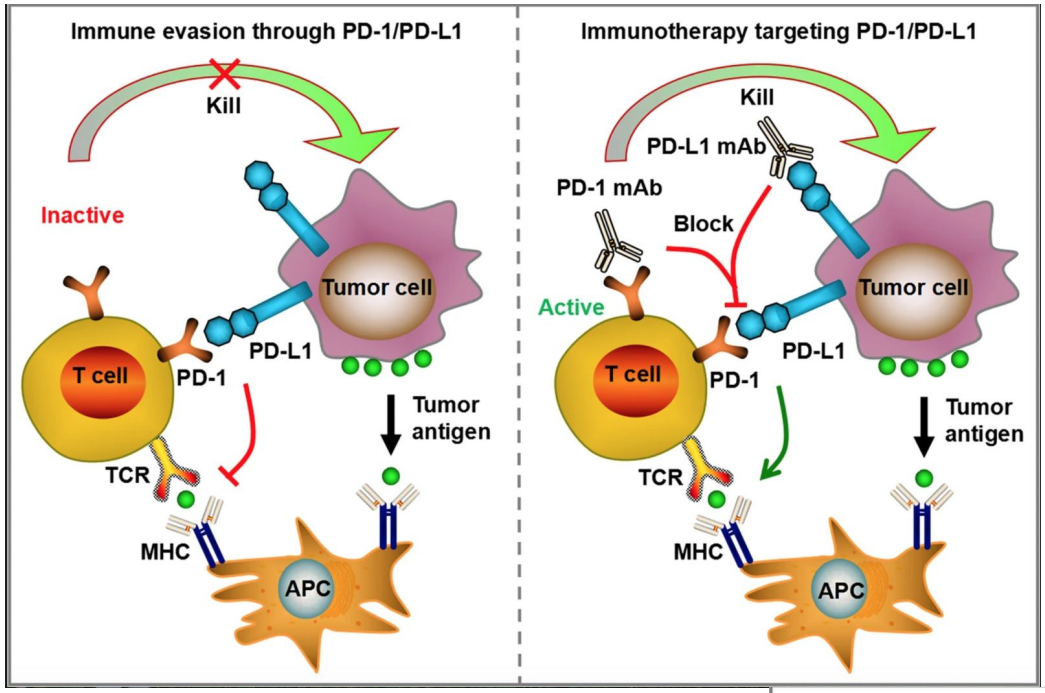
Vascularites sous inhibiteur de check-point immunitaire (ICI)

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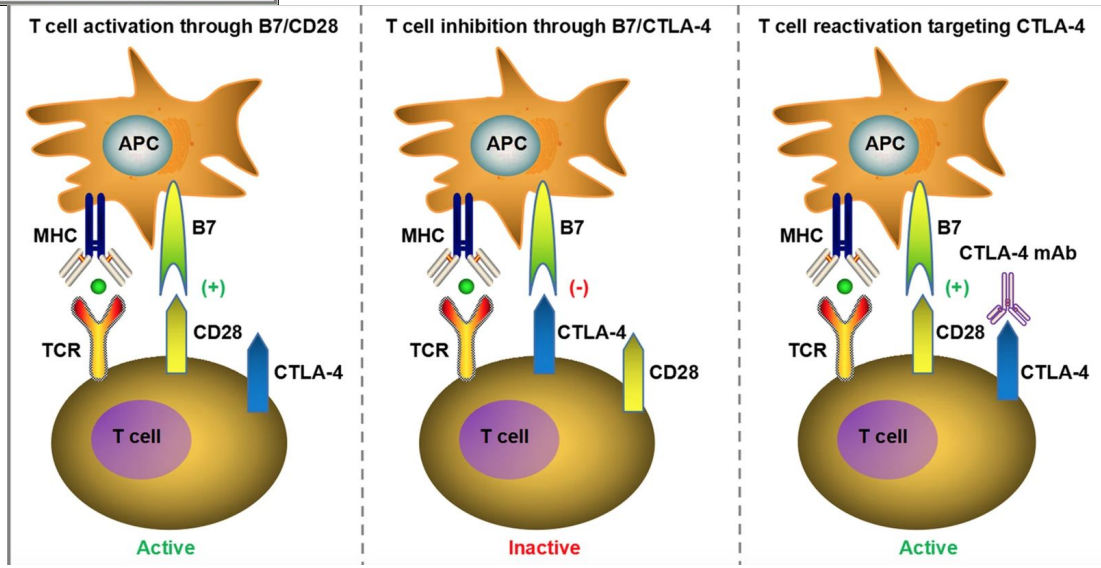


Les points de contrôle immunitaire



- Récepteurs à la surface des cellules immunitaires (LT)
- Empêchent réponse immunitaire excessive / protection tissus sains contre auto-immunité
- Cellules tumorales détournent ces voies pour échapper à la surveillance immunitaire

- Les **inhibiteurs de points de contrôle immunitaire** (immunothérapies anti-checkpoints ou ICI)
  - « lever les freins » inhibiteurs du système immunitaire
  - permettre aux LT de reconnaître et détruire les cellules tumorales



ENDOCRINOPATHIES

Thyroïdite avec hypo/hyper thyroïdie
Insuff surrénalienne,
Hypophysite, diabète de type 1

OCULAIRE

Uvéite, sclérite, sd sec

REINS

Néphrite

PEAU

Vitiligo, rash, psoriasis

ARTHRALGIES et MYALGIES

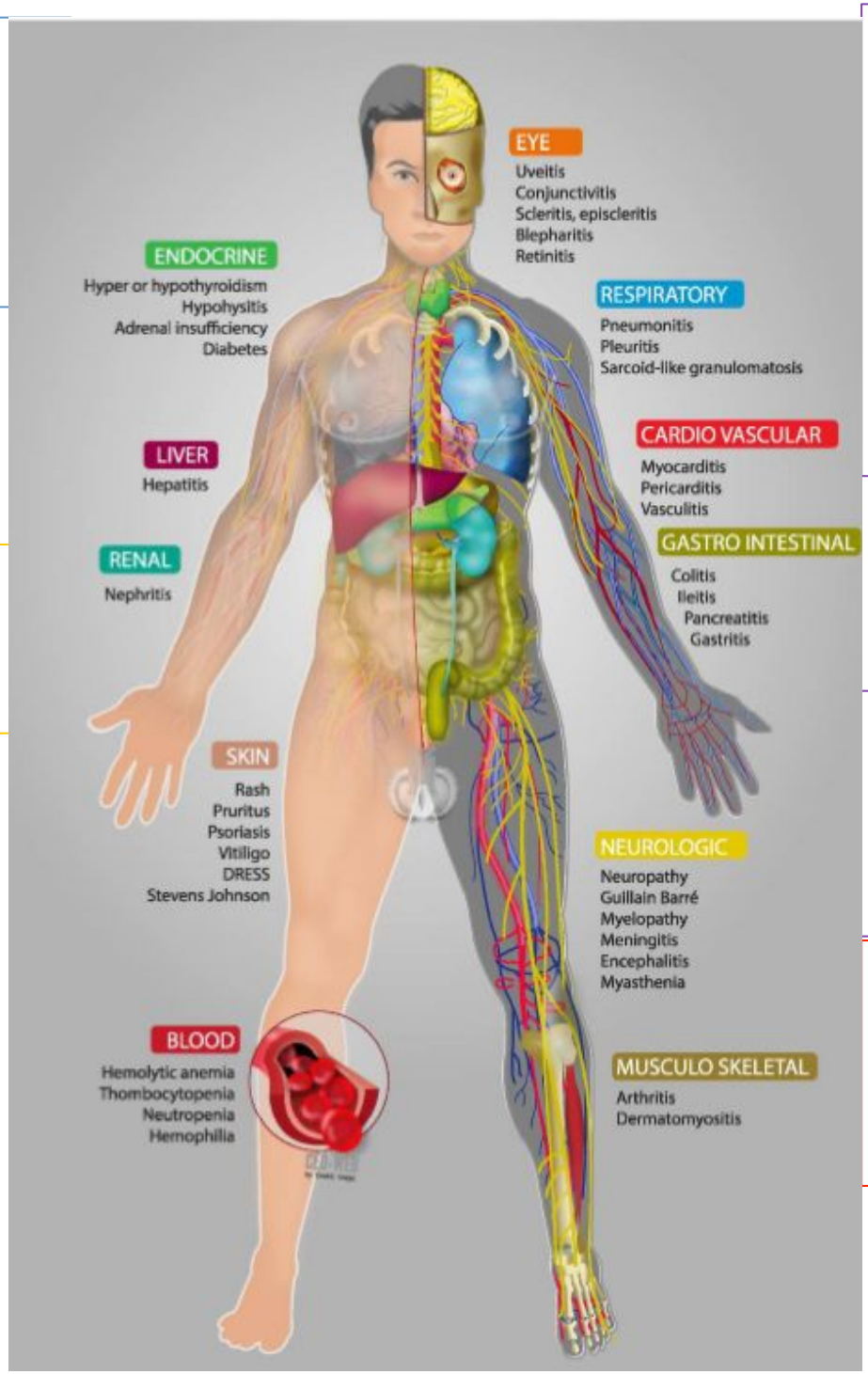
Polyarthrite, myosite…

TUBE DIGESTIF

Colite…

SANG

AHAI, PTI, neutropénie



SNC et SNP:

Neuropathies périphériques
Guillain Barré
Myasthénie
Méningite aseptique
Encephalite

POUMONS

Pneumonie interstitielle,
Sarcoidose

CARDIO-VASCULAIRE

Péricardite, myocardite

FOIE

Hépatites…

Immune related adverse events irAE
Vascularites immuno-induites

- Nombreuses recommandations publiées pour la prise en charge des irAE les plus fréquents, mais AUCUNES sur les vascularites

- Effet secondaire **très rare**
  - Revue littérature (29 cas)
  - Registre REISAMIC
    - Registre prospectif de IGR, prévalence 0,2% (7 cas identifiés entre 2014 et 2023)
  - Réseau ICIR: 28 cas
  - Appel à observation européen LVV: 27 cas

Seminars in Arthritis and Rheumatism 66 (2024) 152440

Contents lists available at ScienceDirect

Seminars in Arthritis and Rheumatism

journal homepage: www.elsevier.com/locate/semarthrit

A scoping review of vasculitis as an immune-related adverse event from checkpoint inhibitor therapy of cancer: Unraveling the complexities at the intersection of immunology and vascular pathology

Chan-Mi Lee [a,b], Margaret Wang [a], Aarthi Rajkumar [b], Cassandra Calabrese [c], Leonard Calabrese [c,*]

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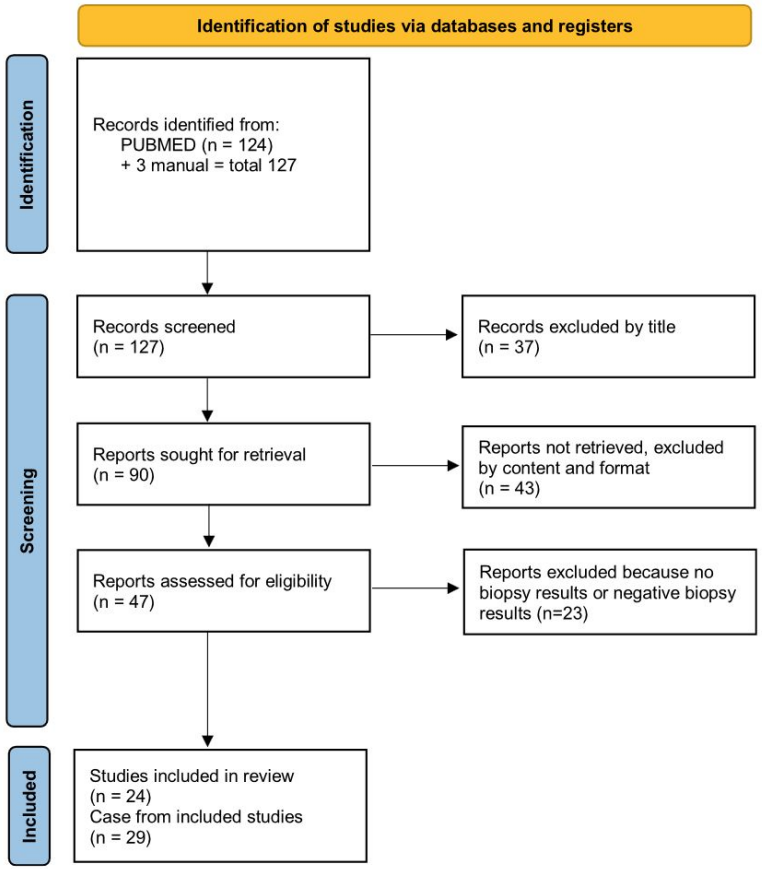


Fig. 1. PRISMA flow diagram of selection of published cases included in the Scoping Review.

- 29 cas, histologiquement prouvées (recueil en 2022) – 24 publi
  - âge moyen 62 ans (H59%, F41%)
  - K poumon (41%), mélanomes (41%), K rein (10%)
  - K stade 4 (75%)
- 21 patients ne remplissent pas critères ACR/EULAR
- 2 GCA, 2GPA, 2MPA, 2 EGPA
- 31% petits vaisseaux, 45% non classifiées, 17% single organ vasculitis
- Anti-PD1 97% cas, antiPD1 mono (65%), combo antiPD1-antiCTLA4 (31%)
- Biopsies cutanées/rénales/testiculaires/bronchique/cérébrales
- 69% atteinte systémique au diagnostic
- Survenue moyenne 7 mois après le début de l'ICI
- Ttt par corticoides 96% cas, amélioration clinique 86%

A scoping review of vasculitis as irAE from ICI. Chan-Mi Lee et al. 2024

Rheumatology, 2025, **64**, 3685–3690
https://doi.org/10.1093/rheumatology/keae711
Advance access publication 23 December 2024
Original Article

British Society for Rheumatology
RHEUMATOLOGY
OXFORD

Clinical science

Immune checkpoint inhibitors-associated vasculitis: a heterogeneous condition with possible severe disease course

Noemie Chanson[1,‡], Alexandre Galvagni[1,‡], Manuel Ramos-Casals[2,3,4], Juan Ignacio Ruiz[5], Karijn P.M. Suijkerbuijk[6], Karolina Gente[7], Philippe Kerschen[8], Jean Denis Karam[9], Rakiba Belkhir[10], Rodereau Outh[11], Fabienne Closs-Prophette[12], Jose Salvador Garcia Morillo[13], Ángel Robles-Marhuenda[14], Jean-Marie Michot[15], Anne Laure Voisin[15], Sabine Messayke[15], Arianne Laparra[16], Caroline Robert[17], Maria Suarez-Almazor[5], Xavier Mariette[10,18], Olivier Lambotte[1,18,*], for the ICIR[§]

- Réseau ICIR (Immuno Cancer International Registry)
  - 40 spécialistes (internistes, rhumato, onco)
  - 18 Pays Europe et USA
  - Etudie les irAE
  - Recueil rétrospectif /grosse base de donnée
  - A permis publications sur le syndrome sec/Sjogren, la sarcoïdose
- Inclusion:
  - critères EULAR/ACR de vascularite
  - Preuve histologique de vascularite (ou aortite typique en imagerie)
  - Exposition ICI
- Exclusion: infections ou autre étiologie possible, utilisation autres traitements concomitants (chimio, iTK)

Table 1. Characteristics of 28 cancer patients treated with immune checkpoint inhibitors with vasculitis

Patients (nb)	28
Country (%)	
USA/EU	54%/46%
Ethnicity (%)	
American Indian	0%
Asian	0%
Black or African American	0%
Hispanic	4 (14%)
Pasific Islander	0%
Caucasian	24 (86%)
Gender (M/F)	17/11
Previous AID	4 (14%)
Prior immunosupressive therapies	3 (11%)
Median age at diagnosis of cancer (y)	66 (25–93)
Type of cancer (*n*)	
Melanoma	10
Pulmonary	8
Other[a]	10
TNM	
Localised	8
Metastatic	20
Type of immunotherapy	
Anti PD-1	19
Anti PD-L1	4
Combination antiPD-1/CTLA4	5
Number of cycles of immunotherapy (median)	4 (1–54)
Median duration until diagnosis of vasculitis (month)	4 (0.5–49)
Median age at diagnosis of vasculitis	66 (30–93)

[a] Other cancers: one hepatocellular carcinoma, one liposarcoma, one cervical cancer, one mesothelioma, one squamous cell carcinoma, one Hodgkin lymphoma, two colorectal cancer, one urothelial carcinoma, one anaplastic thyroid carcinoma.

Table 2. Characteristics of ICI-induced vasculitis

Type of vasculitis	
Small vessels	21 (75%)
IgA vasculitis	5
Unclassified cutaneous vasculitis	6
GPA	1
EGPA	2
Cryoglobulinic vasculitis	3
Unclassified necrotizing vasculitis with renal, cutaneous and/or nervous involvement	4
Medium vessels	3 (11%)
PAN	2
Cerebral vasculitis	1
Large vessels	4 (14%)
GCA/Aortitis	4
Organ involvement	
CNS	3
PNS	4 2
Heart	1 (pericarditis)
ENT	2
Lung[a]	4
Articulation[b]	4
Skin (purpura)	18
Gut[c]	3
Kidney	7

[a] Dyspnea, cough, hemoptysis.
[b] Arthralgia, myalgia.
[c] Abdominal pain and gastrointestinal bleeding.
EGPA: eosinophilic granulomatosis with polyangiitis; GCA: giant cell arteritis; GPA: granulomatosis polyangiitis; PAN: periarteritis nodosa.

ICI-associated vasculitis, Chanson et al. 2025

- 24 biopsies + (3 cas d'aortites TEP +, 1 cryo + avec ulcères, purpura et symptômes au froid)
- 6 PBR
  - 2 néphropathies à IgA (1 proliferation endocapillaire, 1 avec glomerulosclérose)
  - 2 cas vascularite pauci-immune avec croissants (dont une ANCA+)
  - 2 cas de vascularites nécrosantes granulomateuses dont 1 dans le cadre PAN
- *Check-point inhibitor – related renal vasculitis and use of rituximab. Mamlouk 0., et al. J. Immunother Cancer 2020*

- Toxicité grade 2: 5 cas/28 (18%), **grade 3: 15 (54%), grade 4: 8 cas (28%)**
- Pas de décès en lien avec vascularite (mais 8 décès en lien avec K)

ICI-associated vasculitis, Chanson et al. 2025

- Stop ICI 82% cas (23/38)

- Traitement:
  - Corticoides 0,5 à 1mg/kg (90%), 5 bolus, 3 mois en moyenne
  - Traitement associé à la corticothérapie : 10 patients (36%)
    - Immunosuppresseurs : 1 MMF (GCA), 1 EDX (PAN avec atteinte rénale)
    - Rituximab (8 patients, 28%)) (vascularite du SNC, vascularite cryoglobulinémique, vascularite sévère inclassée avec atteinte rénale ou multinevrite), GPA, vascularite à IgA avec atteinte rénale)
    - 3 EP (atteintes rénales sévères, USA)
- Outcome :
  - RC 22 cas (79%)
  - 9 rechallenge (32%) avec 1 seule rechute : EGPA

- **Toxicité RARE mais potentiellement GRAVE (atteinte rénale, système nerveux)**
- **Vascularite des petits vaisseaux prédominantes**
- **Pattern ressemble aux vascularites à ANCA (IRA, atteinte glomérulaire avec des croissants, multinévrites), sans ANCA retrouvé**

ICI-associated vasculitis, Chanson et al. 2025

Focus sur les vascularites des gros vaisseaux LVV

Rheumatology, 2025, **64**, 4546–4554
https://doi.org/10.1093/rheumatology/keaf172
Advance access publication 26 March 2025
Original Article

British Society for Rheumatology RHEUMATOLOGY OXFORD

Clinical science

Immune checkpoint inhibitors–induced large vessel vasculitis: clinical characteristics and management from a European multicentre study

Adrien Cottu[1], Laure Delaval[2], Alexandra Forestier[3], Alessandro Tomelleri[4], Corrado Campochiaro[4], Milena Bond[5], Jérémie Dion[6], Aline Gury[7], Xavier Savary[8], Robin Dhote[9], Albrecht Betrains[10], Laurence Bouillet[11,12], Eric Liozon[13], Eva Bories[14], Arthur Petitdemange[1], Paul Legendre[15], Benjamin Crichi[16], Philippe Kerschen[17], Lucie Carneiro Esteves[18], Guillaume Armengol[19], Roderau Outh[20], Omar Al Tabaa[21], Louis Wolff[22], Maxime Ilzkovitz[22], Mohammad Yassine Cherif[23], Ariane Laparra[24], Lorenzo Dagna[4], Patrick Bruneval[25], Benjamin Terrier[1,26,*]

- Etude européenne (France, Italie, Belgique, Luxembourg) multicentrique rétrospective, entre 2018 et 2024
- Etude cas contrôle (4 LVV primaire pour 1 LVV survenant sous ICI)
- Inclusion: critères ACR/EULAR ACG, BAT+ ou aspect de LVV sur imagerie
- Relecture centralisée BAT quand possible
- Rémission après 1ere ligne, rechute, utilisation IS, mortalité

- 27 patients dont 5 avec atcd ACG ou PPR
- 74% K métastatique
- 30% combo ipi+nivo, 70% antiPD1 ou PDL1
- Apparition symptômes en médiane après 5 perfusion /3 mois après début ICI (max 5,5ans 1 cas)

Immune Check-point inhibitor Induced LVV, Cottu et al. 2025

Table 1. Characteristics of the 27 patients with ICI-induced LVV compared with 108 matched patients with primary LVV

	ICI-induced LVV ($n = 27$)	Primary LVV ($n = 108$)	P
Female, *n* (%)	16 (59.3)	64 (59.3)	1.000
Age at LVV diagnosis, years, median (IQR)	68 (61–74)	68 (60–78)	0.599
Clinical manifestations at LVV diagnosis			
New temporal headache	14 (51.2)	77 (71.3)	0.055
Scalp tenderness	10 (37.0)	45 (41.2)	0.663
Abnormal temporal artery	6 (22.2)	50 (46.3)	**0.024**
Jaw or tongue claudication	8 (29.6)	36 (33.3)	0.715
Blindness	4 (14.8)	7 (6.5)	0.158
PMR	13 (48.1)	47 (43.5)	0.666
Fever	5 (18.6)	47 (43.5)	**0.017**
CRP, mg/l, median (IQR)	80 (51–118)	84 (54–134)	0.641
Imaging tests			
Thickening of the vessel wall on CT scan/MRI	8 (29.6)	19 (17.6)	0.164
Contrast uptake of the vessel wall on CT scan/MRI	5/8 (62.5)	4/19 (21.1)	**0.041**
Aneurysm on CT scan/MRI	0 0	4 (3.7)	0.312
Vascular stenosis on CT scan/MRI	1 (3.7)	1 (0.9)	0.287
Vascular hypermetabolism on PET scan	18 (66.7)	42 (38.9)	**0.010**
Halo sign on echography	4 (14.8)	NA	NA
Arterial involvement			
Aorta	15 (55.6)	46 (42.6)	0.228
Thoracic aorta	11/15 (73.3)	NA	NA
Abdominal aorta	6/15 (40.0)	NA	NA
Supra-aortic arteries	5 (18.5)	27 (25.0)	0.480
Subclavian arteries	6 (22.2)	33 (30.6)	0.395
Iliofemoral arteries	2 (7.4)	18 (16.7)	0.228
Renal arteries	2 (7.4)	4 (3.7)	0.405
Isolated aortitis	7 (25.9)	15 (13.9)	0.131
Temporal artery biopsy	10 (37.0)	93 (86.1)	**<0.001**
Positive	3/10 (30.0)	59/93 (63.4)	**0.041**
Destruction of the internal elastic lamina	2/3 (66.7)	29/37 (78.4)	0.665
Mononuclear infiltrate	3/3 (100)	32/37 (86.5)	0.502
Giant cells	3/3 (100)	20/37 (54.1)	0.126
Granulomas	0/3 0	16/37 (43.2)	0.147
Phenotype presentation			
Cranial involvement only	8 (29.6)	58 (53.7)	**0.026**
Large vessel involvement only	11 (40.7)	14 (13.0)	**0.001**
Combination of cranial and large vessel involvement	8 (29.6)	36 (33.3)	0.715
ACR/EULAR GCA 2022 criteria score, median (IQR)	8 (4–13)	10 (8–12)	0.068
ACR/EULAR GCA 2022 criteria score ≥6	18 (66.7)	101 (93.5)	**<0.001**

Except where indicated otherwise, values are the number (%). ICI: immune checkpoint inhibitor; IQR: interquartile range; LVV: large vessel vasculitis; NA: not available. Bold P value < 0.05.

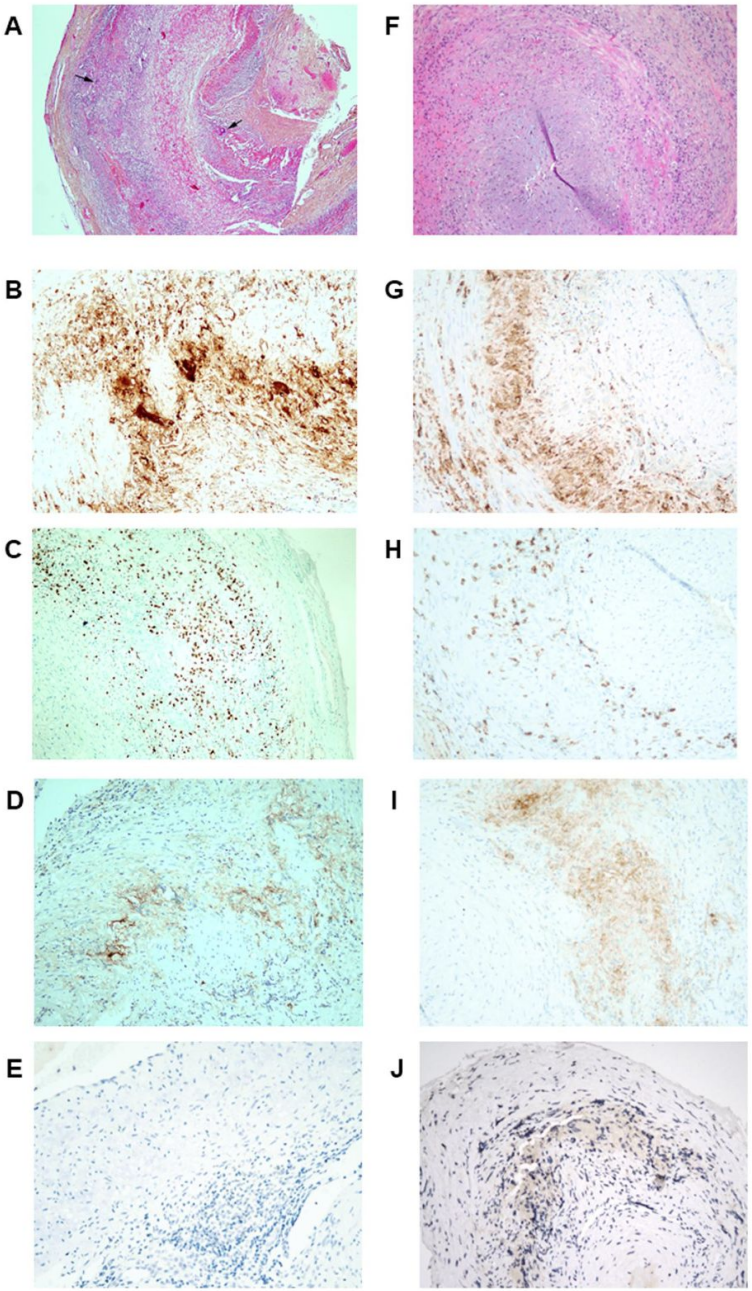
Immune Check-point inhibitor Induced LVV, Cottu et al. 2025

- Relecture centralisée de 6 BAT des patients sous ICI:
  - Pas de granulome
  - Infiltrat LT CD8 et infiltration à cellules géantes
  - PDL1+ sur 2 biopsies sur 6, PD1- sur 6/6

- Relecture 2 BAT de GCA primitive
  - Marquage PDL1+ fort, faible marquage PD1
  - Infiltrat à cellules mononuclées

Immune Check-point inhibitor Induced LVV, Cottu et al. 2025

ICI-LVV P-LVV



Coloration HES

Infiltration macrophagique CD68

Marquage CD8

Marquage PDL1

marquage PD1

Immune Check-point inhibitor Induced LVV, Cottu et al. 2025

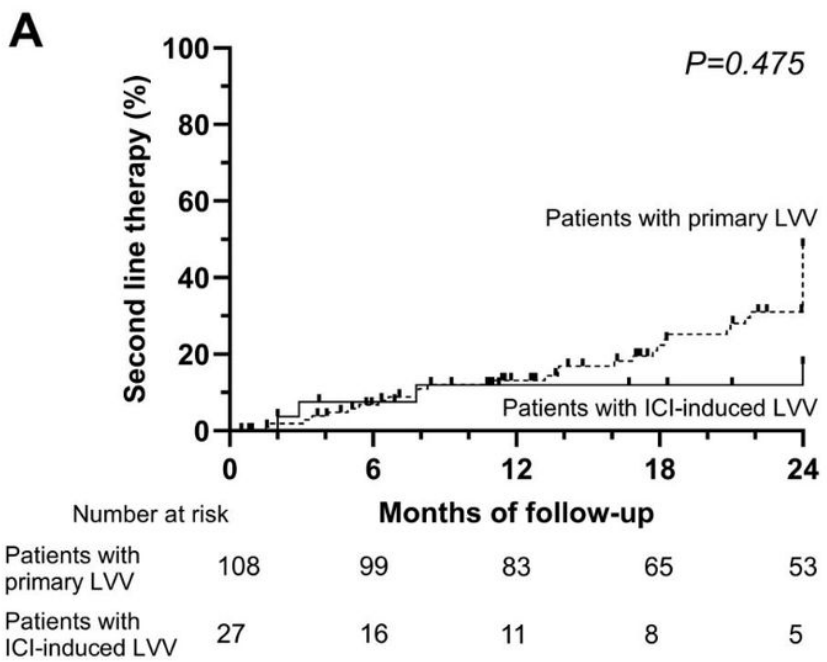
Table 2. Response to therapy in 27 patients with ICI-induced LVV compared with 108 matched patients with primary LVV

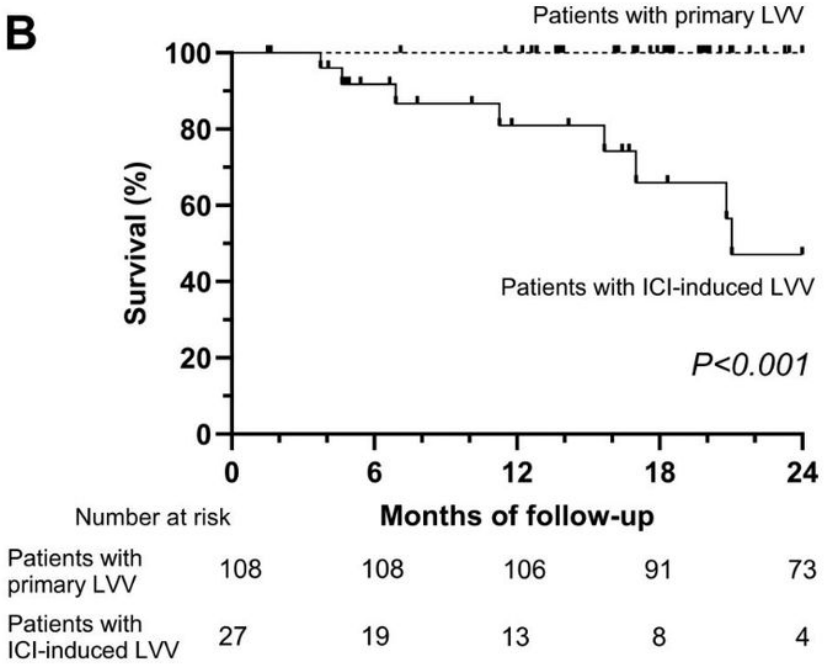
	ICI-induced LVV ($n = 27$)	Primary LVV ($n = 108$)	P
Response to 1st line therapy			
Initial remission	26 (96.3)	104 (96.3)	1.000
Sustained remission	20 (74.1)	60 (55.6)	0.081
Failure	1 (3.7)	4 (3.7)	1.000
Relapse	6 (22.2)	44 (40.7)	0.076
Response to 2nd line therapy			
Initial remission	6/6 (100)	37/38 (97.4)	0.691
Sustained remission	5/6 (83.3)	23 (60.5)	0.286
Failure	0/6 0	2/38 (5.2)	0.570
Relapse	1/6 (16.7)	14/38 (36.8)	0.338
Management of treatment			
GCs withdrawal	12 (44.4)	39 (36.1)	0.426
Immunosuppressant withdrawal	1/11 (9.1)	11/29 (37.9)	0.079
GCs and immunosuppressant withdrawal	7 (25.9)	43 (39.8)	0.183
At last follow-up			
Clinical remission	27 (100)	106 (98.1)	0.478
Sequelae blindness	1 (3.7)	3 (2.8)	0.800
CRP, mg/l, median (IQR)	3 (1–13)	3 (1–6)	0.877
CRP >10 mg/l	6/26 (23.1)	13/82 (15.9)	0.402
GC dose, mg/day, median (IQR)	5 (5–15)	5 (5–9)	0.664
Death	9 (33.3)	2 (1.9)	**<0.001**
Total follow-up since LVV diagnosis, months, median (IQR)	12 (5–20)	37 (20–55)	**<0.001**

Except where indicated otherwise, values are the number (%). GCs: glucocorticoids; ICI: immune checkpoint inhibitor; IQR: interquartile range; LVV: large vessel vasculitis. Bold P value < 0.05.

Immune Check-point inhibitor Induced LVV, Cottu et al. 2025

- Poursuite ICI 15%
- Arrêt définitif de ICI 52%, Interruption transitoire 19%
- 15% n'ont plus d'ICI avant le diagnostic
- Plus de rechute / échec 1er ligne dans le groupe poursuite ICI
- 2 remissions sans ttt sauf arrêt ICI





Immune Check-point inhibitor Induced LVV, Cottu et al. 2025

Physiopathologie ?

Immunoinhibitory checkpoint deficiency in medium and large vessel vasculitis

Hui Zhang[a,1], Ryu Watanabe[a,1], Gerald J. Berry[b], Augusto Vaglio[c], Yaping Joyce Liao[d], Kenneth J. Warrington[e], Jörg J. Goronzy[a], and Cornelia M. Weyand[a,2]

[a]Department of Medicine, Division of Immunology and Rheumatology, Stanford University School of Medicine, Stanford, CA 94305; [b]Department of Pathology, Stanford University School of Medicine, Stanford, CA 94305; [c]Division of Nephrology, University Hospital of Parma, 43100 Parma, Italy; [d]Department of Ophthalmology, Stanford Byers Eye Institute, Stanford University School of Medicine, Stanford, CA 94305; and [e]Division of Rheumatology, Mayo Clinic College of Medicine, Rochester, MN 55905

Edited by Tasuku Honjo, Graduate School of Medicine, Kyoto University, Kyoto, Japan, and approved December 19, 2016 (received for review October 10, 2016)

DOI: 10.1189/jlb.3MA0717-283

JLB JOURNAL OF LEUKOCYTE BIOLOGY

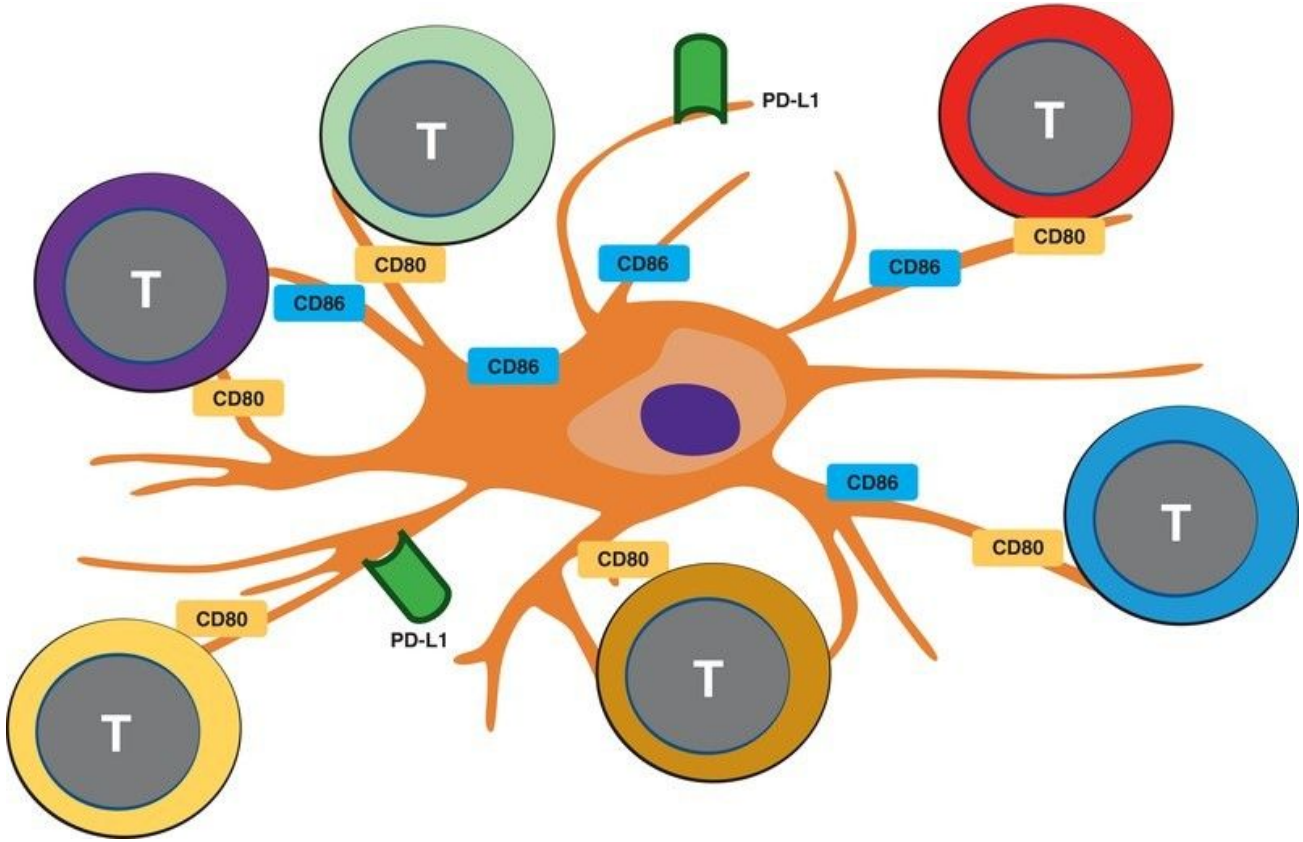
ARTICLE

The immunoinhibitory PD-1/PD-L1 pathway in inflammatory blood vessel disease

Cornelia M. Weyand[1] | Gerald J. Berry[2] | Jörg J. Goronzy[1]

- Cellules stromales et PDC des vaisseaux expriment PD-L1 et envoient un signal négatif aux LT PD1+
- Interruption de ce mécanisme inhibiteur ou diminution constitutive pourrait être associée à une activation des LT et au développement des vascularites

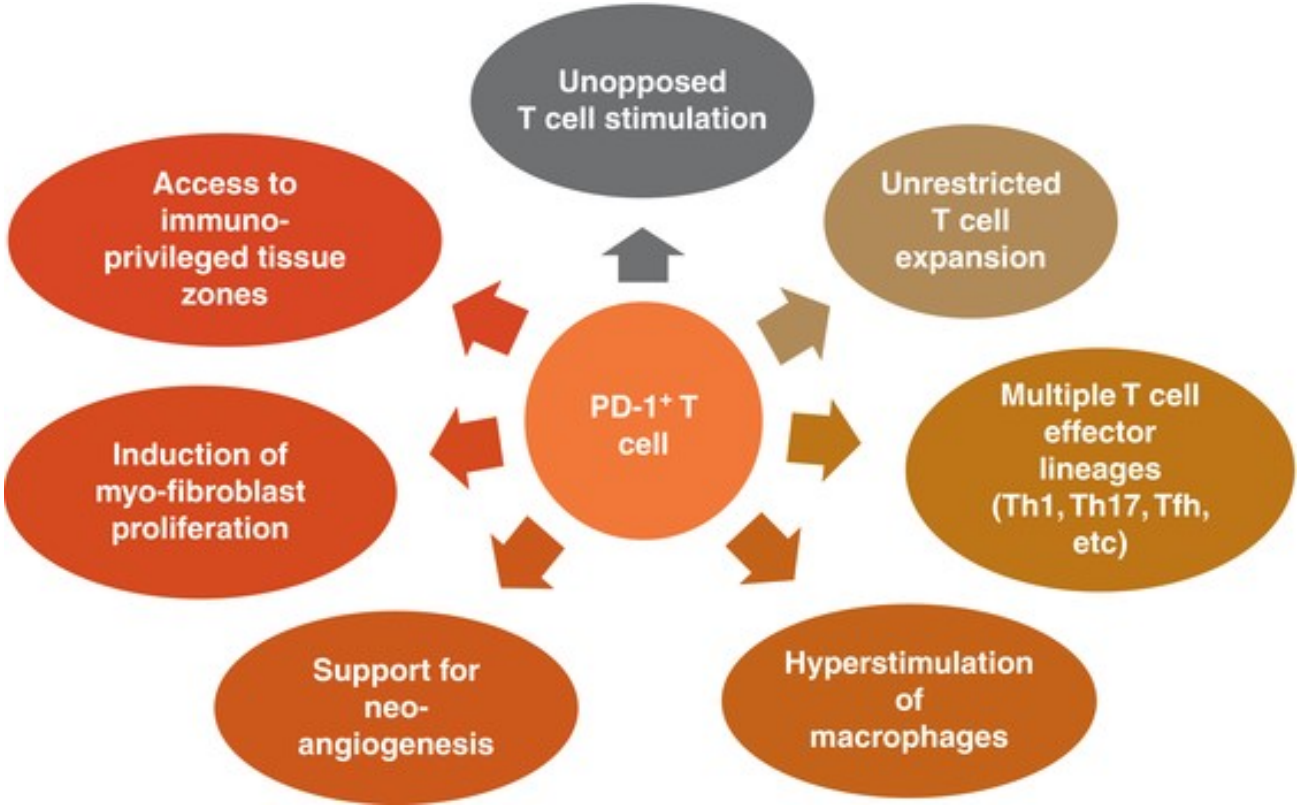
FIGURE 2 PD-L1^{lo} DCs stimulate a diverse array of T effector cells. In GCA, both circulating and ...



J Leukoc Biol., Volume 103, Issue 3, March 2018, Pages 565–575, https://doi.org/10.1189/jlb.3MA0717-283

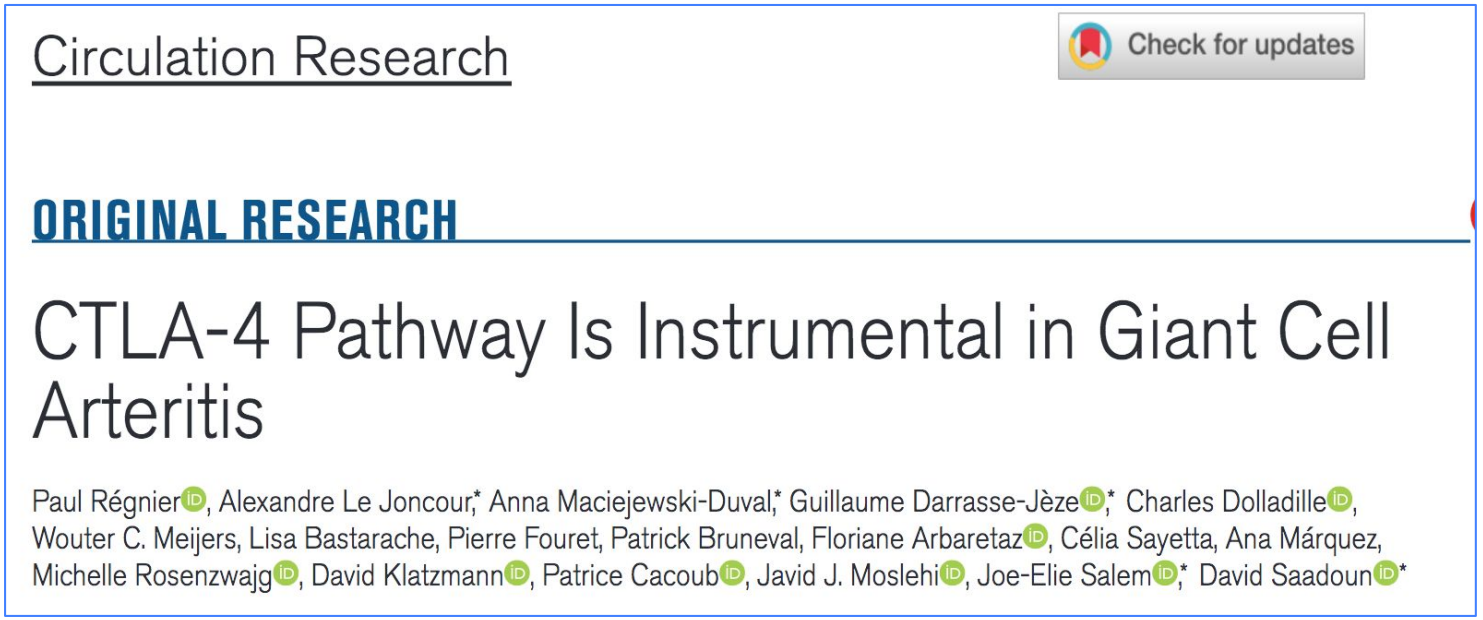
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FIGURE 3 Vasculitogenic T cells in GCA. Tissue-invading T cells in GCA are unopposed because of the failure of PD-L1 ...



J Leukoc Biol., Volume 103, Issue 3, March 2018, Pages 565–575, https://doi.org/10.1189/jlb.3MA0717-283

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Circulation Research

Check for updates

ORIGINAL RESEARCH

CTLA-4 Pathway Is Instrumental in Giant Cell Arteritis

Paul Régnier, Alexandre Le Joncour,* Anna Maciejewski-Duval,* Guillaume Darrasse-Jèze,* Charles Dolladille, Wouter C. Meijers, Lisa Bastarache, Pierre Fouret, Patrick Bruneval, Floriane Arbaretaz, Célia Sayetta, Ana Márquez, Michelle Rosenzwajg, David Klatzmann, Patrice Cacoub, Javid J. Moslehi, Joe-Elie Salem,* David Saadoun*

- hyperexpression CTLA-4 LT sanguin /aorte patients avec GCA
- > activité abatacept CTLA-4 Ig qui va inhiber CD80-CD86 et diminuer rechute GCA
    - Etude 2017, abatacept en add on CT, moins de rechute
    - *Langford CA, Cuthbertson D, Ytterberg SR et al.; for the Vasculitis Clinical Research Consortium. A randomized, double-blind trial of abatacept (CTLA-4Ig) for the treatment of giant cell arteritis. Arthritis Rheumatol Hoboken NJ 2017;69:837–45.*

Peut-on donner des ICI à un patient avec atcd de vascularite?

- 75% des patients avec atcd de MAI vont avoir une poussée de leur maladie (41%) ou un autre irAE (31%) ou les 2 (11%)
- Patients avec atcd de MAI exclus des essais cliniques

ACR Open Rheumatology
Vol. 8, No. 3, March 2026, e90003
DOI 10.1002/acr2.90003
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AMERICAN COLLEGE of RHEUMATOLOGY
Empowering Rheumatology Professionals

Safety of Immune Checkpoint Inhibitors in Cancer Patients With Preexisting Autoimmune Vasculitis

Juan Sevillano, MD,[1] Noha Abdel-Wahab, MD, PhD,[2,3,4,5] Yixuan Zhou, MD,[6] Juan I. Ruiz, MD, MPH,[7] and Maria E. Suarez-Almazor, MD, PhD[4,8]

- patients du MD Anderson Texas
  - Sur 23 332 patients ayant reçu ICI entre 2004 et 2024, 16 (0,07%) avaient une vascularite pré-existante
- Revue de littérature: 9 patients décrits
- 25 patients au total

Table 1. Baseline characteristics of patients at MD Anderson Cancer Center with preexisting vasculitis who received ICIs (n = 16)*

Case no.	Race	Vasculitis type	Disease duration, y	Positive antibodies	Previous vasculitis treatment	Cancer type	Stage	ICI therapy
1	White	GCA	4.4	ANA	Glucocorticoids	Mediastinum	IV	Atezolizumab
2	White	GCA	6.2	None	Glucocorticoids	Melanoma	III	Nivolumab + Ipilimumab
3	White	GCA	4.8	ANA	Glucocorticoids	Colon	IV	Pembrolizumab
4	White	GCA	0.9	None	Glucocorticoids	Melanoma	IV	Pembrolizumab
5	White	GCA	9.9	None	Glucocorticoids, methotrexate, tocilizumab	Lung	IV	Durvalumab
6	White	GCA	10.5	ANA	Glucocorticoids	Thyroid	IV	Pembrolizumab
7	White	GCA	0.5		Glucocorticoids	Thyroid	IV	Pembrolizumab
8	White	GCA	1.8		Glucocorticoids	Sarcoma	IV	Pembrolizumab
9	White	GPA	6.7	C-ANCA, PR3	Glucocorticoids, cyclophosphamide, rituximab, hydroxychloroquine	Melanoma	IV	Pembrolizumab
10	White	GPA	17.6	ANA ANCA	Glucocorticoids, cyclophosphamide	Bladder	IV	Pembrolizumab
11	White	GPA	24.7	ANCA PR3	Glucocorticoids, cyclophosphamide	Endometrium	II	Pembrolizumab
12	White	LCV	2.6	RF	Glucocorticoids, dapsone	Cervix	I	Pembrolizumab
13	Hispanic	LCV	19.2		None	Liver	III	Atezolizumab
14	Hispanic	OSV	3.6	None	Glucocorticoids, mycophenolate mofetil, methotrexate	CLL	NA	Nivolumab
15	White	OSV	25.4	None	Glucocorticoids	Lung	III	Atezolizumab
16	White	HSP	2.1		Glucocorticoids	Liver	III	Pembrolizumab

*ANA, antinuclear antibody; C-ANCA, cytoplasmic antineutrophil cytoplasmic antibody; CLL, itis; GPA, granulomatosis with polyangiitis; HSP, Henoch–Schönlein purpura; ICI, immune c NA, not applicable; OSV, other skin vasculitis; PR3, proteinase 3 antibody; RF, rheumatoid f

9 GCA
8 GPA
2 EGPA
2 vascularites leucoc.
2 autres vascularites cutanées
1 vascu IgA
1 Behcet

Table 2. Baseline characteristics of case reports in the literature with preexisting vasculitis who received ICIs (n = 9)*

Case no. (ref)	Race	Vasculitis type	Disease duration, y	Positive antibodies	Previous vasculitis treatment	Cancer type	Stage	ICI therapy
17 (Nabel et al[19])	NR	GPA	10	PR3	Glucocorticoids, methotrexate, cyclophosphamide	Bladder	IV	Pembrolizumab
18 (Aqeel et al[20])	White	GPA	NR	ANCA PR3	NR	NSCLC	NR	Pembrolizumab
19 (Hung et al[21])	NR	GPA[a]	3	ANCA PR3	None	Melanoma	III	Pembrolizumab
20 (Wulfken et al[46])	NR	GPA	23	ANCA PR3	Glucocorticoids, rituximab, cyclophosphamide, infliximab, azathioprine, methotrexate	Melanoma	IV	Nivolumab
21 (Yamada et al[47])	NR	GPA	4	ANCA MPO	Glucocorticoids, methotrexate	NSCLC	III	Pembrolizumab
22 (Ramos et al[22])	NR	EGPA	0.9	ANCA MPO	Glucocorticoids, rituximab, cyclophosphamide, plasmapheresis	Endometrial	NR	Pembrolizumab
23 (Maul et al[23])	NR	EGPA	6	NR	Glucocorticoids, azathioprine	Melanoma	IV	Ipilimumab
24 (Pedersen et al[24])	NR	Behçet disease	5	NR	Methotrexate, hydroxychloroquine	Melanoma	IV	Ipilimumab
25 (Shanklin et al[18])	White	GCA	14	NR	Glucocorticoids, methotrexate	Bladder	IV	Atezolizumab

*ANCA MPO, antineutrophil cytoplasmic antibody – myeloperoxidase subtype; ANCA PR3, antineutrophil cytoplasmic antibody – proteinase 3 subtype; EGPA, eosinophilic granulomatosis with polyangiitis; GCA, giant cell arteritis; GPA, granulomatosis with polyangiitis; ICI, immune checkpoint inhibitor; NR, not reported; NSCLC, non–small cell lung cancer.
[a]Sinus-limited case.

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Table 3. Clinical outcomes of patients with vasculitis who received ICIs (n = 26)*

Case no. (ref)	Disease activity at ICI initiation	Vasculitis treatment at ICI initiation	Vasculitis flare	Flare treatment	Flare outcome	Time to flare, wk	Other irAEs	Cancer outcome	Follow-up time, y
Giant cell arteritis (n = 9)									
1	Inactive	None	Yes	Glucocorticoids + sarilumab	Controlled	34	No	Remission	1.0
2	Inactive	Glucocorticoids	No	NA	NA	NA	Grade 4 hepatitis	Remission	3.1
3	Inactive	None	No	NA	NA	NA	Grade 3 AHA	Progression	0.1
4	Inactive	Glucocorticoids	No	NA	NA	NA	No	Progression	1.8
5	Inactive	Tocilizumab	No	NA	NA	NA	No	Remission	0.5
6	Inactive	None	No	NA	NA	NA	No	Progression	1.1
7	Inactive	None	No	NA	NA	NA	No	Stable	3.8
8	Inactive	None	No	NA	NA	NA	No	Progression	2.3
25 (Shanklin et al[18])	Inactive	None	Yes	Glucocorticoids + cyclophosphamide	Poor	30.4	No	NA	0.1
Total, n (%)		3 (33)	2 (22)				2 (22)		
Median						32			1.1
Granulomatosis with polyangiitis (n = 8)									
9	Inactive	None	Yes	Glucocorticoids + rituximab	Controlled	1.3	No	Progression	0.6
10	Inactive	None	No	NA	NA	NA	No	Progression	1.7
11	Inactive	Rituximab	No	NA	NA	NA	No	Partial response	1.2
17 (Nabel et al[19])	Inactive	None	Yes	Glucocorticoids + rituximab	Controlled	1	NA	Stable disease	0.2
18 (Aqeel et al[20])	NA	NA	Yes	Glucocorticoids + rituximab	Controlled	4	NA	Progression	0.4
19 (Hung et al[21])	Active	None	Yes	Glucocorticoids + rituximab, infliximab	Controlled	3	NA	NA	NA
20 (Wulfken et al[46])	Inactive	Rituximab	No	No	NA	NA	No	Partial response	1.2
21 (Yamada et al[47])	Inactive	Glucocorticoids + methotrexate	No	No	NA	NA	No	Partial response	0.5
Total, n (%)		3/7 (43)	4 (50)				0		
Median						2.2			0.6
Cutaneous vasculitis (n = 4)									
12	Active	None	Yes	Supportive	Controlled	27	No	Progression	1.1
13	Inactive	None	No	NA	NA	NA	No	Progression	1.9
14	Inactive	Glucocorticoids	Yes	Glucocorticoids	Controlled	10.1	No	Remission	1.3
15	Inactive	None	No	NA	NA	NA	No	Remission	2.0
Total, n (%)		1 (25)	2 (50)				0		
Median						18.5			1.6
Other vasculitis (n = 4)									
Eosinophilic granulomatosis with polyangiitis									
22 (Ramos et al[22])	Inactive	Rituximab	No	No	NA	NA	No	Progression	0.3
23 (Maul et al[23])	Inactive	Glucocorticoids	No	No	NA	NA	Grade 3 colitis	Partial response	2.1
Behçet disease									
24 (Pedersen et al[24])	Active	None	No	No	NA	NA	No	Progression	0.3
Henoch–Schönlein purpura									
16	Inactive	None	No	NA	NA	NA	No	Progression	2.1
Total, n (%)		2 (40)	0				1 (25)		
Median									1.2

*AHA, autoimmune hemolytic anemia; ICI, immune checkpoint inhibitor; irAEs, immune relates adverse events; NA, not applicable.

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- 25 patients
- 8 poussée de vascularite (4 GPA, 2 GCA, 3 vascularites cutanées)
- 3 autre irAE
- Sur les 3 patients avec vascularite active à l'initiation, 2 poussées
- Sur 16 patients sans immunosuppresseurs a l'initiation ICI, 7 (55%) vont faire une poussée et 1 de novo irAE
- Sur 9 patients sous IS avant initiation ICI, 1 seule poussée de vascularite, 2 autres irAE
- Sur les 11 patients avec poussée de vascularite ou autre irAE, 10 ont nécessité majoration ou ajout immunosuppresseur
- 1 décès du à la vascularite (AVC GCA)
- 4 rechallenge, pas de nouvelle poussée
- Devenir K:
  - Patients sous IS: 56% rémission ou stable, 44% progression
  - Patients sans IS: 36% réponse ou stable, 64% progression

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- En conclusion:
  - 1/3 patients environs vont avoir une poussée de leur vascularite
  - Moins de risque quand sous ttt IS avant initiation de l'ICI
  - Bon pronostic global de la vascularite
  - 52% cas, progression du cancer
    - Rôle néfaste IS, diminuer efficacité ICI ?

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Quel traitement ?

- Pas de recommandations. Discussion collégiale RCP itox

- Arrêt ICI? Définitif? Temporaire?
- Poursuite ICI en cas de symptômes mineurs?

- Comme autres irAE, décroissance rapide corticoïdes, durée courte
- Corticoides > 10mg/j, diminution survie
  - *Arbour KC, Mezquita L, Long N, et al. Impact of baseline steroids on efficacy of programmed cell death-1 and programmed death-ligand 1 blockade in patients with non-small-cell lung cancer. J Clin Oncol 2018;36:2872–8.*
- Eviter immunosuppresseurs dans le contexte de cancer?

- AntiCD20:
  - bien tolérés, très efficace particulièrement si vANCA
  - MAIS rôle du LB dans la réponse aux ICI (*Helmink BA, Reddy SM, Gao J et al. B cells and tertiary lymphoid structures promote immunotherapy response. Nature 2020; 577:549–55.*
- Tocilizumab, bien toléré, pas d'impact négatif sur le cancer

En Conclusion

- Vascularite induites par ICI = effet secondaire rare mais potentiellement grave (atteinte rénale / neuro / ophtalmo dans LVV)
  - Eduquer oncologues: purpura, surveillance protéinurie/Hu
- Ne remplissent pas critères ACR/EULAR de vascularites
- Pattern ANCA like, sans ANCA
- Vascularites paranéoplasiques (IgA) et non irAE?
- Balance bénéfice risque entre traitement de la vascularite / ne pas perdre l'efficacité anti-tumorale.