considered in severe $_L$HES.

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SHE lymphoïde 3-4+: vers de nouveaux TTT ?

bjh correspondence

Pegylated interferon alpha 2a is an effective and well-tolerated treatment option for lymphocyte-variant hypereosinophilic syndrome

Choi, Br J Hematol 2020

Dramatic and Durable Responses to Romidepsin in Two Patients With the Lymphocytic Variant of Hypereosinophilic Syndrome

Aleeza Abbasi,[1] Catherine Chung,[2] Basem M. William[1]

Abbasi, Clin Lymphoma Myeloma Leuk 2020

Clinical Communications

Assessment of T-cell polarization on the basis of surface marker expression: Diagnosis and potential therapeutic implications in lymphocytic variant hypereosinophilic syndrome

Ledoult, JACI In Pract 2020

Clinical Implications

- Lymphocytic variant hypereosinophilic syndrome ($_L$HES)-associated $CD3^-CD4^+$ T cells in blood homogeneously express chemokine receptors associated with T_H2 polarization ($CCR_6^-CCR_4^+$)—even if the patients are treated with steroids. New treatment strategies such as $CRTh_2$ antagonists and anti-CCR_4 antibody could be considered in severe $_L$HES.

➢ **TTT par mogamulizumab ?**

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Gastro-entérites à éosinophiles

Original Article

Single-Organ and Multisystem Hypereosinophilic Syndrome Patients with Gastrointestinal Manifestations Share Common Characteristics

Fei Li Kuang, MD, PhD[a,b], Bryan F. Curtin, MD[c], Hawwa Alao, MD[c], Brent Piligian, BS[a], Alexis Berry, BS[a], Nicole Holland-Thomas, RN, BSN[a], Astin Powers, MD[d], Martha Quezado, MD[d], Keith Lumbard, MS[e], Michael P. Fay, PhD[f], Amy D. Klion, MD[a], Sheila Kumar, MD, MS[c], and Paneez Khoury, MD, MHSc[a] *Bethesda, Md; and Chicago, Ill*

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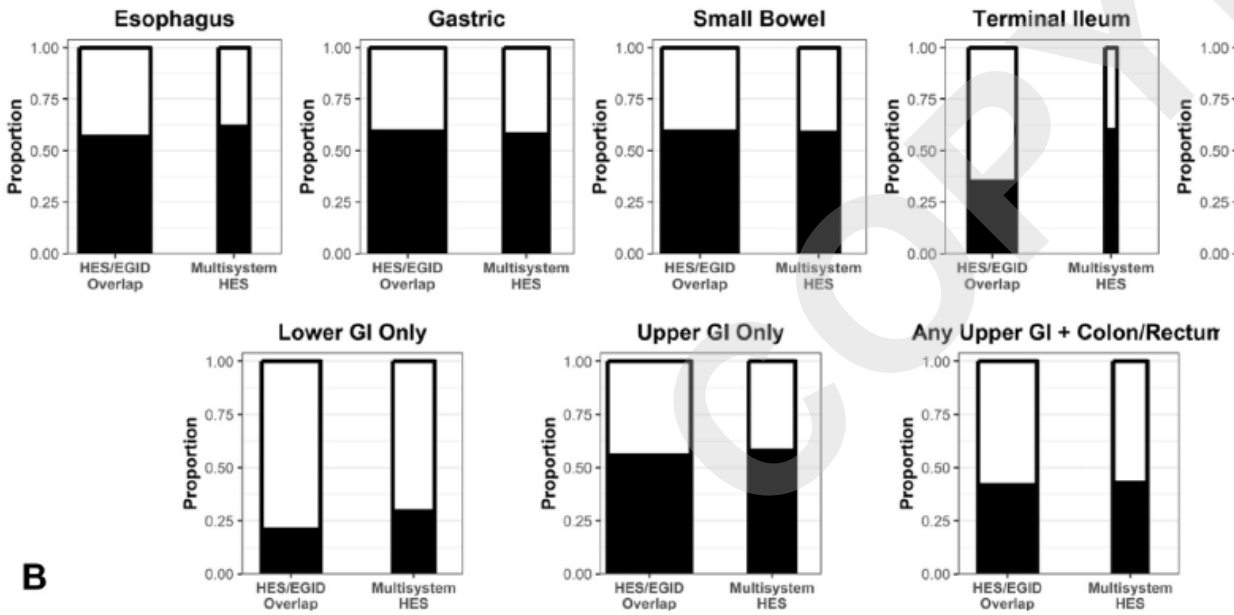
Gastro-entérites à éosinophiles

Original Article

Single-Organ and Multisystem Hypereosinophilic Syndrome Patients with Gastrointestinal Manifestations Share Common Characteristics

Fei Li Kuang, MD, PhD[a,b], Bryan F. Curtin, MD[c], Hawwa Alao, MD[c], Brent Piligian, BS[a], Alexis Berry, BS[a], Nicole Holland-Thomas, RN, BSN[a], Astin Powers, MD[d], Martha Quezado, MD[d], Keith Lumbard, MS[e], Michael P. Fay, PhD[f], Amy D. Klion, MD[a], Sheila Kumar, MD, MS[c], and Paneez Khoury, MD, MHSc[a] *Bethesda, Md; and Chicago, Ill*



- ✓ EGID « isolée » (n=34) *vs*. SHE « systémique » (n=22, tous s/types)
- ✓ Présentation clinicobiologique similaire
- ✓ 2/3 patients ont atteinte multi-étagée
- ✓ 1/3 des patients « systémiques » débutent par EGID
- ✓ Formes systémiques:moins de régime mais plus de TTT généraux

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Pneumopathies chroniques idiopathiques à éosinophiles

CHRONIC EOSINOPHILIC PNEUMONIA

Real-Life Study of Mepolizumab in Idiopathic Chronic Eosinophilic Pneumonia

Emeline Brenard[1] · Charles Pilette[1] · Caroline Dahlqvist[2] · Benoît Colinet[3] · Florence Schleich[4] · Florence Roufosse[5] · Antoine Froidure[1]

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Brenard, Lung 2020

Pneumopathies chroniques idiopathiques à éosinophiles

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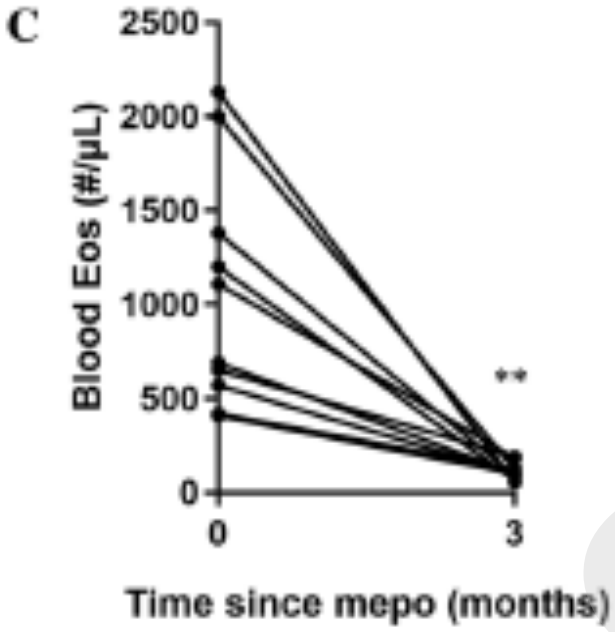

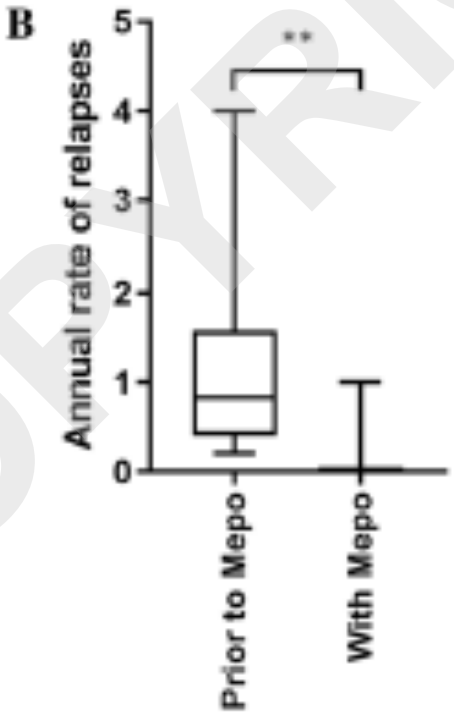

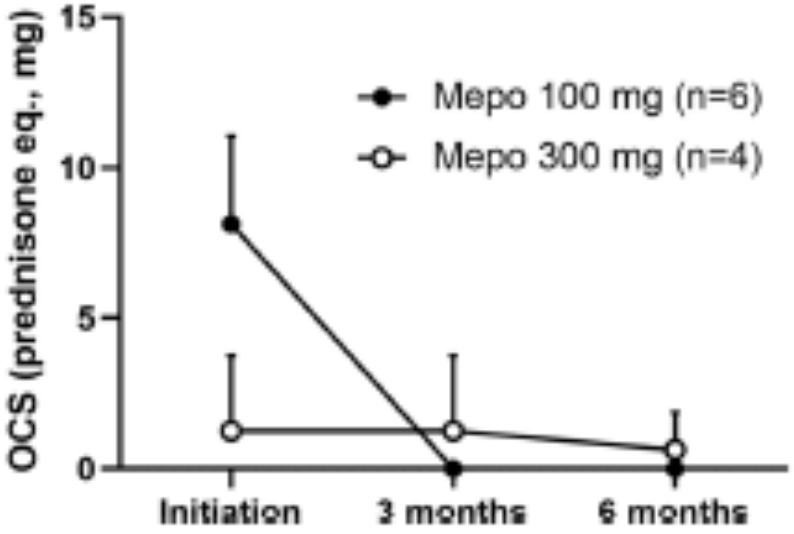

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Brenard, Lung 2020

Granulomatose Eosinophilique avec Polyangéite

ARTICLE

Differential clinicopathologic features of EGPA-associated neuropathy with and without ANCA

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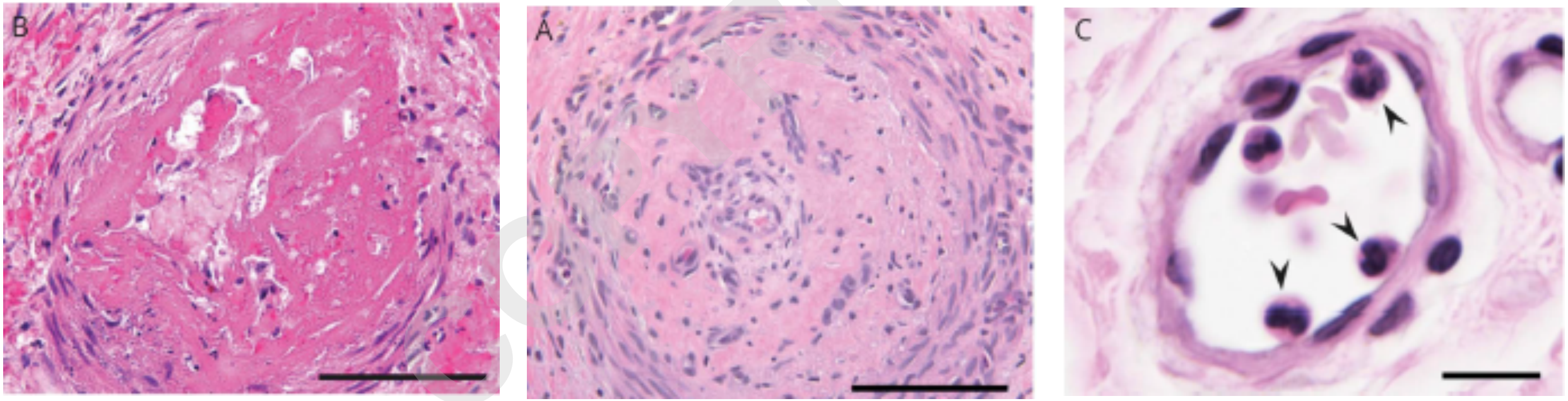
Nishi, Neurology 2020

Granulomatose Eosinophilique avec Polyangéite

ARTICLE

Differential clinicopathologic features of EGPA-associated neuropathy with and without ANCA



ANCA MPO-POS

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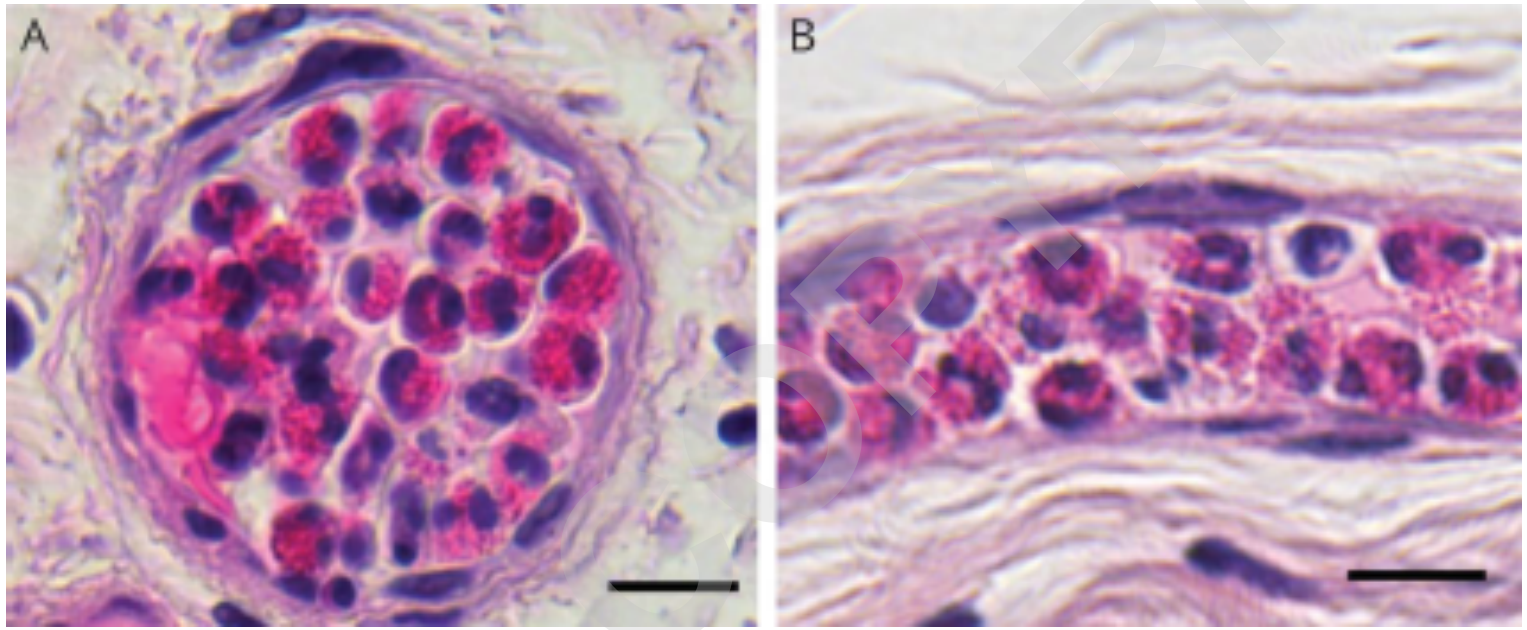
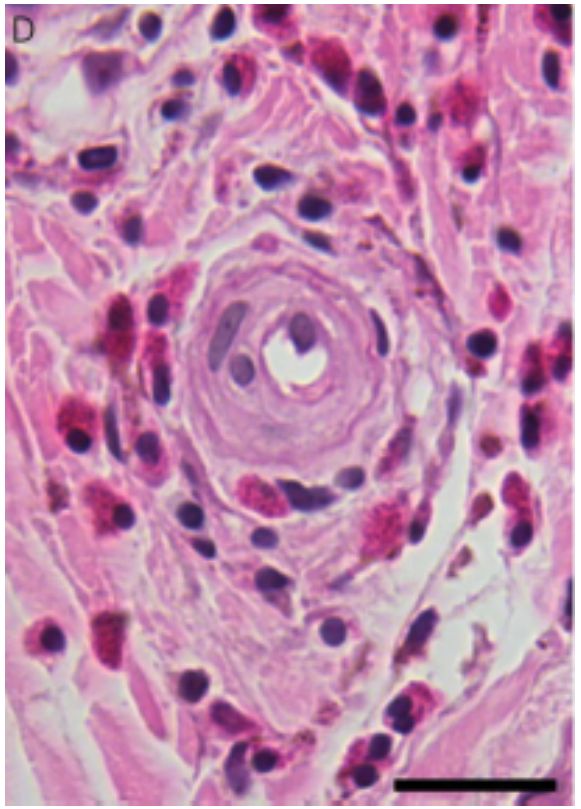
Granulomatose Eosinophilique avec Polyangéite

ARTICLE

Differential clinicopathologic features of EGPA-associated neuropathy with and without ANCA





ANCA MPO-NEG

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Granulomatose Eosinophilique avec Polyangéite

ARTICLE

Differential clinicopathologic features of EGPA-associated neuropathy with and without ANCA

MPO-ANCA–positive	MPO-ANCA–negative
• Higher frequency of findings suggestive of vasculitis-defined as the destruction of vascular structures with or without fibrinoid necrosis • Higher serum levels of C-reactive protein • Higher frequency of upper-limb symptoms as initial neuropathic manifestation	• Higher number of eosinophils in vascular lumen • Higher number of vessels occluded by eosinophils • Higher incidence of eosinophil infiltration in the endoneurium

« Given the differential mechanisms of tissue damage between ANCA-positive and ANCA-negative patients, consideration of the status of ANCA and eosinophil-related markers may both be needed for managing patients with EGPA and designing future clinical trials »

Nishi, Neurology 2020

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The Journal of Allergy and Clinical Immunology: In Practice
Available online 6 August 2020
In Press, Journal Pre-proof

Original Article

Antineutrophil cytoplasmic antibodies and organ-specific manifestations in eosinophilic granulomatosis with polyangiitis: A systematic review and meta-analysis

Hua-Ching Chang MD, MS [a, b], Pai-Chien Chou MD, PhD [c, d], Chun-Yu Lai MD [a], Hsiou-Hsin Tsai MD, PhD [a, e]

Chang, JACI In Practice 2020

Leukemia
https://doi.org/10.1038/s41375-019-0632-4

ARTICLE

Chronic myeloproliferative neoplasms

Prognostic impact of eosinophils in mastocytosis: analysis of 2350 patients collected in the ECNM Registry

Hanneke C. Kluin-Nelemans[1] · Andreas Reiter[2] · Anja Illerhaus[3] · Bjorn van Anrooij[1,4] · Karin Hartmann[3,5] · Lambertus F. R. Span[1] · Aleksandra Gorska[6] · Marek Niedoszytko[6] · Magdalena Lange[7] · Luigi Scaffidi[8] · Roberta Zanotti[8] · Patrizia Bonadonna[9] · Cecelia Perkins[10] · Chiara Elena[11] · Luca Malcovati[11] · Khalid Shoumariyeh[12,13] · Nikolas von Bubnoff[12,13,14] · Roberta Parente[15] · Massimo Triggiani[15] · Juliana Schwaab[2] · Mohamad Jawhar[2] · Francesca Caroppo[16] · Anna Belloni Fortina[16] · Knut Brockow[17] · Alexander Zink[17] · David Fuchs[18] · Alex Kilbertus[19] · Akif Selim Yavuz[20] · Michael Doubek[21] · Mattias Mattsson[22] · Hans Hagglund[22] · Jens Panse[23] · Vito Sabato[24] · Elisabeth Aberer[25] · Dietger Niederwieser[26,27] · Christine Breynaert[28] · Judit Várkonyi[29] · Vanessa Kennedy[10] · Olivier Lortholary[30] · Thilo Jakob[31,32,33] · Olivier Hermine[34] · Julien Rossignol[35] · Michel Arock[36] · Jason Gotlib[10] · Peter Valent[37,38] · Wolfgang R. Sperr[37,38]

Kluin-Nelemans, Leukemia 2020

Blood. 2020 Jul 13;blood.2020005161. doi: 10.1182/blood.2020005161. Online ahead of print.

Glucocorticoid-induced eosinopenia results from CXCR4-dependent bone marrow migration

So Gun Hong [1], Noriko Sato [2], Fanny Legrand [3], Manasi Gadkari [4], Michelle Makiya [4], Kindra Stokes [5], Katherine N Howe [4], Shiqin Yu [6], Nathaniel Seth Linde [7], Randall R Clevenger [7], Timothy Hunt [7], Zonghui Hu [8], Peter L Choyke [9], Cynthia E Dunbar [10], Amy D Klion [11], Luis Miguel Franco [4]

blood

Hong, Blood 2020

Mechanisms of allergic diseases

Eosinophil responses during COVID-19 infections and coronavirus vaccination

Check for updates

Andrew W. Lindsley, MD, PhD, Justin T. Schwartz, MD, PhD, and Marc E. Rothenberg, MD, PhD *Cincinnati, Ohio*

Lindsley, JACI 2020

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