Banff 2019 ve Sonrası: Transplantasyon Biyopsisinde Tanısal Kriterlerde Güncellemeler

Dr. Yasemin Özlük
İ.Ü. İstanbul Tıp Fakültesi
Tıbbi Patoloji Anabilim Dalı

Sunum içeriği

- Mikrovasküler inflamasyonun (MVI) yeniden değerlendirilmesi ve spesifitesi
  - DSA-neg ve C4d-neg MVI
  - Olası AMR
  - AMR, “olası AMR” ve “DSA-neg ve C4d-neg MVI” raporlaması ve klinik önemi
- Akut tubuler hasar, arteryal intimal fibrozis
- caTCMR
- Aktivite ve kronisite indeksleri

Banff 2019 **Banff 2022**

- Banff 2024 raporu özeti

Kidney International, Vol. 44 (1993), pp. 411–422

International standardization of criteria for the histologic diagnosis of renal allograft rejection: The Banff working classification of kidney transplant pathology

Kidney International, Vol. 55 (1999), pp. 713–723

The Banff 97 working classification of renal allograft pathology

American Journal of Transplantation 2014; 14: 272–283
Wiley Periodicals Inc.

doi: 10.1111/ajt.12590

Meeting Report

Banff 2013 Meeting Report: Inclusion of C4d-Negative Antibody-Mediated Rejection and Antibody-Associated Arterial Lesions

Received: 5 November 2017 | Revised: 6 December 2017 | Accepted: 7 December 2017
DOI: 10.1111/ajt.14625

MEETING REPORT

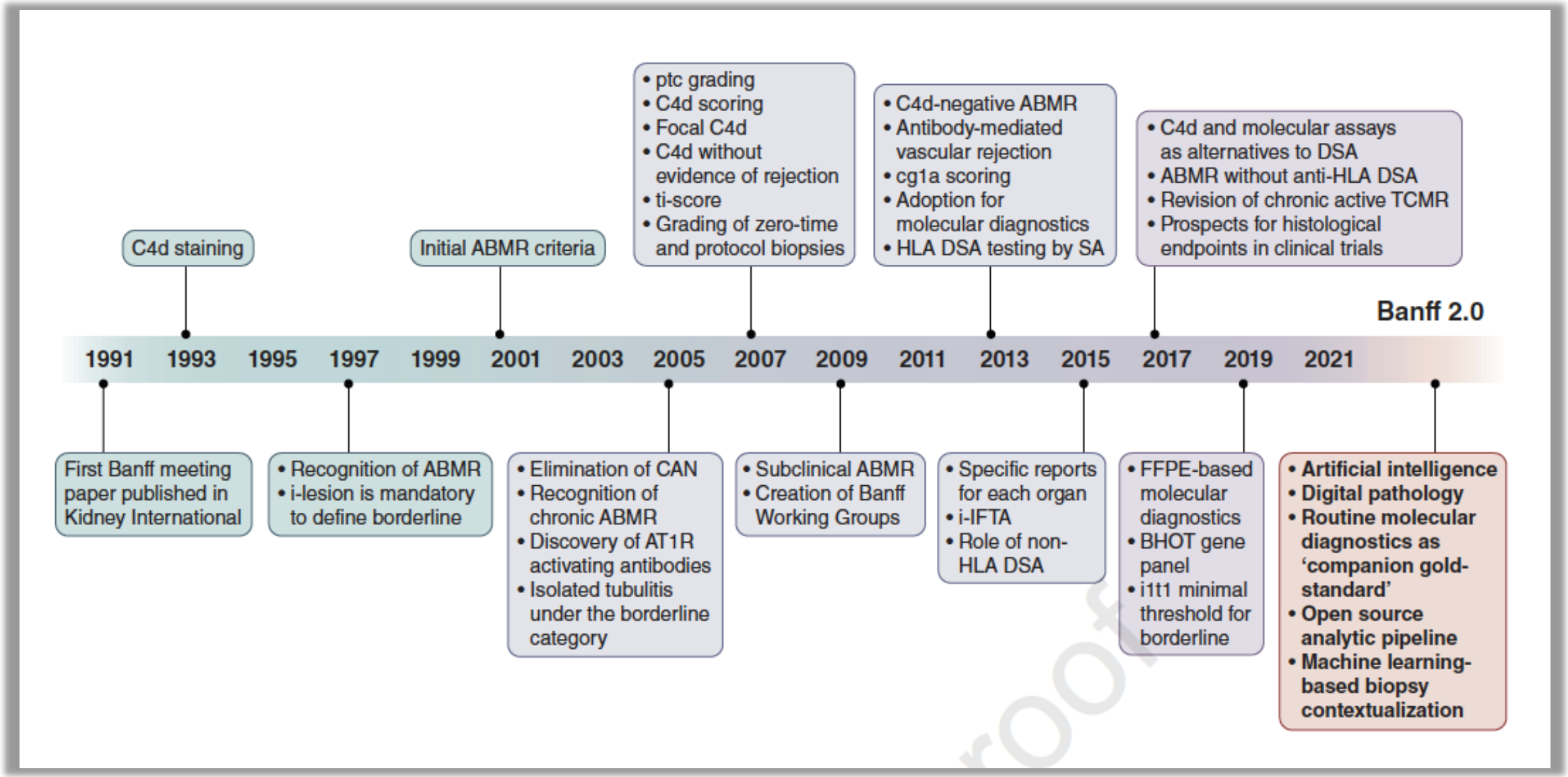
AJT

The Banff 2017 Kidney Meeting Report: Revised diagnostic criteria for chronic active T cell–mediated rejection, antibody-mediated rejection, and prospects for integrative endpoints for next-generation clinical trials

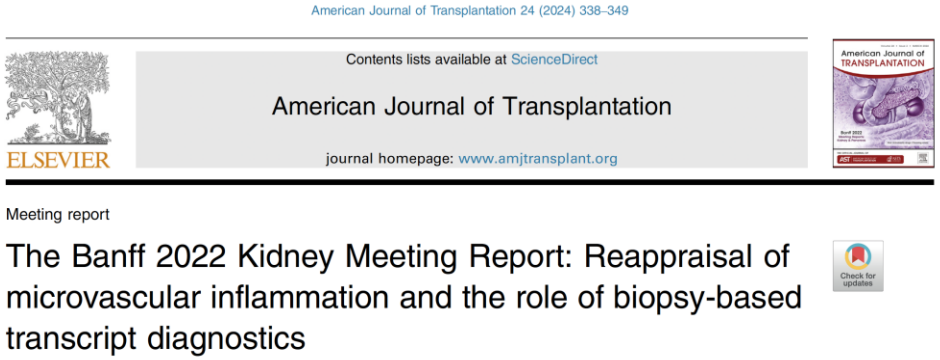
MEETING REPORT

The Banff 2019 Kidney Meeting Report (I): Updates on and clarification of criteria for T cell– and antibody-mediated rejection

BANFF SINIFLAMASI



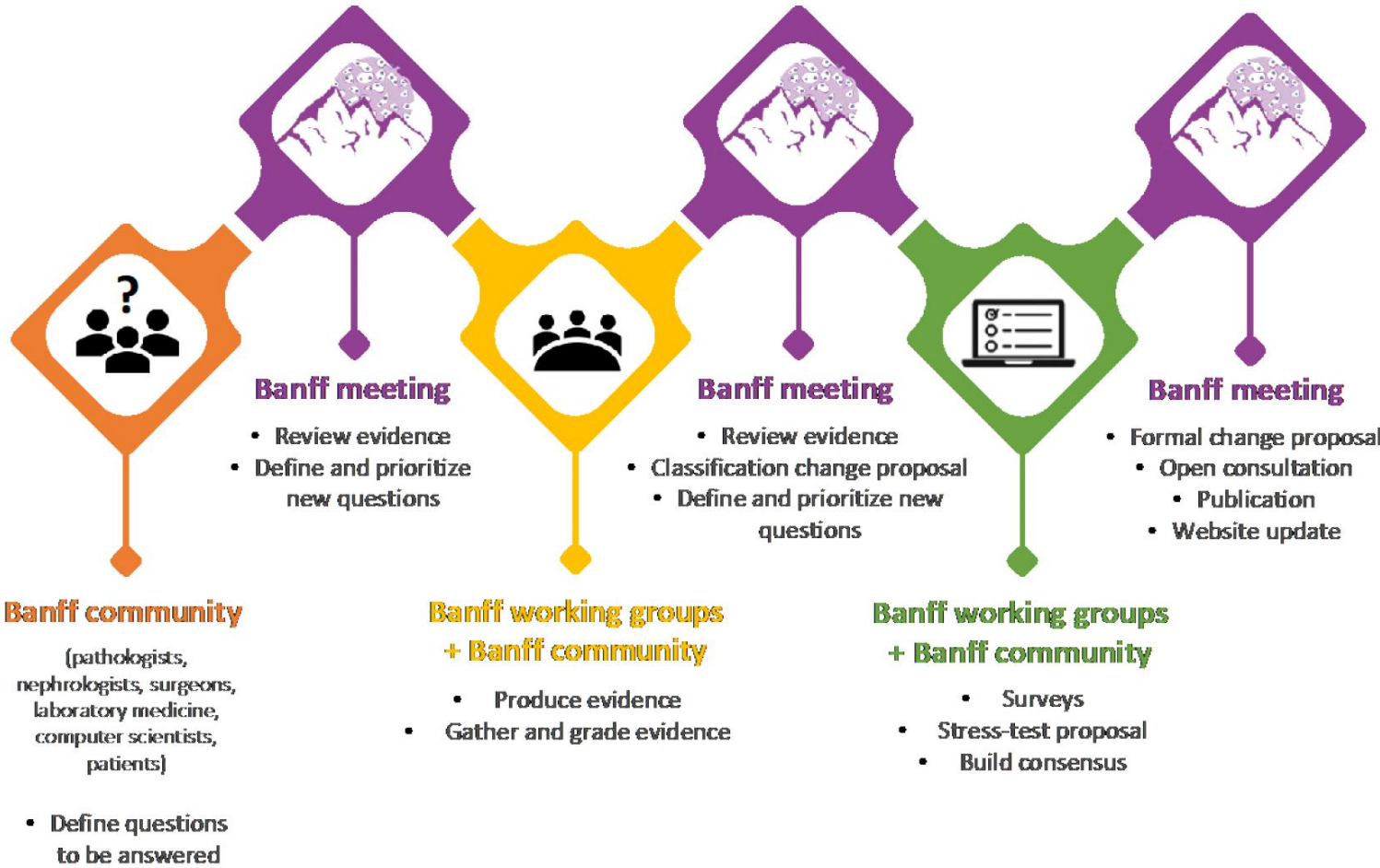
Loupy A, Mengel M, Haas M, 30 years of the International Banff Classification for Allograft Pathology: The Past, Present and Future of Kidney Transplant Diagnostics Kidney International (2022), doi: https://doi.org/10.1016/j.kint.2021.11.028.



Naesens M et al. Am J Transplant. 2024;24(3):338-349. doi: 10.1016/j.ajt.2023.10.016.

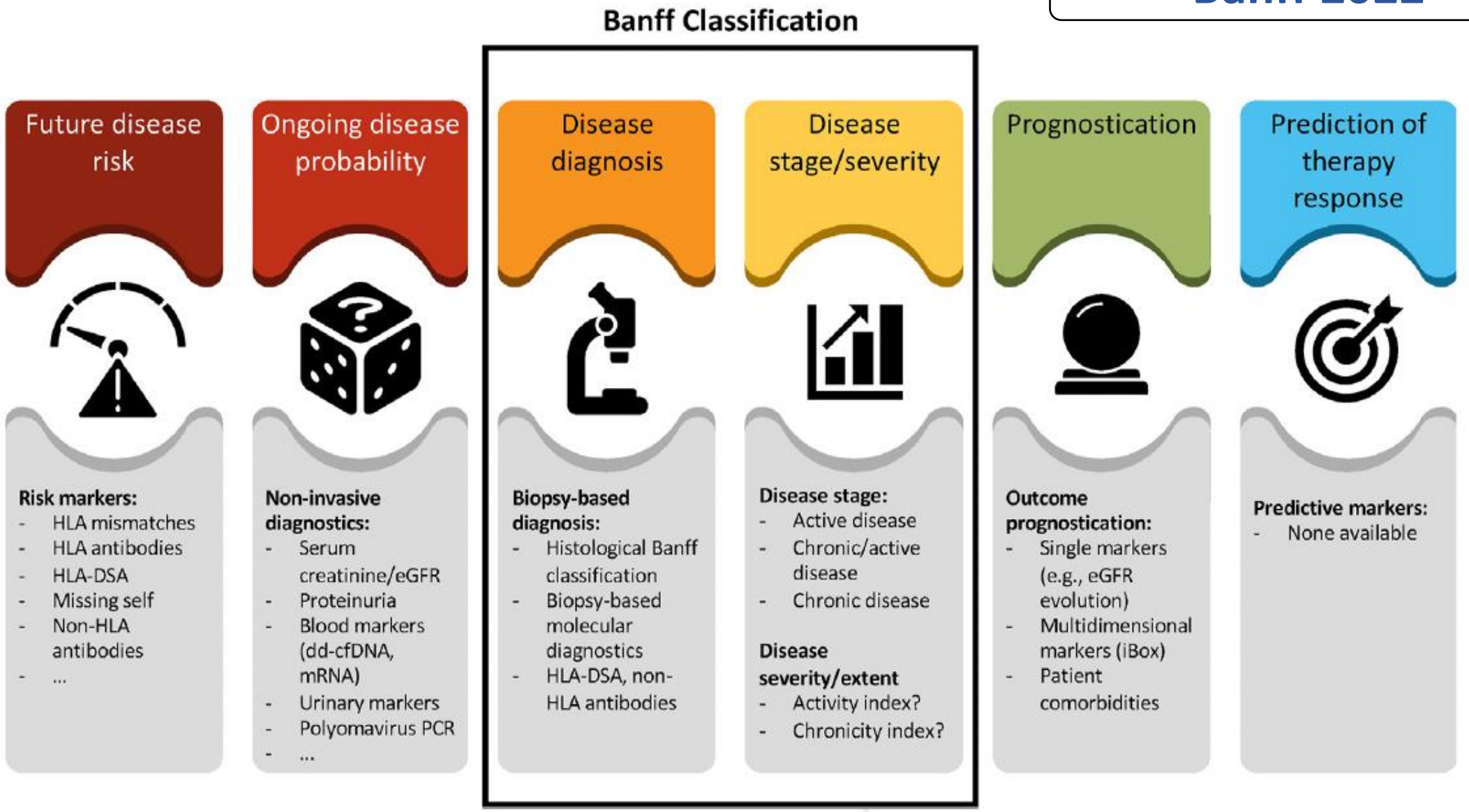
Roufosse C et al. Am J Transplant. 2024;24(3):350-361. doi: 10.1016/j.ajt.2023.10.031.

https://banfffoundation.org/central-repository-for-banff-classification-resources-3/



Naesens M et al. Am J Transplant. 2024;24(3):338-349.
doi: 10.1016/j.ajt.2023.10.016.

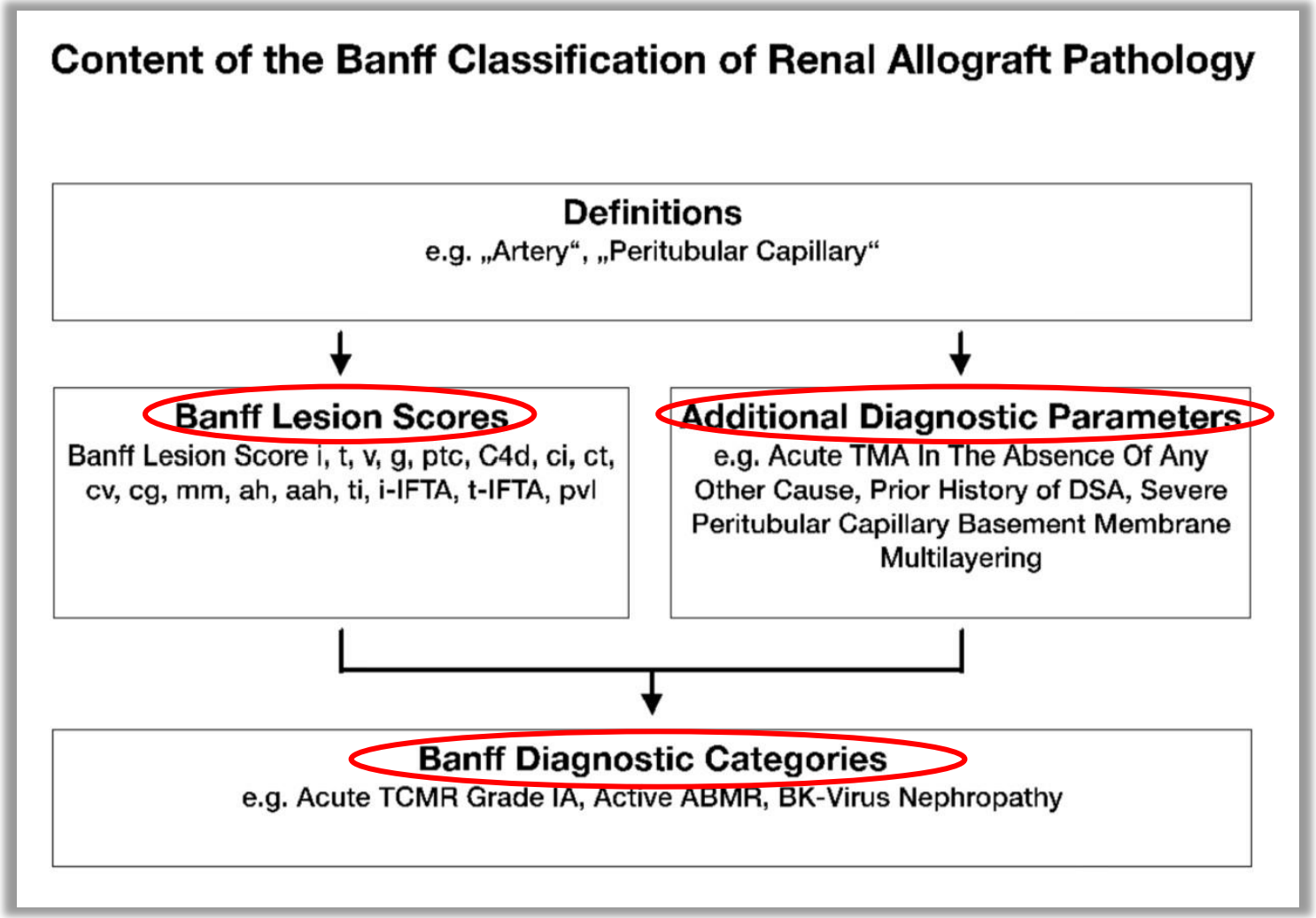
Banff 2022



Naesens M et al. Am J Transplant. 2024;24(3):338-349.
doi: 10.1016/j.ajt.2023.10.016.

Banff lesion score,	Abbreviation	0	1	2	3
Interstitial inflammation	*i*	<10%	10-25%	26-50%	>50%
Tubulitis	*t*	None	1-4/tubular cross section or 10 tubular epithelial cells	5-10	>10 OR (foci of tubular basement membrane destruction with i ≥ 2 and t2 elsewhere)
Intimal arteritis	*v*	None	<25% luminal area lost	≥25% luminal area lost	Transmural and/or fibrinoid change and medial smooth muscle necrosis
Glomerulitis	*g*	None	<25%	25-75%	>75%
Peritubular capillaritis	*ptc*	<3 leukocytes/PTC	≥1 leukocyte in ≥10% of PTCs with max. of 3-4/PTC	≥1 leukocyte in ≥10% of PTCs with max. of 5-10/PTC	≥1 leukocyte in ≥10% of PTCs with max. of >10/PTC
C4d	*C4d*	None	<10%	10-50%	>50%
Interstitial fibrosis	*ci*	≤5%	6-25%	26-50%	>50%
Tubular atrophy	*ct*	None	≤25%	26-50%	>50%
Vascular fibrous Intimal thickening	*cv*	None	≤25%	26-50%	>50% reduction in luminal area
GBM double contours	*cg*	None	1a: only by EM 1b: ≤25% by LM	26-50%	>50% of the most affected nonischemic, nonsclerotic glomerulus
Mesangial matrix expansion	*mm*	None	≤25%	26-50%	>50% of nonsclerotic glomeruli
Arteriolar hyalinosis	*ah*	None	Mild to moderate in ≥1	Moderate to severe in >1	Severe in many
Hyaline arteriolar thickening	*aah*	None	1 without circumferential	≥1 without circumferential	circumferential
Total inflammation	*ti*	<10%	10-25%	26-50%	>50%
Inflammation in the area of IFTA	*i-IFTA*	<10%	10-25%	26-50%	>50%
Tubulitis in areas of interstitial fibrosis	*t-IFTA*	None	1-4/tubular cross section or 10 tubular epithelial cells	5-10	>10
Polyomavirus Load	*pvl*	≤1%	>1%	≤10%	>10%

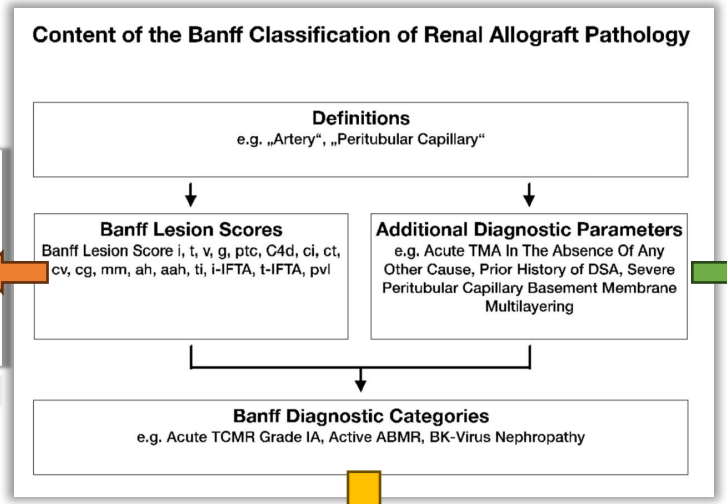
https://banfffoundation.org/central-repository-for-banff-classification-resources-3/



https://banfffoundation.org/central-repository-for-banff-classification-resources-3/

Acute Banff scores	Grading (0, 1, 2, 3)	Chronic Banff scores	Grading (0, 1, 2, 3)	Acute & chronic Banff scores	Grading (0, 1, 2, 3)
i		ci		ti	
t		ct		i-IFTA	
v		cv		t-IFTA	
g		cg		pvl	
ptc		ptcml			
C4d					

Am J Transplant. 2020;20:2318–2331



Tanı kategorileri

- Normal/nonspesifik değişiklikler
- Antikor aracılı rejeksiyon (aktif/kronik)
- Borderline değişiklikler
- T hc aracılı rejeksiyon (aktif/kronik)
- IFTA-NOS
- Diğer

Parameters
Acute Thrombotic Microangiopathy In The Absence Of Any Other Cause
Absence Of Recurrent Or De Novo Glomerulonephritis
Infection
Biopsy-based transcript diagnostics for AMR/MVI above a defined threshold, if thoroughly validated for use as a substitute for AMR/MVI and available
Severe Peritubular Capillary Basement Membrane Multilayering
Arterial Intimal Fibrosis With Mononuclear Cell Inflammation In Fibrosis And Formation Of Neointima
Prior Evidence Of DSA
Serologic Evidence Of DSAs (DSA To HLA Or Other Antigens)
Prior Documented Diagnosis Of Active Or Chronic Active AMR
Prior History Of TCMR
Evidence Of Chronic TMA
C4d Staining On Fresh-Frozen Or Paraffin-Embedded Tissue
Polyomavirus Nephropathy, Posttransplant Lymphoproliferative Disorder, Calcineurin Inhibitor Toxicity, Acute Tubular Injury Recurrent Disease, De Novo Glomerulopathy (Other Than TG), Pyelonephritis, Drug-Induced Interstitial Nephritis
Other Known Causes Of i-IFTA Ruled Out

Transplantation 2018;102: 1795–1814

https://banfffoundation.org/central-repository-for-banff-classification-resources-3/

BANFF 2022

Tanı kategorileri	Değişiklik durumu
Normal/nonspesifik değişiklikler	YOK
Antikor aracılı rejeksiyon (aktif/kronik)	Revizyon VAR
Borderline değişiklikler	Sıklıkla değişiklik YOK
T hc aracılı rejeksiyon (aktif/kronik)	Aktif TCMR için YOK, Kronik için revizyon VAR
IFTA-NOS	YOK
Diğer	YOK

AMR

Borderline/caTCMR

Aktivite ve kronisite indeksleri

AMR

Borderline/caTCMR

Aktivite ve kronisite indeksleri

AMR

- Heterojen kategori
- Kurallar komplike
- Tanımlamalarda belirsizlik ve uygulama sıkıntıları

Antikor aracılı rejeksiyon

Banff 2022

AMR İLİŞKİLİ MORFOLOJİK LEZYONLAR
(aktif / kronik)(g, ptc, v, TMA, cg)

Kriter 1

+

EN AZ BİRİNİN VARLIĞI
(pozitif C4d / g + ptc ≥ 2 / moleküler belirteçler)

Kriter 2

+

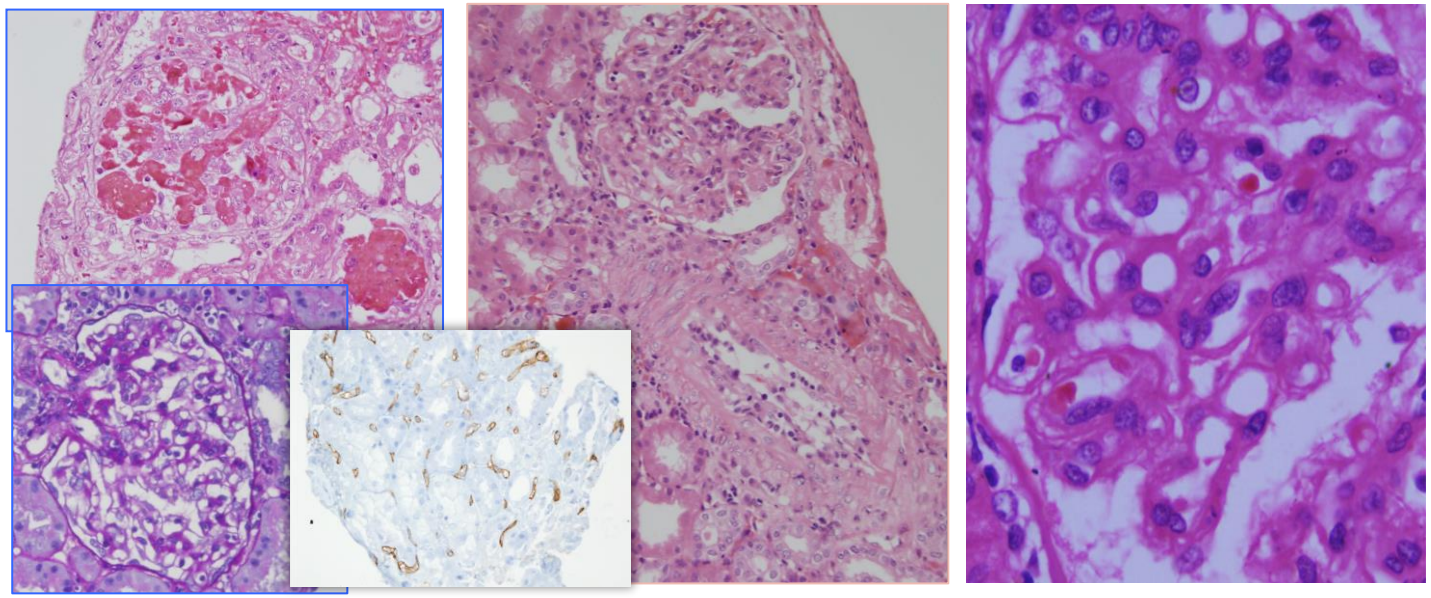
DONÖR SPESİFİK ANTİKORLAR
(negatif ise poz-C4d ve/veya moleküler belirteçler)

Kriter 3

Banff 2022

C4d-pozitif

Mikrovasküler hasar + Pozitif C4d + DSA/poz-C4d	İntimal arterit + Pozitif C4d + DSA/poz-C4d	Kronik doku hasarı + Pozitif C4d + DSA/poz-C4d
C4d-pozitif aktif antikor aracılı rejeksiyon Tip/Grade II	C4d-pozitif aktif antikor aracılı rejeksiyon Tip/Grade III	C4d-pozitif kronik aktif antikor aracılı rejeksiyon



Morfoloji

C4d-poz

DSA/C4d-poz

C4d-negatif

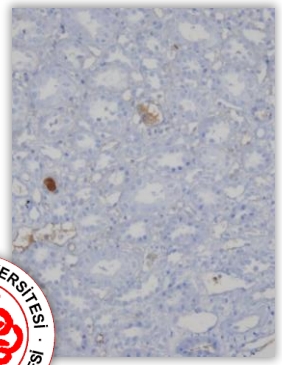
Banff 2022

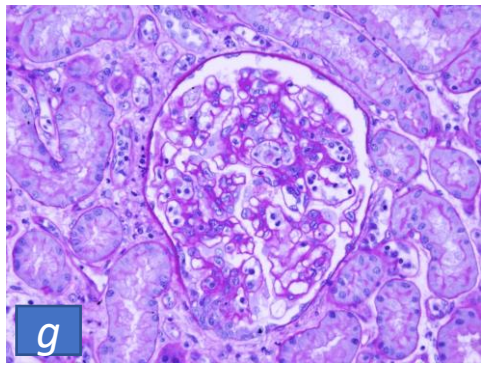
Mikrovasküler hasar
+
g+ptc≥2
+
DSA

C4d-negatif aktif antikor aracılı rejeksiyon

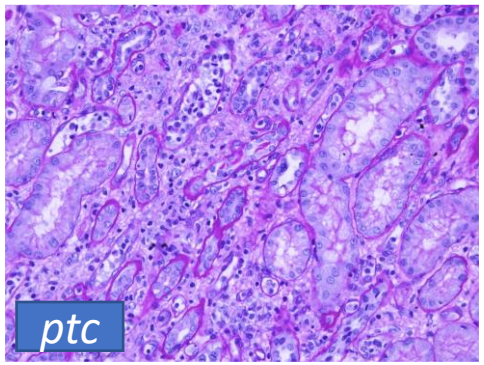
Kronik doku hasarı
+
g+ptc≥2
+
DSA

C4d-negatif kronik aktif antikor aracılı rejeksiyon





+



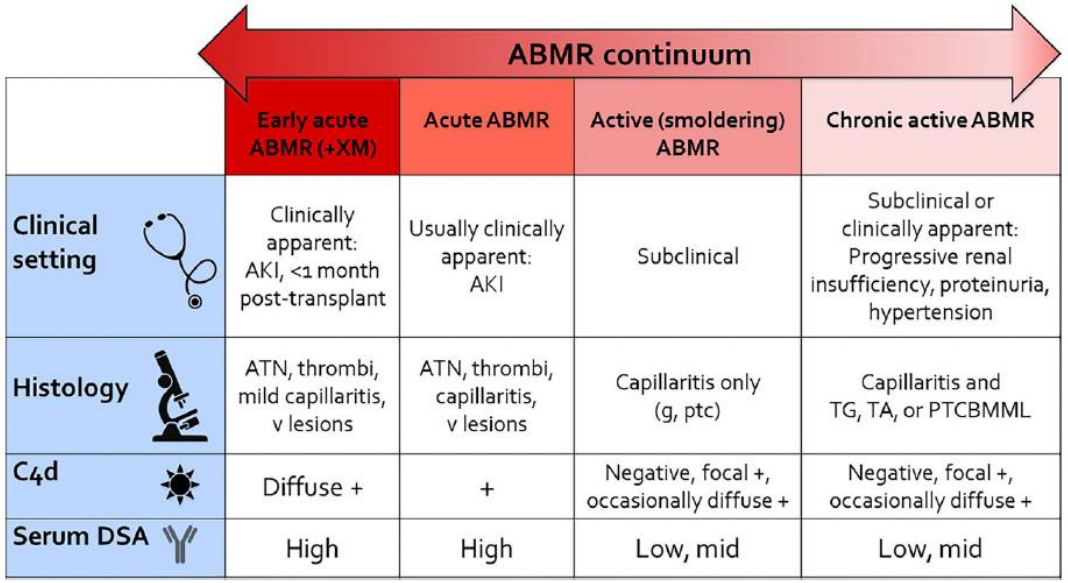
B-HOT
MMDx

Morfoloji

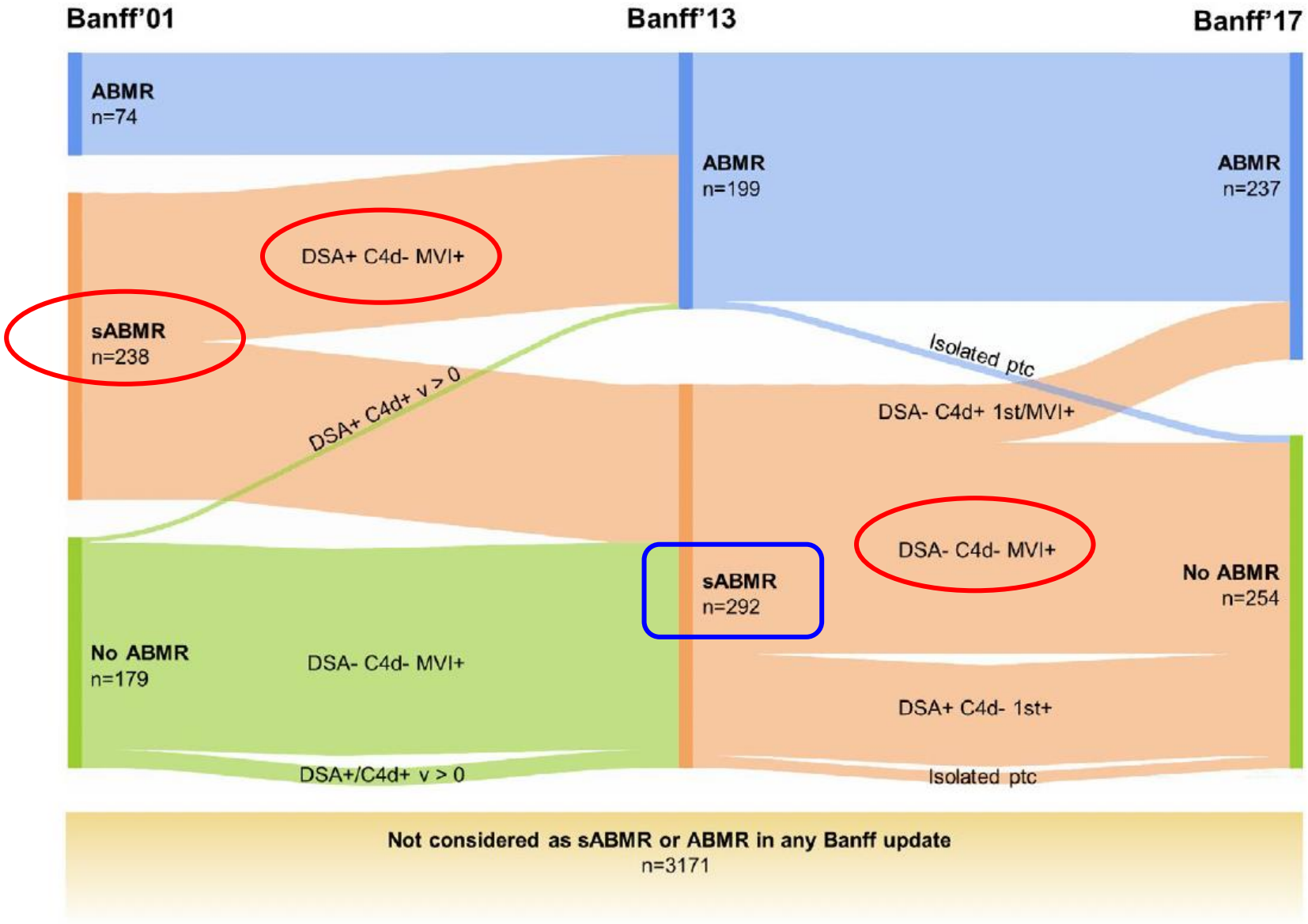
C4d-neg
MVI≥2

DSA

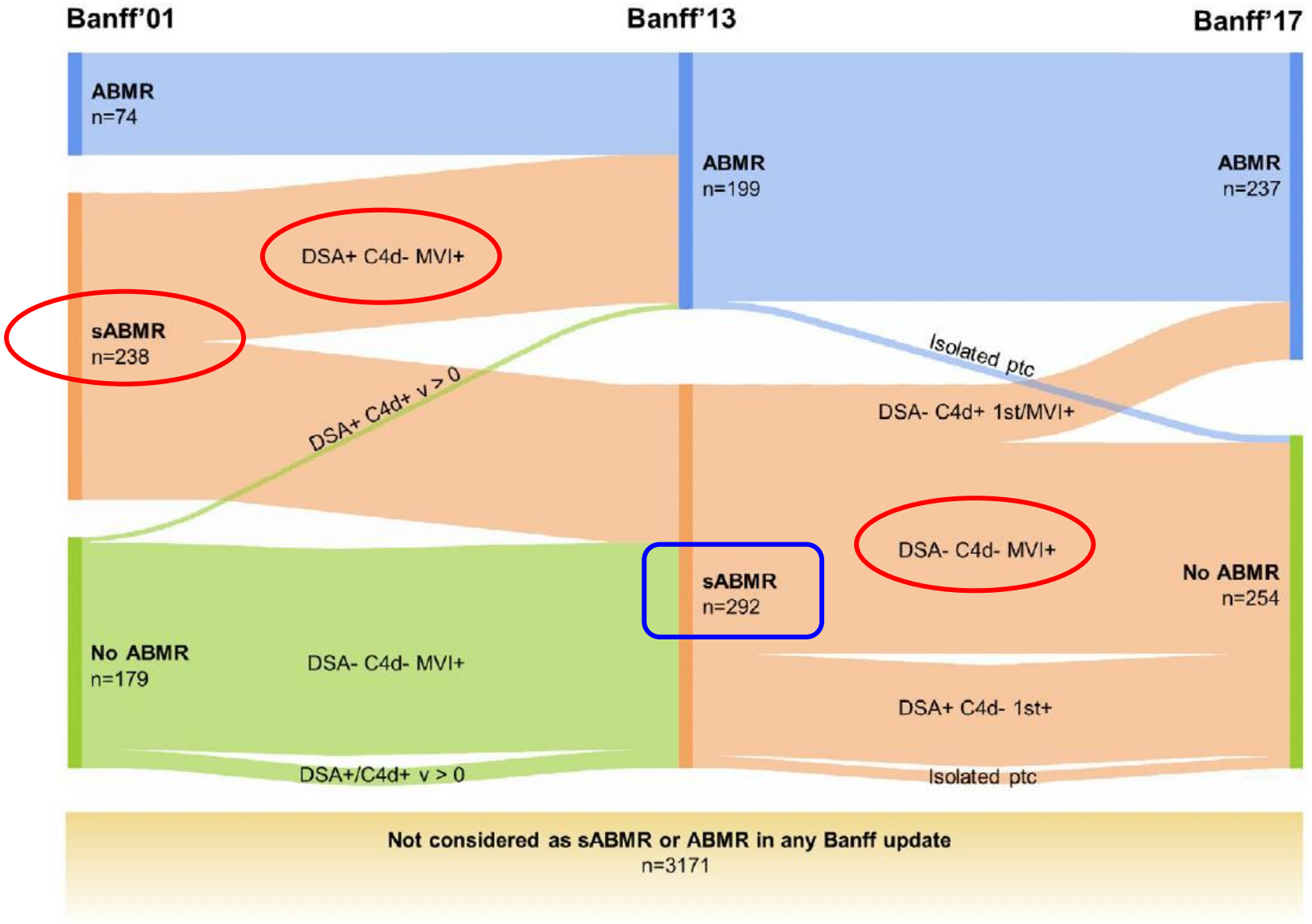
Erken AMR Sensitize hastalar	Geç AMR de novo DSA
Hafıza yanıtı	De novo antikor yanıtı
DSA yüksek titrede pozitif	DSA titreleri değişken, hatta düşük
Kreatininde hızlı artış	Kreatinin yüksekliği olmayabilir, subklinik (?)
Akut tubuler hasar, TMA, v MVI ise yok ya da minimal C4d sıklıkla pozitif	MVI, kronik AMR lezyonları veya mikst rejeksiyon
Tedaviye yanıtlı, kronisite daha geç	Tedaviye sınırlı yanıt



Cornell LD. Front. Immunol. 2021; 12: 718122.



Callemeyn et al. Am J Transplant. 2021;21(7):2413-2423.
doi: 10.1111/ajt.16474.

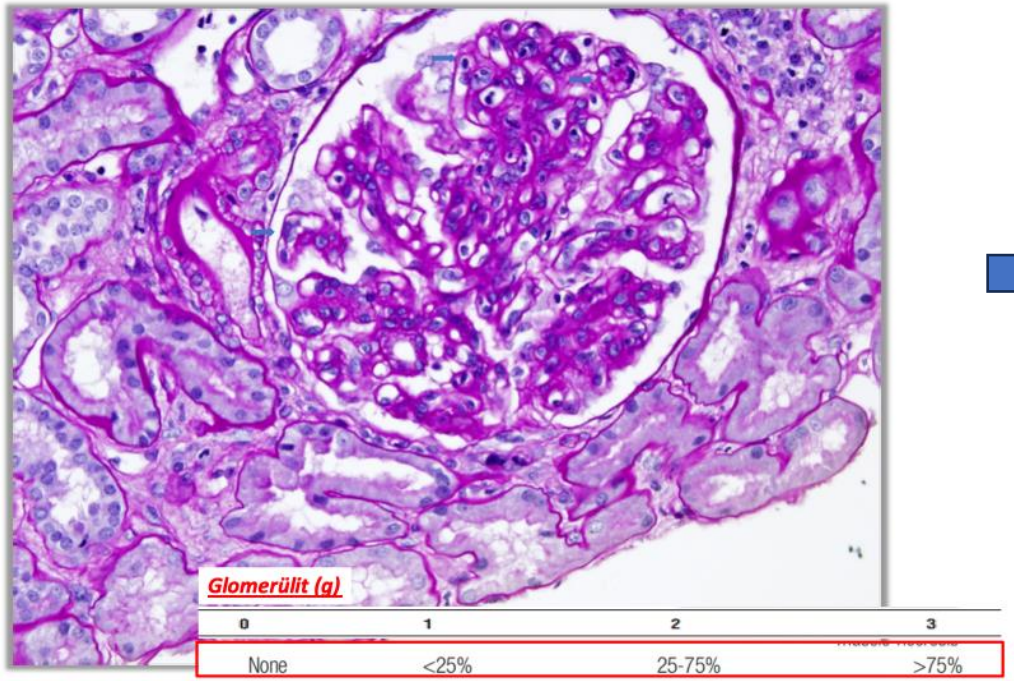


DSA ve C4d-negatif MVI

Banff 2022

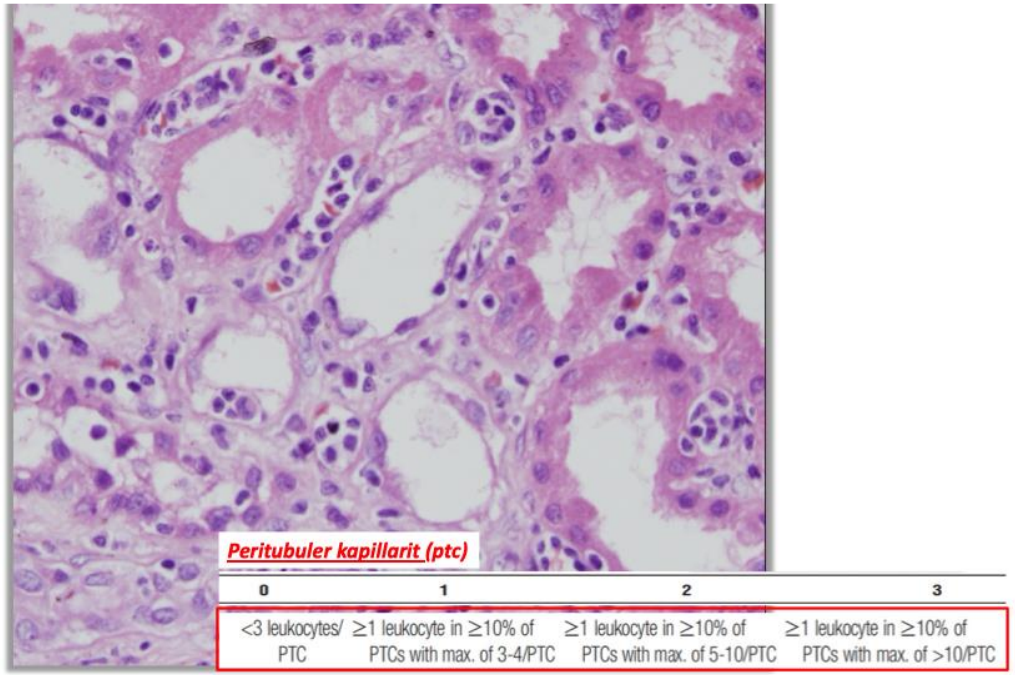
Callemeyn et al. Am J Transplant. 2021;21(7):2413-2423. doi: 10.1111/ajt.16474.

MİKROVASKÜLER İNFLAMASYON



Glomerülit (g)

0	1	2	3
None	<25%	25-75%	>75%



Peritubuler kapillarit (ptc)

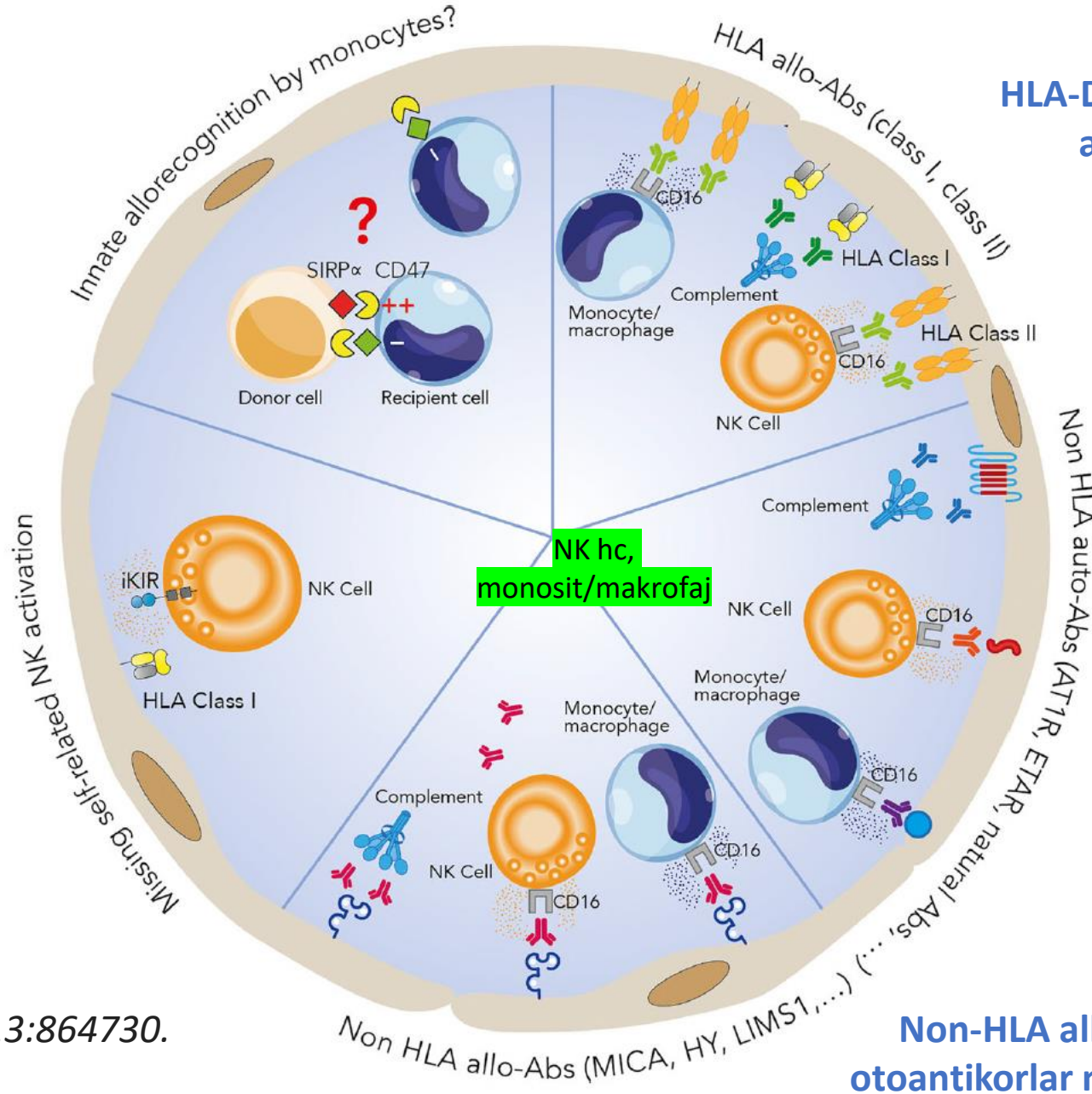
0	1	2	3
<3 leukocytes/ PTC	≥1 leukocyte in ≥10% of PTCs with max. of 3-4/PTC	≥1 leukocyte in ≥10% of PTCs with max. of 5-10/PTC	≥1 leukocyte in ≥10% of PTCs with max. of >10/PTC

DSA-negatif MVI

- Histolojik AMR (hAMR)'lerin yaklaşık yarısı
- Erken dönemde
- Moleküler çalışmalarda test skorları düşük
- Lezyonlarda aktivite yok ve C4d genellikle negatif
- Seyir değişken

Senev et al., Am J Transplant. 2019;19(3):763-780.
Callemeyn et al., J Am Soc Nephrol. 2020;31(9):2168-2183.
Halloran et al., Am J Transplant. 2022;22(8):1976-1991.

Doğal immünite
Antikor bağımsız
Self vs. non-self



HLA-DSA kompleman aktivasyonu

- **MVI bir paterndir, tanı değildir !!!!!**

Lebraud et al., Front Immunol. 2022;13:864730.

Non-HLA alloantikorlar veya otoantikorlar muhtemelen benzer şekilde kompleman aktivasyonu

American Journal of Transplantation 24 (2024) 338-349

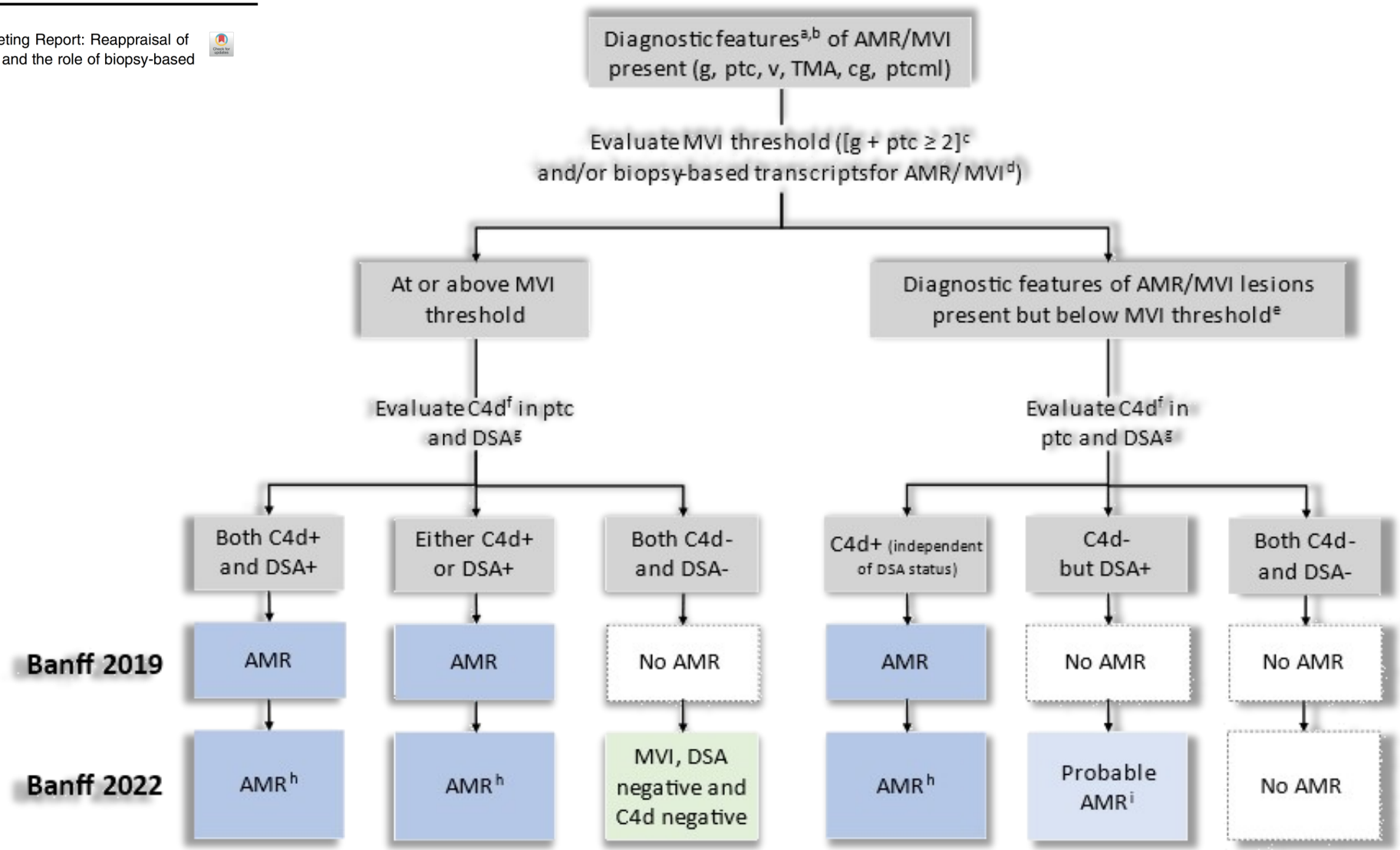
Contents lists available at ScienceDirect

American Journal of Transplantation

journal homepage: www.amjtransplant.org

Meeting report

The Banff 2022 Kidney Meeting Report: Reappraisal of microvascular inflammation and the role of biopsy-based transcript diagnostics



https://banfffoundation.org/central-repository-for-banff-classification-resources-3/

Naesens M et al. Am J Transplant. 2024;24(3):338-349.
doi: 10.1016/j.ajt.2023.10.016.

American Journal of Transplantation 24 (2024) 338-349

Contents lists available at ScienceDirect

American Journal of Transplantation

journal homepage: www.amjtransplant.org

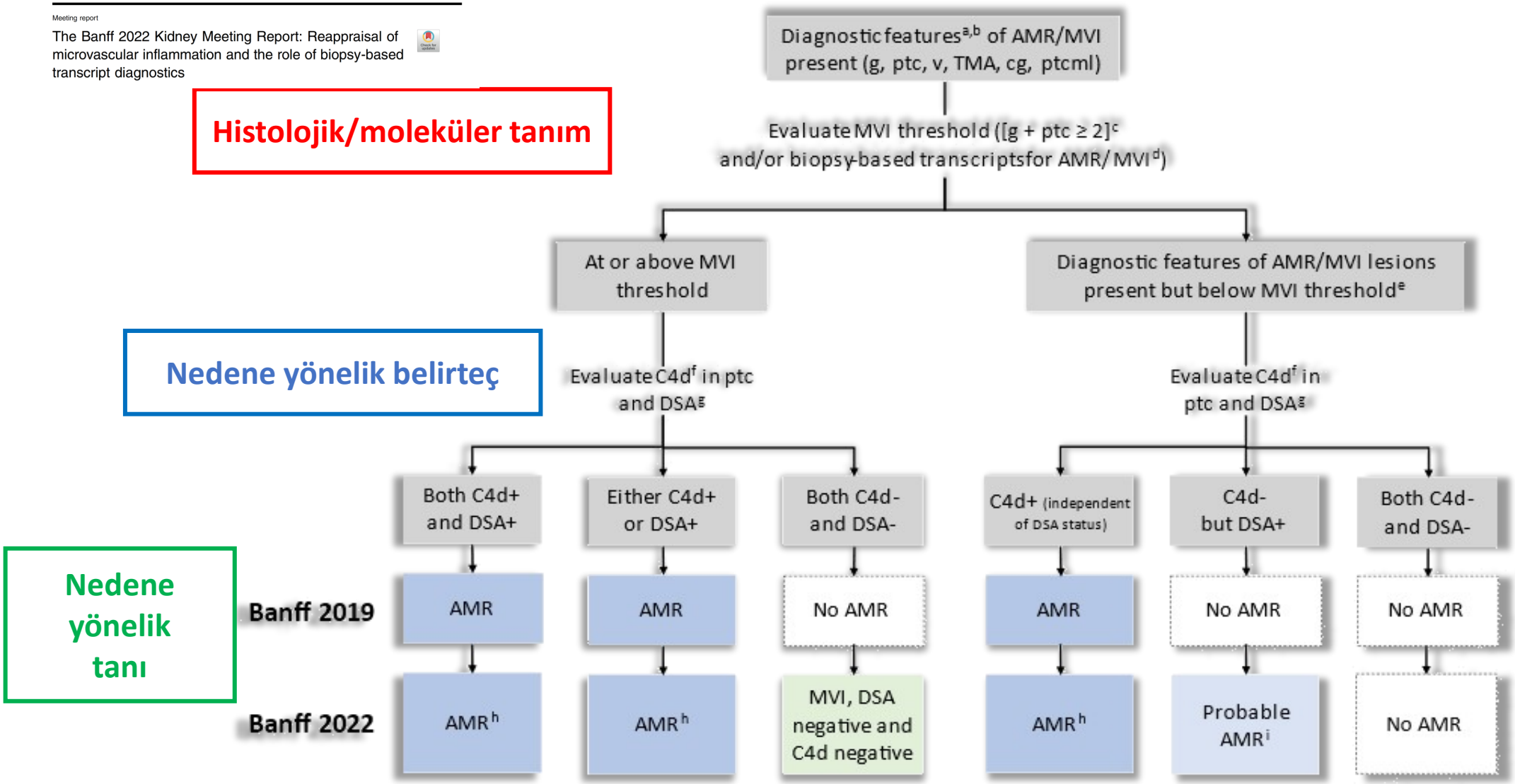
Meeting report

The Banff 2022 Kidney Meeting Report: Reappraisal of microvascular inflammation and the role of biopsy-based transcript diagnostics

Histolojik/moleküler tanım

Nedene yönelik belirteç

Nedene yönelik tanı



https://banfffoundation.org/central-repository-for-banff-classification-resources-3/

Naesens M et al. Am J Transplant. 2024;24(3):338-349. doi: 10.1016/j.ajt.2023.10.016.

American Journal of Transplantation 24 (2024) 338-349

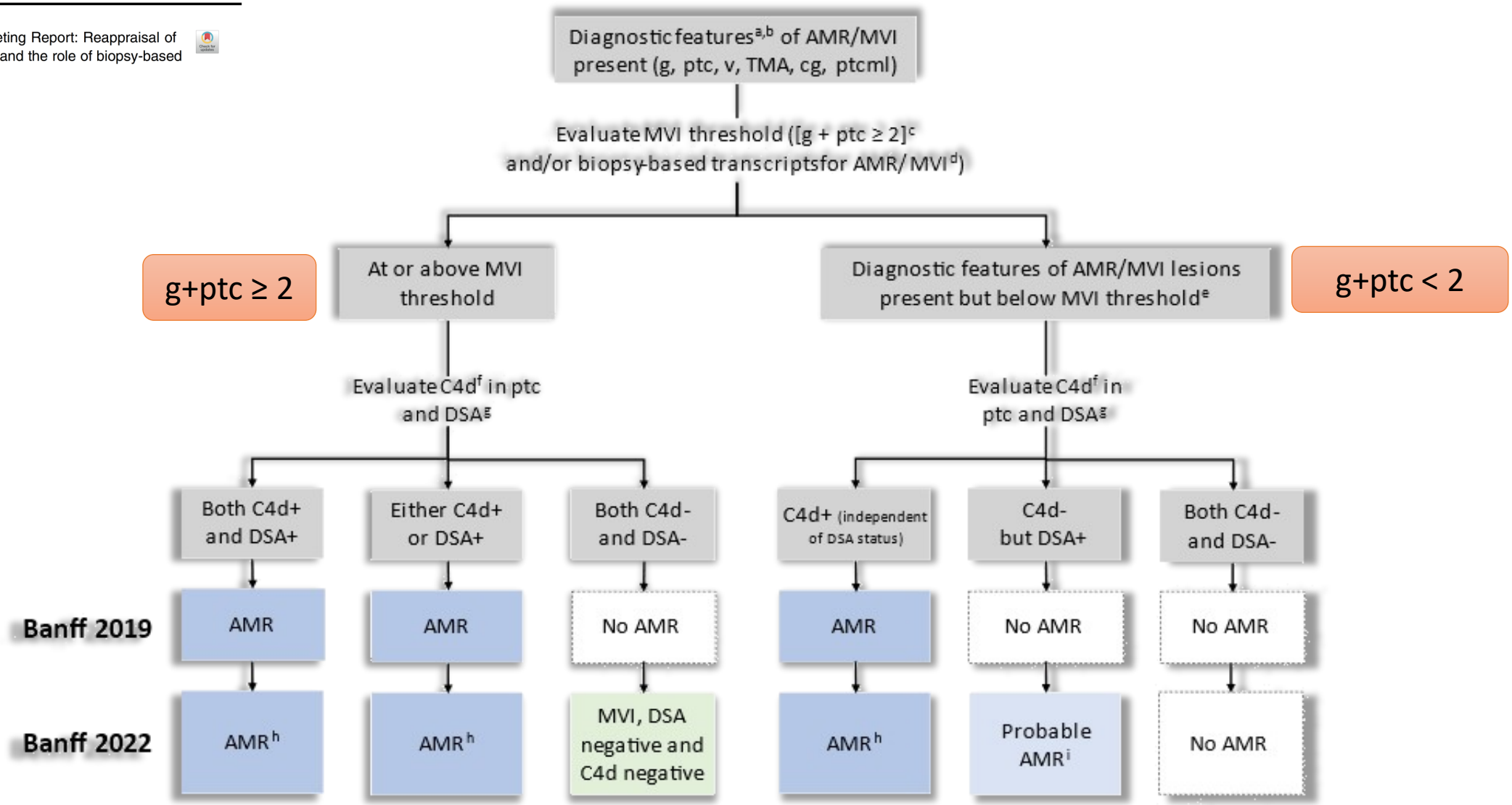
Contents lists available at ScienceDirect

American Journal of Transplantation

journal homepage: www.amjtransplant.org

Meeting report

The Banff 2022 Kidney Meeting Report: Reappraisal of microvascular inflammation and the role of biopsy-based transcript diagnostics



https://banfffoundation.org/central-repository-for-banff-classification-resources-3/

Naesens M et al. Am J Transplant. 2024;24(3):338-349. doi: 10.1016/j.ajt.2023.10.016.

American Journal of Transplantation 24 (2024) 338-349

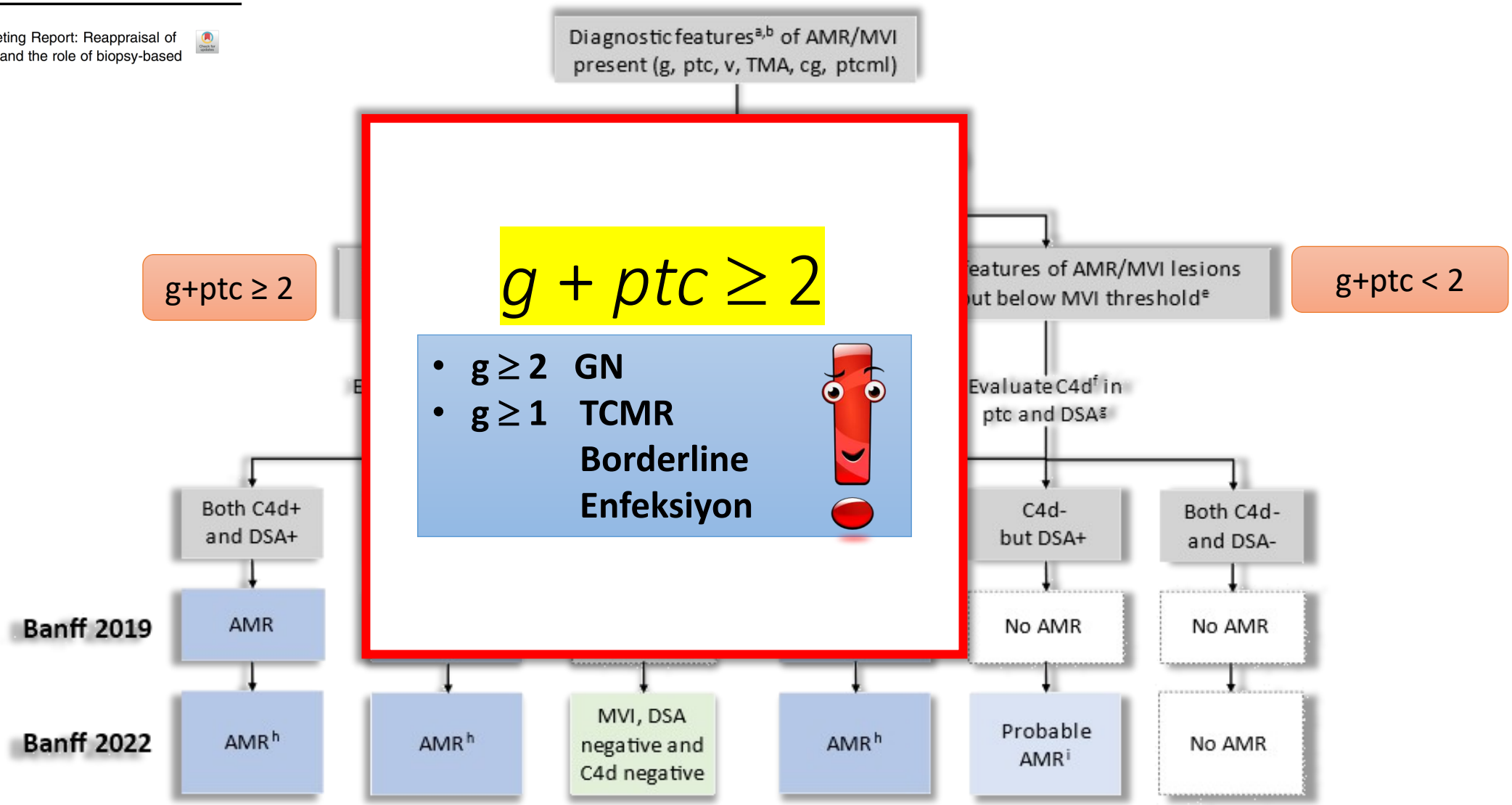
Contents lists available at ScienceDirect

American Journal of Transplantation

journal homepage: www.amjtransplant.org

Meeting report

The Banff 2022 Kidney Meeting Report: Reappraisal of microvascular inflammation and the role of biopsy-based transcript diagnostics



https://banfffoundation.org/central-repository-for-banff-classification-resources-3/

Naesens M et al. Am J Transplant. 2024;24(3):338-349.
doi: 10.1016/j.ajt.2023.10.016.

American Journal of Transplantation 24 (2024) 338-349

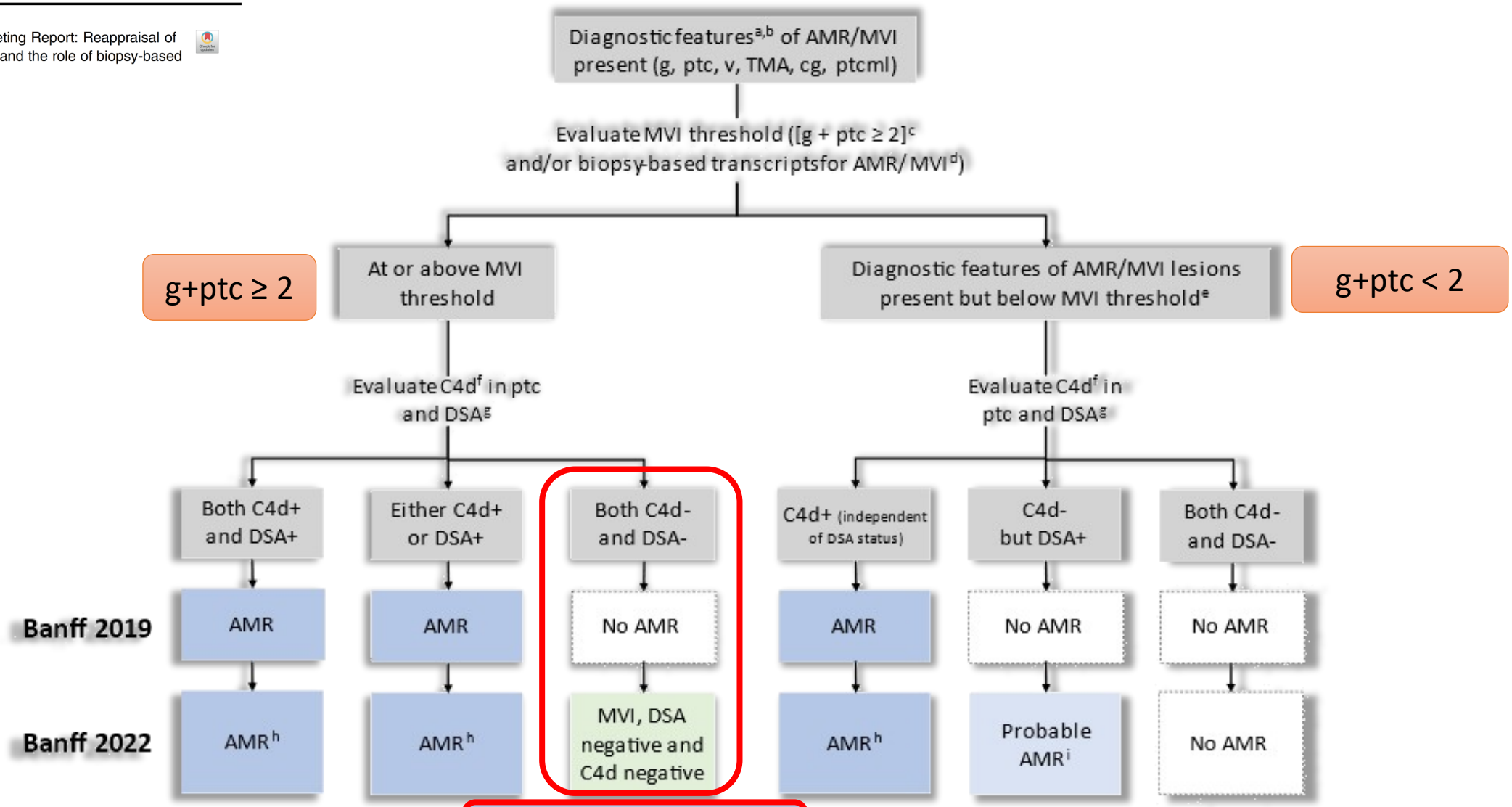
Contents lists available at ScienceDirect

American Journal of Transplantation

journal homepage: www.amjtransplant.org

Meeting report

The Banff 2022 Kidney Meeting Report: Reappraisal of microvascular inflammation and the role of biopsy-based transcript diagnostics



https://banfffoundation.org/central-repository-for-banff-classification-resources-3/

Naesens M et al. Am J Transplant. 2024;24(3):338-349. doi: 10.1016/j.ajt.2023.10.016.

American Journal of Transplantation 24 (2024) 338-349

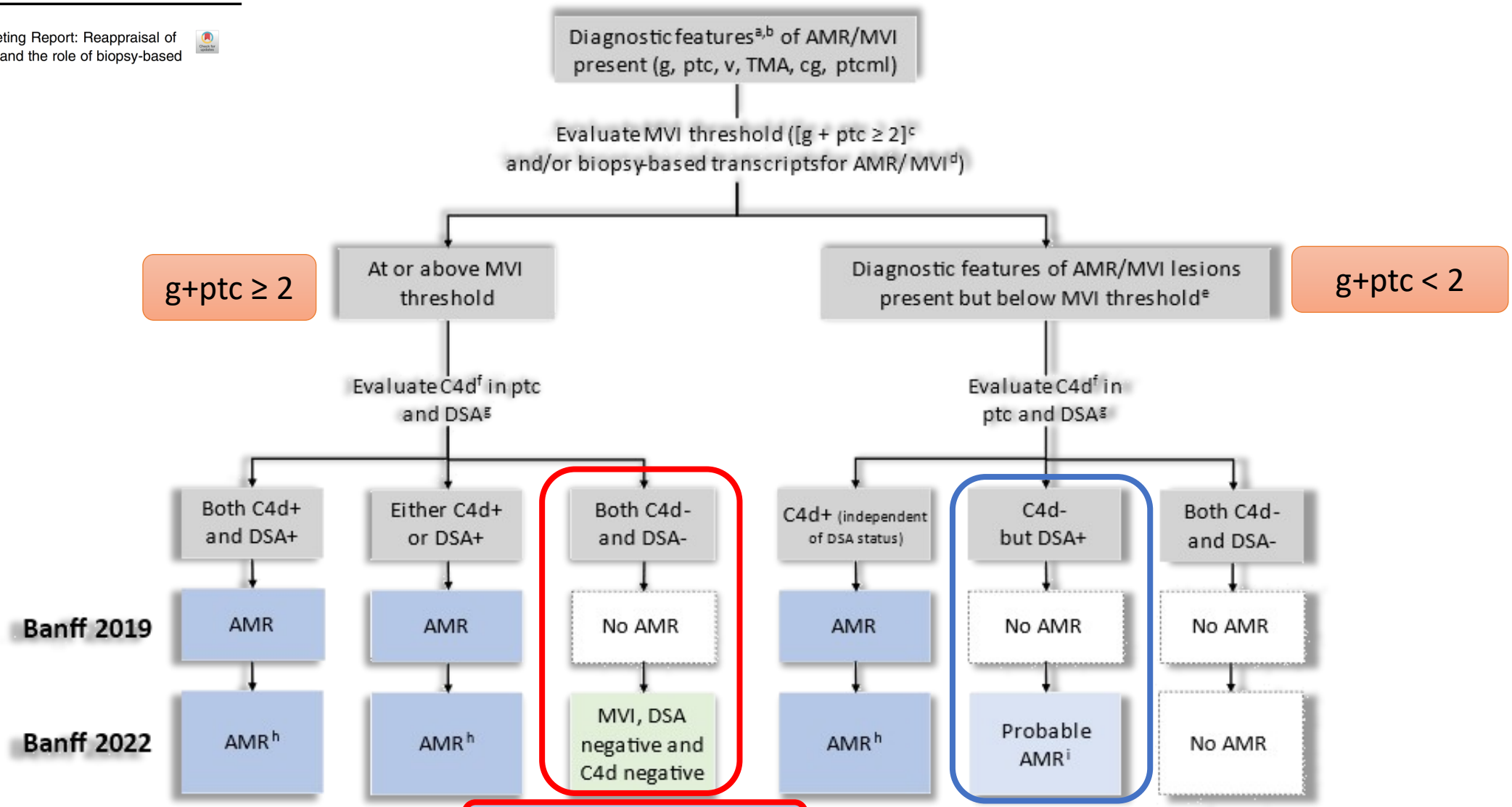
Contents lists available at ScienceDirect

American Journal of Transplantation

journal homepage: www.amjtransplant.org

Meeting report

The Banff 2022 Kidney Meeting Report: Reappraisal of microvascular inflammation and the role of biopsy-based transcript diagnostics



https://banfffoundation.org/central-repository-for-banff-classification-resources-3/

. 2024;24(3):338-349.
doi: 10.1016/j.ajt.2023.10.016.

American Journal of Transplantation 24 (2024) 338-349

Contents lists available at ScienceDirect

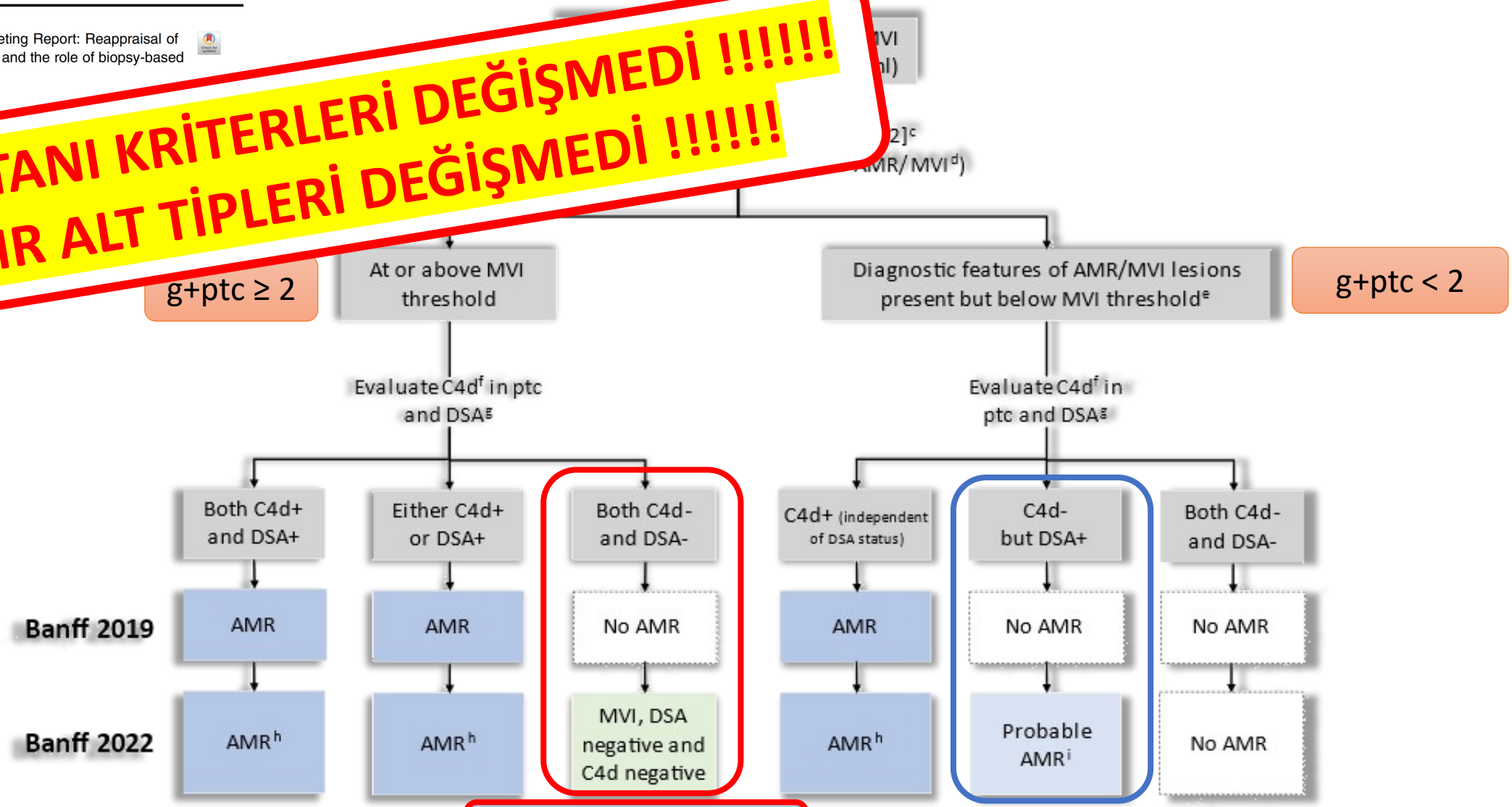
American Journal of Transplantation

journal homepage: www.amjtransplant.org

Meeting report

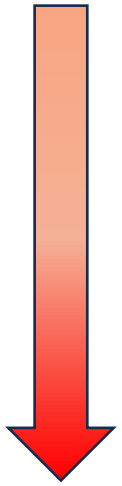
The Banff 2022 Kidney Meeting Report: Reappraisal of microvascular inflammation and the role of biopsy-based transcript diagnostics

AMR TANI KRİTERLERİ DEĞİŞMEDİ !!!!!!
AMR ALT TİPLERİ DEĞİŞMEDİ !!!!!!



https://banfffoundation.org/central-repository-for-banff-classification-resources-3/

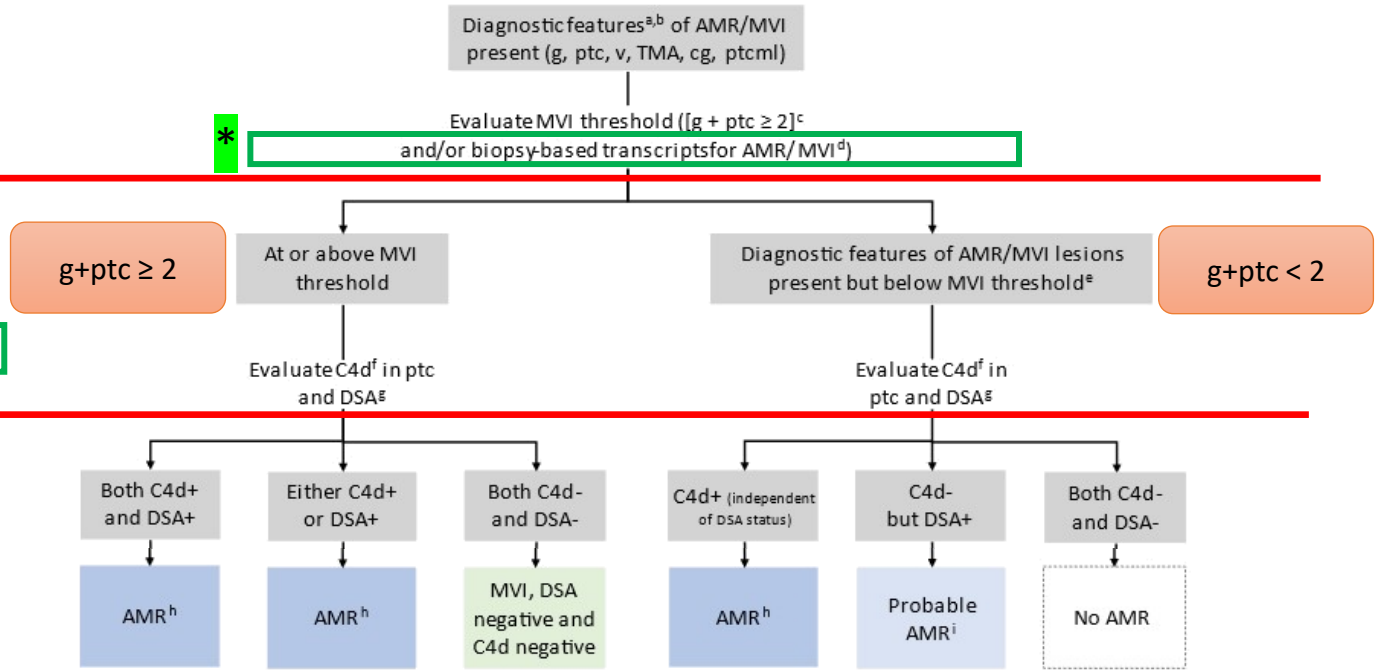
. 2024;24(3):338-349.
doi: 10.1016/j.ajt.2023.10.016.



“SUSPICIOUS”: KUŞKULU/ŞÜPHELİ
“POSSIBLE”: MÜMKÜN/OLANAKLI
“PROBABLE”: OLASI/MUHTEMEL

Table S1

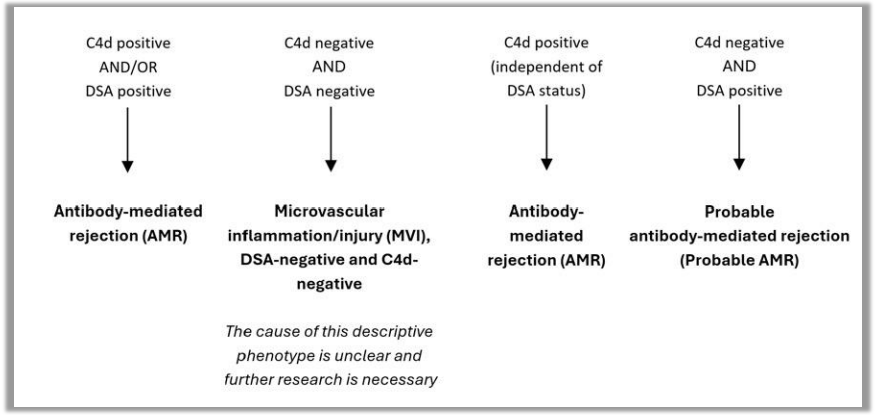
Banff 2022 Category 2: Antibody-mediated rejection and microvascular inflammation/injury (AMR/MVI): a reasoning framework		
1. Diagnostic features* of AMR/MVI present: g, ptc, v, acute TMA, cg, ptcml		
• *Active lesions:* g>0 in the absence of glomerulonephritis; ptc > 0 in the absence of acute TCMR or borderline (suspicious) for acute TCMR; v>0; acute thrombotic microangiopathy (TMA) in the absence of any other cause; • *Chronic lesions:* cg > 0 (by LM or EM if available), if no evidence of chronic TMA and if absence of recurrent or de novo glomerulonephritis; severe ptcml: 7 or more layers in 1 cortical peritubular capillary and 5 or more in 2 additional capillaries, avoiding portions cut tangentially by EM, if available *Other lesions can be observed in AMR and strengthen the diagnosis but are not diagnostic by themselves: arterial intimal fibrosis (cv) of new onset, excluding other causes; leukocytes within the sclerotic intima favour chronic AMR if there is no prior history of TCMR; acute tubular injury, in the absence of any other apparent cause		
Histomolecular Description *Needs further causal annotation, see next row*	**MVI at or above threshold** *at least moderate MVI (g + ptc ≥ 2) in the absence of recurrent or de novo glomerulonephritis. If borderline (suspicious) for or acute TCMR, or infection are present, (g + ptc ≥ 2) is not sufficient and Banff lesion score g≥1 is required* AND/OR	**MVI below threshold** *any of the diagnostic features* of AMR/MVI is present (g, ptc, v, acute TMA, cg, ptcml) but g + ptc < 2; if borderline (suspicious) for or acute TCMR, or infection are present, ptc=1 is not sufficient* AND
* **Biopsy-based transcript diagnostics for AMR/MVI above a defined threshold** *if thoroughly validated for use as substitute for MVI and available*		**Biopsy-based transcript diagnostics for AMR/MVI below a defined threshold** *if thoroughly validated for use as substitute for MVI and available*
Causal Diagnosis or Descriptive Phenotype *If thorough testing for DSA (anti-HLA or other specificity) has not yet been performed, this should be done, following the STAR guidelines.*[1-4] *Detection of non-HLA antibodies (including ABO antibodies in ABO-incompatible transplantation) can be used as serologic Banff criterion for diagnosis of AMR, if the testing protocols are sufficiently standardized and clinically validated for the appropriate clinical context* *C4d deposition should be evaluated in peritubular capillaries and vasa recta (C4d positive = C4d2 or C4d3 by IF on frozen sections, C4d >0 by IHC on paraffin sections)*	C4d positive AND/OR DSA positive → **Antibody-mediated rejection (AMR)** C4d negative AND DSA negative → **Microvascular inflammation/injury (MVI), DSA-negative and C4d-negative** *The cause of this descriptive phenotype is unclear and further research is necessary*	C4d positive (independent of DSA status) → **Antibody-mediated rejection (AMR)** C4d negative AND DSA positive → **Probable antibody-mediated rejection (Probable AMR)**
Activity/Chronicity *Upon diagnosis of AMR, further differentiation of disease stage*	• **Active AMR**: presence of only active lesions (including C4d positivity) (cg=0; ptcml=0) • **Chronic active AMR**: presence of both active (including C4d positivity) and chronic (cg>0 and/or severe ptcml) lesions • **Chronic AMR**: cases with "Probable AMR" with chronic lesions (cg>0 and/or severe ptcml). For these cases, prior documented diagnosis of active or chronic active AMR, or documented prior evidence of DSA, also count as DSA positivity	
2. No diagnostic features of AMR/MVI (g, ptc, v, acute TMA, cg, ptcml) present		
Acute tubular injury and C4d positive *Acute tubular injury (ATI) is present without histological features of AMR/MVI (g, ptc, v, acute TMA, cg, ptcml), C4d positive*	• ABO-incompatibility -> *likely* **"Accommodation"** • Early posttransplant in DSA sensitised (crossmatch positive) patient -> **"Probable AMR"** • DSA negative in conventional transplants -> **"No AMR"**	
C4d staining without evidence of rejection *All 4 features must be present for diagnosis*	• C4d positive • No diagnostic features of AMR/MVI (g, ptc, v, acute TMA, cg, ptcml) present • Biopsy-based transcript diagnostics for AMR/MVI below a defined threshold, *if thoroughly validated and available* • No acute or chronic active TCMR, or borderline changes	



Aktif
Kronik aktif
Kronik

Naesens M et al. Am J Transplant. 2024;24(3):338-349.
doi: 10.1016/j.ajt.2023.10.016.

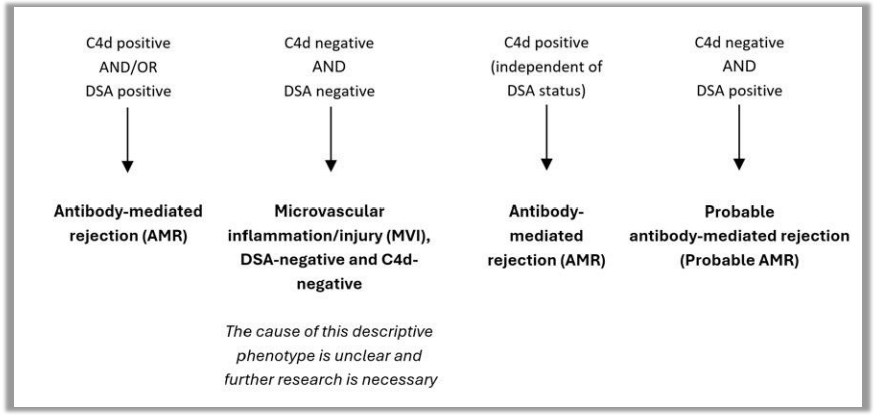
https://banfffoundation.org/central-repository-for-banff-classification-resources-3/



Clinical interpretation of Banff Category 2: Antibody-mediated rejection and microvascular inflammation/injury (AMR/MVI)	
Antibody-mediated (AMR)	AMR can be diagnosed in patients with normal or abnormal kidney function. Further differentiation into active AMR, chronic active AMR, and chronic AMR, can guide therapeutic decision-making.
Probable AMR MVI <2 C4d-neg DSA-poz	In the context of circulating DSA, individual lesions of MVI (g, ptc, v, acute TMA, cg, ptcml) below the histological threshold for MVI (g+ptc<2) and in the absence of C4d deposition in peritubular capillaries, probably indicate antibody activity. This phenotype can be diagnosed in patients with normal or abnormal kidney function. Depending on the clinical context, antibody-targeted treatment could be considered. Further research is necessary to determine the prevalence, impact, and best treatment for this phenotype.
MVI, DSA-negative and C4d-negative MVI >2 C4d-neg DSA-neg	MVI above the histological threshold, without circulating DSA and with negative C4d staining in peritubular capillaries has been observed in patients with normal or abnormal kidney function. This is a purely descriptive phenotype, and the cause remains unclear. Before assigning cases as DSA negative, thorough evaluation of all loci and interactions with the HLA laboratory is necessary, following the STAR guidelines, and the limitations of DSA testing should be considered. Further research is necessary to determine the prevalence, the causes and related biological processes and best treatment for this pattern. These cases may represent missed HLA-DSA, alloreactive T cell mediated responses; autoreactive or alloreactive non-HLA antibodies; primary NK cell activation through missing self; viral infection; other mechanisms of innate immune activation; ischemia reperfusion injury, etc.

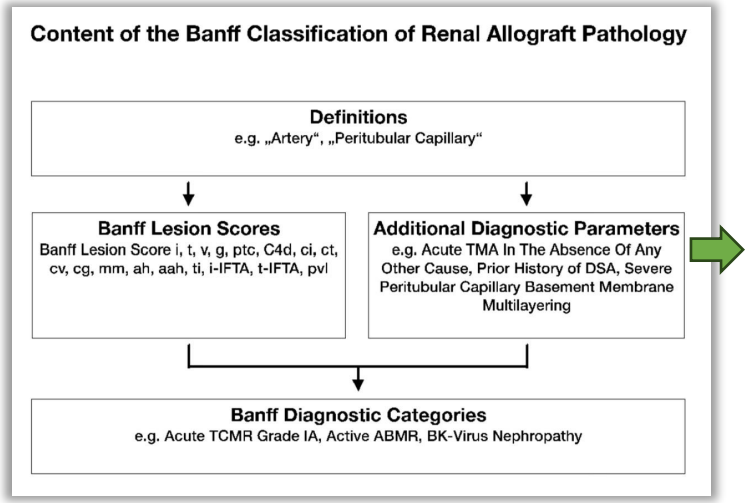
https://banfffoundation.org/central-repository-for-banff-classification-resources-3/

https://banfffoundation.org/central-repository-for-banff-classification-resources-3/



Clinical interpretation of Banff Category 2: Antibody-mediated rejection and microvascular inflammation/injury (AMR/MVI)	
Antibody-mediated (AMR)	AMR can be diagnosed in patients with normal or abnormal kidney function. Further differentiation into active AMR, chronic active AMR, and chronic AMR, can guide therapeutic decision-making.
Probable AMR MVI **<2** C4d-neg DSA-poz	In the context of circulating DSA, individual lesions of MVI (g, ptc, v, acute TMA, cg, ptcml) below the histological threshold for MVI (g+ptc<2) and in the absence of C4d deposition in peritubular capillaries, probably indicate antibody activity. This phenotype can be diagnosed in patients with normal or abnormal kidney function. Depending on the clinical context, antibody-targeted treatment could be considered. Further research is necessary to determine the prevalence, impact, and best treatment for this phenotype.
MVI, DSA-negative and C4d-negative MVI **>2** C4d-neg DSA-neg "DSA-poz AMR" DEĞİL ! "AMR yok" DEĞİL !	MVI above the histological threshold, without circulating DSA and with negative C4d staining in peritubular capillaries has been observed in patients with normal or abnormal kidney function. This is a purely descriptive phenotype, and the cause remains unclear. Before assigning cases as DSA negative, thorough evaluation of all loci and interactions with the HLA laboratory is necessary, following the STAR guidelines, and the limitations of DSA testing should be considered. Further research is necessary to determine the prevalence, the causes and related biological processes and best treatment for this pattern. These cases may represent missed HLA-DSA, alloreactive T cell mediated responses; autoreactive or alloreactive non-HLA antibodies; primary NK cell activation through missing self; viral infection; other mechanisms of innate immune activation; ischemia reperfusion injury, etc.

https://banfffoundation.org/central-repository-for-banff-classification-resources-3/



Ek tanısal bulgular: Tanımı yetersiz olanlar

Parameters
Acute Thrombotic Microangiopathy In The Absence Of Any Other Cause
Absence Of Recurrent Or De Novo Glomerulonephritis
Infection
Biopsy-based transcript diagnostics for AMR/MVI above a defined threshold, if thoroughly validated for use as a substitute for AMR/MVI and available
Severe Peritubular Capillary Basement Membrane Multilayering
Arterial Intimal Fibrosis With Mononuclear Cell Inflammation In Fibrosis And Formation Of Neointima
Prior Evidence Of DSA
Serologic Evidence Of DSAs (DSA To HLA Or Other Antigens)
Prior Documented Diagnosis Of Active Or Chronic Active AMR
Prior History Of TCMR
Evidence Of Chronic TMA
C4d Staining On Fresh-Frozen Or Paraffin-Embedded Tissue
Polyomavirus Nephropathy, Posttransplant Lymphoproliferative Disorder, Calcineurin Inhibitor Toxicity, Acute Tubular Injury Recurrent Disease, De Novo Glomerulopathy (Other Than TG), Pyelonephritis, Drug-Induced Interstitial Nephritis
Other Known Causes Of i-IFTA Ruled Out

Transplantation 2018;102: 1795–1814

https://banfffoundation.org/central-repository-for-banff-classification-resources-3/

Table S1

Banff 2022 Category 2: Antibody-mediated rejection and microvascular inflammation/injury (AMR/MVI): a reasoning framework

1. Diagnostic features* of AMR/MVI present: g, ptc, v, acute TMA, cg, ptcml

- *Active lesions: g>0 in the absence of glomerulonephritis; ptc > 0 in the absence of acute TCMR or borderline (suspicious) for acute TCMR; v>0; acute thrombotic microangiopathy (TMA) in the absence of any other cause;*
- *Chronic lesions: cg > 0 (by LM or EM if available), if no evidence of chronic TMA and if absence of recurrent or de novo glomerulonephritis; severe ptcml: 7 or more layers in 1 cortical peritubular capillary and 5 or more in 2 additional capillaries, avoiding portions cut tangentially by EM, if available*

**Other lesions can be observed in AMR and strengthen the diagnosis but are not diagnostic by themselves: arterial intimal fibrosis (cv) of new onset, excluding other causes; leukocytes within the sclerotic intima favour chronic AMR if there is no prior history of TCMR; acute tubular injury, in the absence of any other apparent cause*

	MVI at or above threshold		MVI below threshold	
Histomolecular Description *Needs further causal annotation, see next row*	*at least moderate MVI (g + ptc ≥ 2) in the absence of recurrent or de novo glomerulonephritis. If borderline (suspicious) for or acute TCMR, or infection are present, (g + ptc ≥ 2) is not sufficient and Banff lesion score g≥1 is required* AND/OR **Biopsy-based transcript diagnostics for AMR/MVI above a defined threshold** *if thoroughly validated for use as substitute for MVI and available*		*any of the diagnostic features* of AMR/MVI is present (g, ptc, v, acute TMA, cg, ptcml) but g + ptc < 2; if borderline (suspicious) for or acute TCMR, or infection are present, ptc=1 is not sufficient* AND **Biopsy-based transcript diagnostics for AMR/MVI below a defined threshold** *if thoroughly validated for use as substitute for MVI and available*	
Causal Diagnosis or Descriptive Phenotype *If thorough testing for DSA (anti-HLA or other specificity) has not yet been performed, this should be done, following the STAR guidelines.*[1-4] *Detection of non-HLA antibodies (including ABO antibodies in ABO-incompatible transplantation) can be used as serologic Banff criterion for diagnosis of AMR, if the testing protocols are sufficiently standardized and clinically validated for the appropriate clinical context* *C4d deposition should be evaluated in peritubular capillaries and vasa recta (C4d positive = C4d2 or C4d3 by IF on frozen sections, C4d >0 by IHC on paraffin sections)*	C4d positive AND/OR DSA positive	C4d negative AND DSA negative	C4d positive (independent of DSA status)	C4d negative AND DSA positive
	Antibody-mediated rejection (AMR)	**Microvascular inflammation/injury (MVI), DSA-negative and C4d-negative** *The cause of this descriptive phenotype is unclear and further research is necessary*	**Antibody-mediated rejection (AMR)**	**Probable antibody-mediated rejection (Probable AMR)**
Activity/Chronicity *Upon diagnosis of AMR, further differentiation of disease stage*	• **Active AMR**: presence of only active lesions (including C4d positivity) (cg=0; ptcml=0) • **Chronic active AMR**: presence of both active (including C4d positivity) and chronic (cg>0 and/or severe ptcml) lesions • **Chronic AMR**: cases with "Probable AMR" with chronic lesions (cg>0 and/or severe ptcml). For these cases, prior documented diagnosis of active or chronic active AMR, or documented prior evidence of DSA, also count as DSA positivity			

2. No diagnostic features of AMR/MVI (g, ptc, v, acute TMA, cg, ptcml) present

Acute tubular injury and C4d positive *Acute tubular injury (ATI) is present without histological features of AMR/MVI (g, ptc, v, acute TMA, cg, ptcml), C4d positive*	• ABO-incompatibility -> *likely* **"Accommodation"** • Early posttransplant in DSA sensitised (crossmatch positive) patient -> **"Probable AMR"** • DSA negative in conventional transplants -> **"No AMR"**
C4d staining without evidence of rejection *All 4 features must be present for diagnosis*	• C4d positive • No diagnostic features of AMR/MVI (g, ptc, v, acute TMA, cg, ptcml) present • Biopsy-based transcript diagnostics for AMR/MVI below a defined threshold, *if thoroughly validated and available* • No acute or chronic active TCMR, or borderline changes

Akomodasyon: ABOi
Olası AMR: XMpoz/sens hasta erken dönemde
AMR değil: konvansiyonel tx, çoğunda DSA negatif

Akut tubuler hasar + C4d-poz
ATI çok sık ve yeterince tanımlı değil (!)
Aktif AMR bulgusu olmamalı (!)

Rejeksiyon olmaksızın C4d-poz
Moleküler testler rej değerlerinin altında (!)
Eş zamanlı borderline/TCMR/AMR olmamalı (!)

Yeni ortaya çıkan intimal fibrozis

- Sık rastlanan bir lezyon
- HLA antikorları ateroskleroz yapabilir
- Arter örneklemesi çoğu zaman yeterli değil
- Tekrarlanabilirlik düşük
- Eski bir bx varlığını gerektirir

- Çoğu patolog cg/ptcml olmadan cv ile AMR tanısı vermiyor (!)

AMR diğer sorunları

- GN varlığında g
- BL/TCMR varlığında ptc
- İzole v ve izole TMA
- C4d’nin DSA yerine kullanımı

AMR

Borderline/caTCMR

Aktivite ve kronisite indeksleri

MEETING REPORT

AJT

The Banff 2017 Kidney Meeting Report: Revised diagnostic criteria for chronic active T cell–mediated rejection, antibody-mediated rejection, and prospects for integrative endpoints for next-generation clinical trials

Category 3: Borderline changes

Suspicious (Borderline) for acute TCMR

Foci of tubulitis (t > 0) with minor interstitial inflammation (i0 or i1), or moderate-severe interstitial inflammation (i2 or i3) with mild (t1) tubulitis; retaining the i1 threshold for borderline with t > 0 is permitted although this must be made transparent in reports and publications

MEETING REPORT

AJT

The Banff 2019 Kidney Meeting Report (I): Updates on and clarification of criteria for T cell– and antibody-mediated rejection

Category 3: Borderline (Suspicious) for acute TCMR

Foci of tubulitis (t1, t2, or t3) with **mild interstitial inflammation (i1)** or mild (t1) tubulitis with moderate-severe interstitial inflammation (i2 or i3)

No intimal or transmural arteritis (v = 0)

Kidney International, Vol. 55 (1999), pp. 713–723

The Banff 97 working classification of renal allograft pathology

American Journal of Transplantation 2017; 17: 28–41
Wiley Periodicals Inc.

doi: 10.1111/ajt.14107

Meeting Report

The Banff 2015 Kidney Meeting Report: Current Challenges in Rejection Classification and Prospects for Adopting Molecular Pathology

Received: 12 November 2017 | Revised: 30 March 2018 | Accepted: 31 March 2018
DOI: 10.1111/ajt.14888

ORIGINAL ARTICLE

AJT

Does tubulitis without interstitial inflammation represent borderline acute T cell mediated rejection?

Brian J. Nankivell[1] | Chow H. P'Ng[2] | Jeremy R. Chapman[1]

www.nature.com/scientificreports

scientific reports

Check for updates

OPEN

Molecular patterns of isolated tubulitis differ from tubulitis with interstitial inflammation in early indication biopsies of kidney allografts

Are borderline changes real rejection? Current viewpoints

Sook Hyeon Park[a] *and John J. Friedewald*[a,b]

Table 2. Borderline rejection article summary

Authors	Study participants	Key findings
Nankivell *et al.* [19▪▪]	*N*=775	1,5- year graft survival: isolated tubulitis (98.8%, 92.7%) and normal controls (98.1%, 91.7%) were higher than BL with i1ti (94.5%, 81.7%) ($P<0.001$).
McRae *et al.* [20]	*N*=172, *N*=56 for external validation	BL with ≥ i1t1 had higher HR for CCE (hazard ratio = 3.4; 95% CI, 1.4–8.2; $P<0.01$) than BL with t1i0
Nankivell *et al.* [21▪▪]	*N*=551	BL with ≥ t1i1 developed more microvascular inflammation, positive C4d, TG, and dnDSA than normal controls BL is associated with lower 5-year graft and patient survival
Seifert *et al.* [22]	*N*=120	BL is associated with an increased adjusted hazard ratio 2.6 for CCE The treated BL had a lower incidence of CCE (41%, $P<0.001$) than the untreated group (67%)
Mehta *et al.* [23]	*N*=200	SCI (excluding Banff ≥ 1A) at 3 -month protocol biopsy (i+t > 0) had higher serum creatinine at 24-month than normal controls (1.65 ± 0.85 mg/dl vs. 1.39 ± 0.45 mg/dl, $P=0.02$) The SCI group had higher allograft chronicity score at 12-month than normal controls (2.4 ± 1.35 vs. 1.9 ± 1.2, $P=0.02$)
Wiebe *et al.* [24▪]	*N*=803	BL with ≥ t1i1 is associated with reduced graft survival (hazard ratio = 2.4; $P=0.003$) than normal controls HLA DR/DQ eplet mismatch is associated with BL and TCMR

Kidney allograft outcomes with borderline changes.
BL, borderline changes; CCE, composite clinical endpoints; dnDSA, *de novo* DSA; KT, kidney transplant; SCI, subclinical inflammation; TG, transplant glomerulopathy.

- Borderline (≥t1i1) graft kaybı ve artmış inflamasyon-fibrozis ile ilişkili
- Protokol bx'lerde tedavi edilen veya edilmeyenlerde seyir değişken

Curr Opin Nephrol Hypertens 2020, 29:656–662

Are borderline changes real rejection? Current viewpoints

Sook Hyeon Park[a] *and John J. Friedewald*[a,b]

Banff 2022 i1 eşik değerini koruyor !!

Table 2. Borderline rejection article summary

Authors	Study participants	Key findings
Nankivell *et al.* [19▪▪]	*N*=775	1,5- year graft survival 91.7%) were hi
McRae *et al.* [20]	*N*=172, *N*=56 for external validation	BL with ≥ i th .01)
Nankivell *et al.* [21▪▪]	*N*=551	e C4d, TG, and ival
Seifert *et al.* [22]		d hazard ratio 2.6 for CCE e of CCE (41%, *P*<0.001) than the untreated group
Mehta *et al.* [23]	*N*=2	TA) at 3 -month protocol biopsy (i+t > 0) had higher serum month than normal controls (1.65±0.85 mg/dl vs. 1.39±0.45 mg/ 2) group had higher allograft chronicity score at 12-month than normal controls (2.4±1.35 vs. 1.9±1.2, *P*=0.02)
Wiebe *et al.* [24▪]	*N*=803	BL with ≥ t1i1 is associated with reduced graft survival (hazard ratio = 2.4; *P*=0.003) than normal controls HLA DR/DQ eplet mismatch is associated with BL and TCMR

Kidney allograft outcomes with borderline changes.
BL, borderline changes; CCE, composite clinical endpoints; dnDSA, *de novo* DSA; KT, kidney transplant; SCI, subclinical inflammation; TG, transplant glomerulopathy.

- Borderline (≥t1i1) graft kaybı ve artmış inflamasyon-fibrozis ile ilişkili
- Protokol bx'lerde tedavi edilen veya edilmeyenlerde seyir değişken

Curr Opin Nephrol Hypertens 2020, 29:656–662

T HÜCRE ARACILI REJEKSİYON, AKUT

i=1 + t≥1 + v=0

i≥2 + t=1 + v=0

Akut T hücre aracılı rejeksiyon kuşkusu "Borderline"

i≥2 + t=2 + v=0

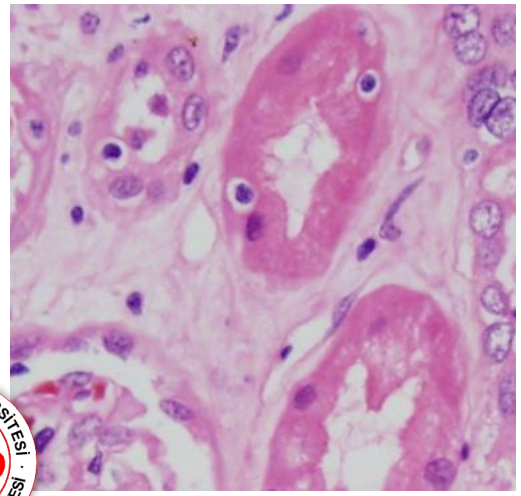
Akut T hücre aracılı rejeksiyon Tip/Grade IA

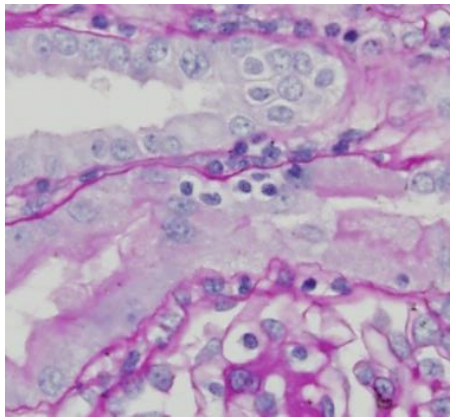
i≥2 + t=3 + v=0

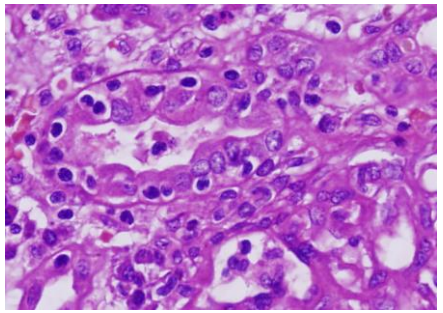
Akut T hücre aracılı rejeksiyon Tip/Grade IB

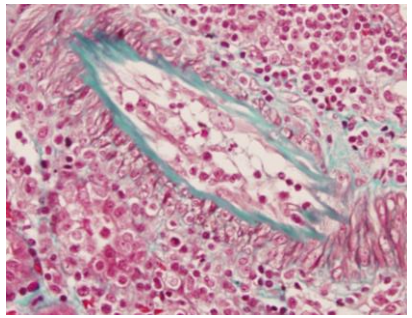
v > 0

Akut T hücre aracılı rejeksiyon Tip/Grade II









i

t

v

İSTANBUL ÜNİVERSİTESİ · İSTANBUL TIP FAKÜLTESİ ·

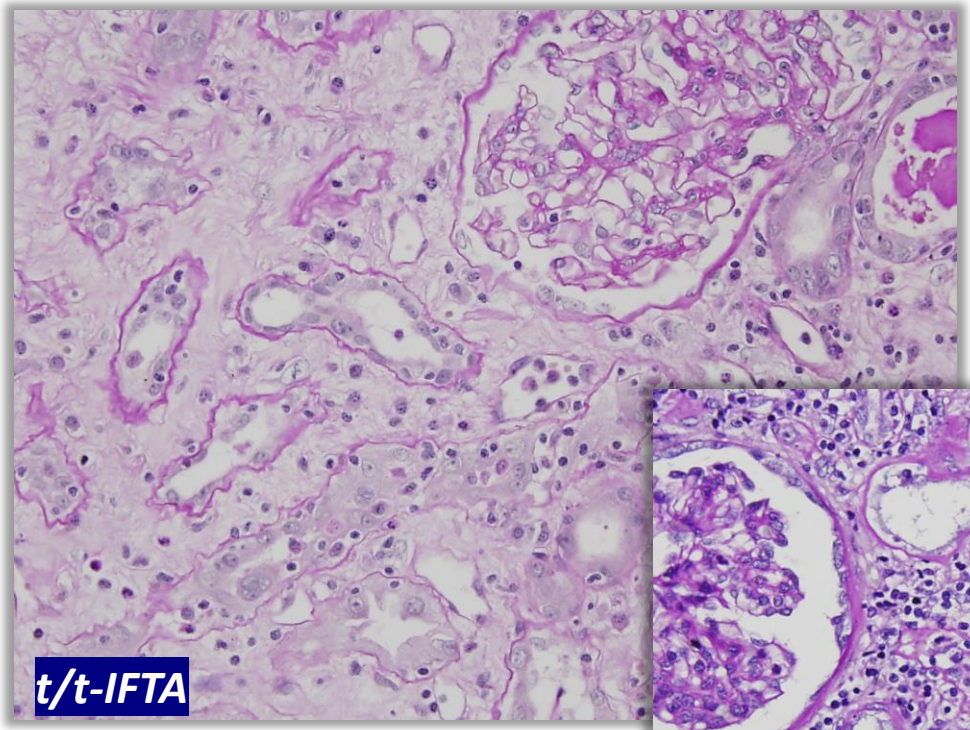
ti

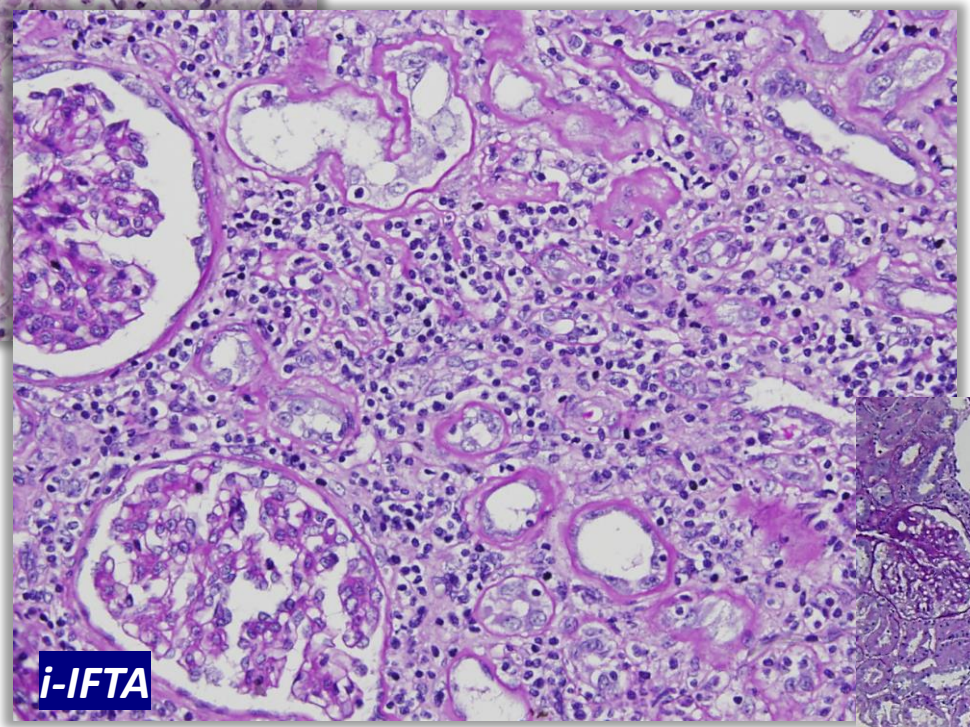
Total inflamasyon (ti): 5 / 11

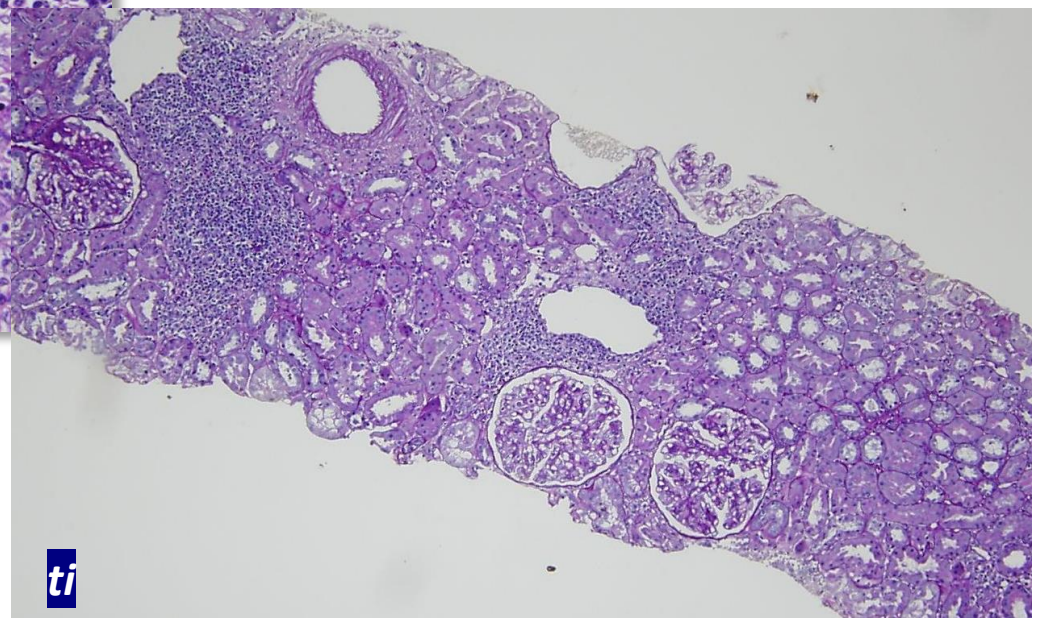
İnterstisyel inflamasyon (i): 3 / 6

Skarlı kortekste inflamasyon (i-IFTA): 2 / 5

PAY / PAYDA







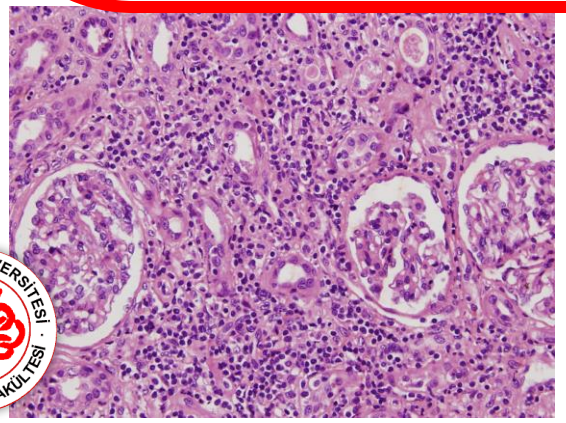
İSTANBUL ÜNİVERSİTESİ
İSTANBUL TIP FAKÜLTESİ

T HÜCRE ARACILI REJEKSİYON, KRONİK AKTİF

Banff 2019

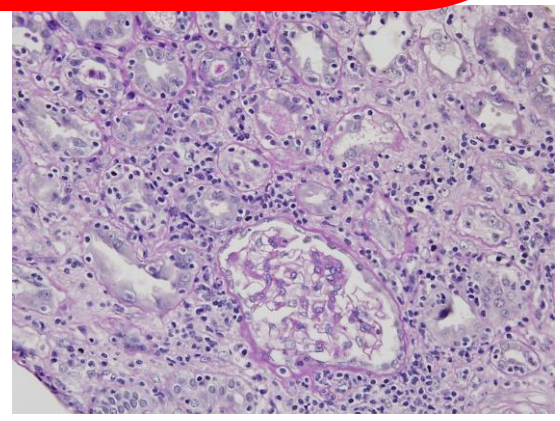
ti≥2
+
i-IFTA≥2
+
t=2 / t-IFTA=2

→ Kronik aktif T hücre aracılı rejeksiyon
Tip/Grade IA



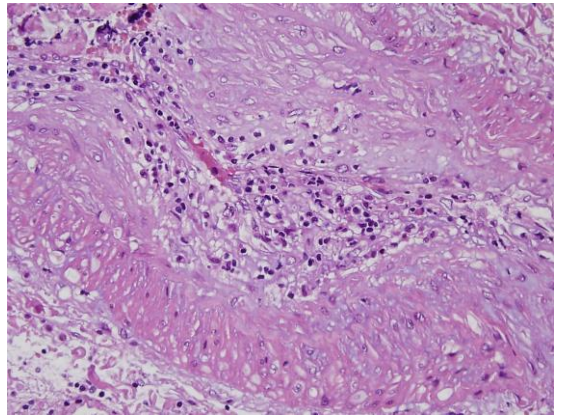
ti≥2
+
i-IFTA≥2
+
t=3 / t-IFTA=3

→ Kronik aktif T hücre aracılı rejeksiyon
Tip/Grade IB



cv > 0
(mononükleer inflamasyon ile birlikte,neointima)

→ Kronik aktif T hücre aracılı rejeksiyon
Tip/Grade II



ti

i-IFTA

t/t-IFTA

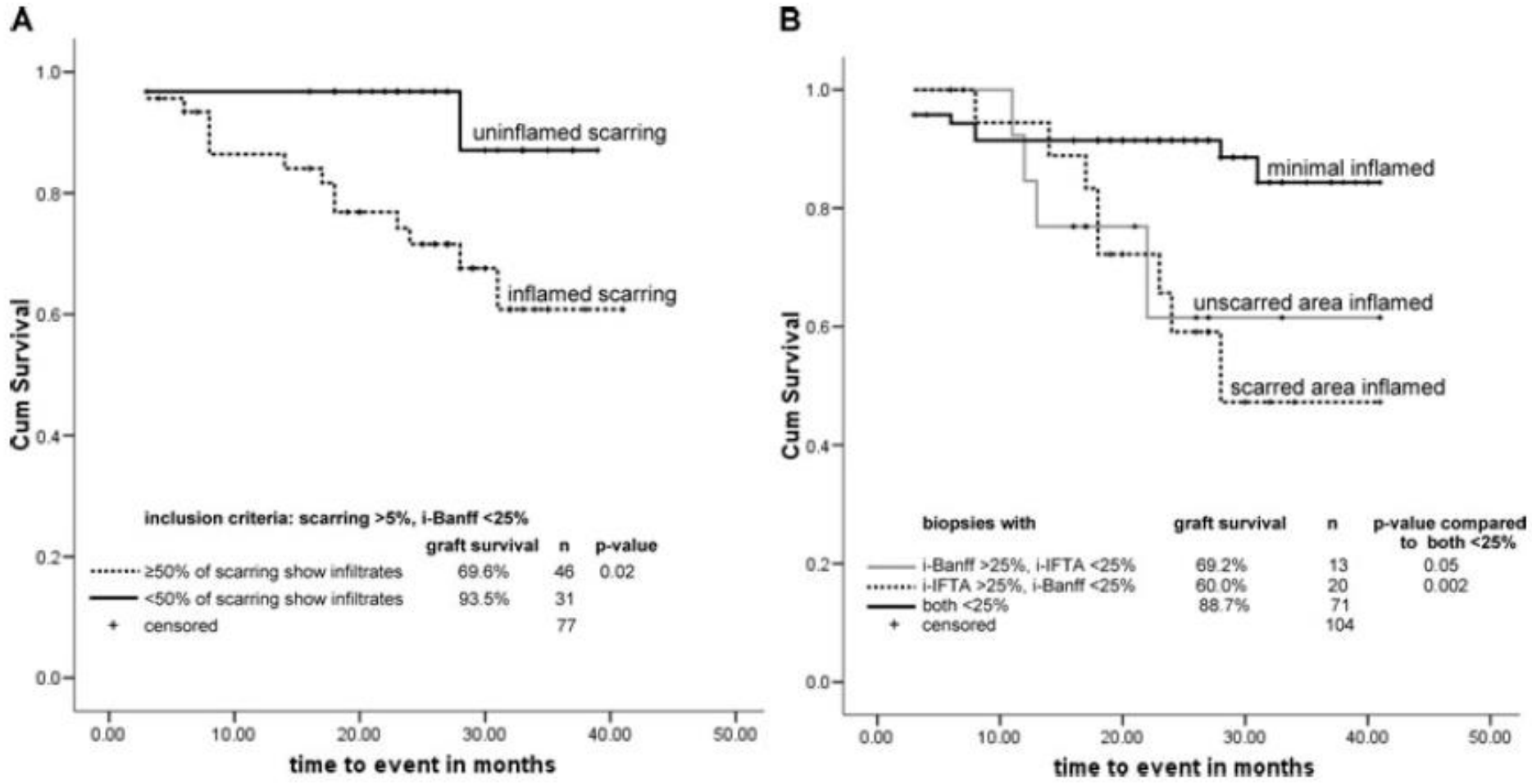
cv

İSTANBUL ÜNİVERSİTESİ · İSTANBUL TIP FAKÜLTESİ ·

caTCMR

- Kriterlerde değişiklik yok
- Tanımlamalar (***t-IFTA***) revize edilse de hala karmaşık

i-IFTA

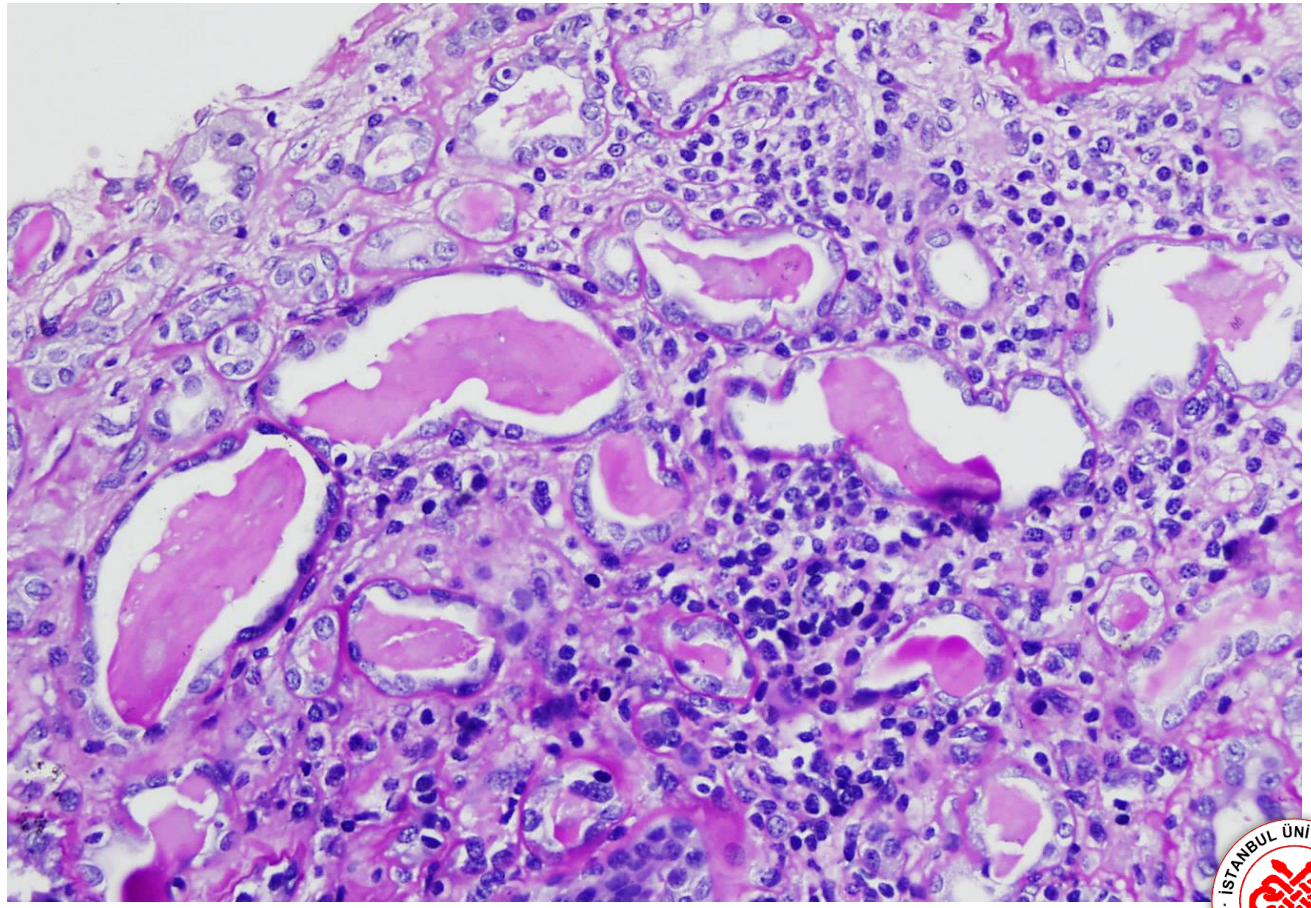


Mengel et al., Am J Transplant 2009; 9: 169–178

i-IFTA

- **Belirsizlikler devam ediyor:**
  - Non-immün nedenler: BKN, kronik pyelonefrit/obstrüksiyon, rekürren hastalık, donör hastalığı, CNIT, kontrolsüz HT
  - Nativ biyopsilerde de ci-ct alanlarında inflamasyon
  - Alloantijenlere spesifik yanıt değil
  - Kriter olarak kabul edilmesi ve tedavi edilmesine rağmen tedavi yanıtı olmayabilir
  - Aşırı immünsüpresyon komplikasyon riski

- **Moleküler olarak immünolojik hasar:**
  - *Halloran et al., Am J Transplant 2012;12:191*
  - *Modena et al., Am J Transplant 2016;16:1982*
  - *Naesens et al., Kidney Int; 80: 1364*



i-IFTA

ORIGINAL ARTICLE

T cell–mediated rejection is a major determinant of inflammation in scarred areas in kidney allografts

Carmen Lefaucheur[1,2] | Clément Gosset[3] | Marion Rabant[4] | Denis Viglietti[1,2] | Jérôme Verine[3] | Olivier Aubert[2] | Kevin Louis[2] | Denis Glotz[1,2] | Christophe Legendre[2,5] | Jean-Paul Duong Van Huyen[2,4] | Alexandre Loupy[2,5]

%78 öncesinde TCMR yok.

expression study in kidney allograft biopsies showing a similar TCMR molecular profile between biopsies with acute TCMR compared to biopsies with IF/TA and inflammation.[25]

We observed that the majority of patients (78%) with i-IF/TA at 1 year after transplantation did not experience previous acute TCMR proven by indication allograft biopsy. There may be 2 explanations for this lack of specificity of i-IF/TA for TCMR. First, i-IF/TA may be observed in other contexts,[26] such as BK virus–associated nephropathy, pyelonephritis, drug-induced interstitial nephritis, and posttransplant lymphoproliferative disorders, which can be recognized by clinical, biological, immunohistochemical, and specific histological findings (SV40-T staining and plasma BK virus load, uroculture, presence of eosinophil infiltrates and medication history, monotypic infiltrate, and imaging, respectively) and should be eliminated before considering an alloimmune process underlying i-IF/TA. This is also the case for most of the other elementary histologic lesions defined by the Banff classification (eg, glomerulitis is included in the antibody-mediated changes

AJT

ORIGINAL ARTICLE

Molecular phenotype of kidney transplant indication biopsies with inflammation in scarred areas

Philip F. Halloran[1,2] | Arthur Matas[3] | Bertram L. Kasiske[4] | Katelynn S. Madill-Thomsen[2] | Martina Mackova[1] | Konrad S. Famulski[1]

In kidney transplant biopsies, inflammation in areas of atrophy-fibrosis (i-IFTA) is associated with increased risk of failure, presumably because inflammation is evoked by recent parenchymal injury from rejection or other insults, but some cases also have rejection. The present study explored the frequency of rejection in i-IFTA, by using histology Banff 2015 and a microarray-based molecular diagnostic system (MMDx). In unselected indication biopsies (108 i-IFTA, 73 uninflamed IFTA [i0-IFTA], and 53 no IFTA), i-IFTA biopsies occurred later, showed more scarring, and had more antibody-mediated rejection (ABMR) based on histology (28%) and MMDx (45%). T cell–mediated rejection (TCMR) was infrequent in i-IFTA based on histology (8%) and MMDx (16%). Twelve i-IFTA biopsies (11%) had molecular TCMR not diagnosed by histology, although 6 were called borderline and almost all had histologic TCMR lesions. The prominent feature of i-IFTA biopsies was molecular injury (eg, acute kidney injury [AKI] transcripts). In multivariate analysis of biopsies >1 year posttransplant, the strongest associations with graft loss were AKI transcripts and histologic atrophy-scarring; i-IFTA was not significant when molecular AKI was included. We conclude that i-IFTA in indication biopsies reflects recent/ongoing parenchymal injury, often with concomitant ABMR but few with TCMR. Thus, the application of Banff i-IFTA in the population of late biopsies needs to be reconsidered.

KEYWORDS
basic (laboratory) research/science, biopsy, kidney transplantation/nephrology, rejection

Çoğunda histolojik (%28) ve MMDx (%45) olarak AMR var.

caTCMR

- Klinik önemi (?)
- Tedavi edelim mi (?)

AMR

Borderline/caTCMR

Aktivite ve kronisite indeksleri

MEETING REPORT

AJT

The Banff 2019 Kidney Meeting Report (I): Updates on and clarification of criteria for T cell– and antibody-mediated rejection

Raporlama; Aktivite ve Kronisite

Acute Banff scores	Grading (0, 1, 2, 3)	Chronic Banff scores	Grading (0, 1, 2, 3)	Acute & chronic Banff scores	Grading (0, 1, 2, 3)
i		ci		ti	
t		ct		i-IFTA	
v		cv		t-IFTA	
g		cg		pvl	
ptc		ptcml			
C4d					

Am J Transplant. 2020;20:2318–2331

Toward Activity and Chronicity Indices for the Evaluation of Kidney Transplant Rejection: A Viewpoint by the Banff Working Group

Maarten Naesens, MD, PhD,[1] Lynn D. Cornell, MD,[2] Surya V. Seshan, MD,[3] and Mark Haas, MD, PhD[4]

Vaulet et al.
J Am Soc Nephrol. 2021;32:1084–1096
J Am Soc Nephrol. 2022;33:2026–2039

Haas et al.
Kidney Int. 2023;103:187–195

TABLE 1.

Active and chronic Banff lesion scores and suggested composition of these indices to be considered in the calculation of activity and chronicity indices

Banff lesion scores	
Active Banff lesions	**Chronic Banff lesions**
• Interstitial inflammation (i) • Tubulitis (t) • Intimal arteritis (v) • Glomerulitis (g) • Peritubular capillaritis (ptc) • Acute thrombotic microangiopathy (TMA) • C4d deposition in peritubular capillaries (C4d)	• Interstitial fibrosis (ci) • Tubular atrophy (ct) • Vascular fibrous intimal thickening (cv) • Glomerular basement membrane double contours (cg) • Mesangial matrix expansion (mm) • Global glomerulosclerosis (gs) • Arteriolar hyalinosis (ah) • Peritubular capillary basement membrane multilayering (ptcml; needs electron microscopy)

Suggested composition of the activity and chronicity indices		
	Activity index[a]	**Chronicity index[a]**
Purpose	Global estimation of inflammation	Global estimation of chronicity
Formulation	i + t + v + g + ptc + C4d (0–2)[b]	ci + ct + cv + (cg × 2)
Range	0–17	0–15

[a]It is recognized that the activity and chronicity indices represent pure forms of active and chronic lesions, those most commonly seen in rejection. However, it is strongly suggested that all Banff lesions be scored and included in the biopsy report and comments regarding the significance of additional individual lesions (eg, TMA in active AMR) be included separately. The definitions of these lesion scores should follow the most recent version of the Reference Guide to the Banff Classification (https://banfffoundation.org/central-repository-for-banff-2019-resources-3/).
[b]In the activity index, C4d is dichotomized as present (score 2) or absent (score 0), following the definitions for C4d positivity in the Banff classification for AMR/MVI.
AMR, antibody-mediated rejection; MVI, microvascular inflammation; TCMR, T cell–mediated rejection; TMA, thrombotic microangiopathy.

Transplantation. 2025 Jan 28.
doi: 10.1097/TP.0000000000005336.

Banff tanımları ve süreçlerindeki zorluklar ve geliştirilmesi gereken noktalar

- Değişikliklerin etkileri tam olarak değerlendirilmeden uygulamaya alınması (bazı klinik olarak faydalı kategorilerin kaybı)
- Kullanıcı geri bildirimi eksikliği (sık yapılan değişiklikler ve yetersiz bilgilendirme, uygulama güçlüğü)
- Karmaşık kurallar (patoloji ve klinik arasında iletişim zorlukları, hasta yönetimine olumsuz etki)
- AMR tanımlarındaki değişkenlik, klinik çalışmalarda tutarsızlık ve sistematik incelemelerde zorluklar
- Yardımcı tekniklerin (EM, moleküler yöntemler) yaygın uygulanabilirliğinin olmaması kısıtlayıcı
- Dikotom kategorizasyonun hastalık sürecini yansıtmaması

Naesens M et al. Am J Transplant. 2024;24(3):338-349.
doi: 10.1016/j.ajt.2023.10.016.

Banff 2024 raporu

BANFF FOUNDATION FOR ALLOGRAFT PATHOLOGY

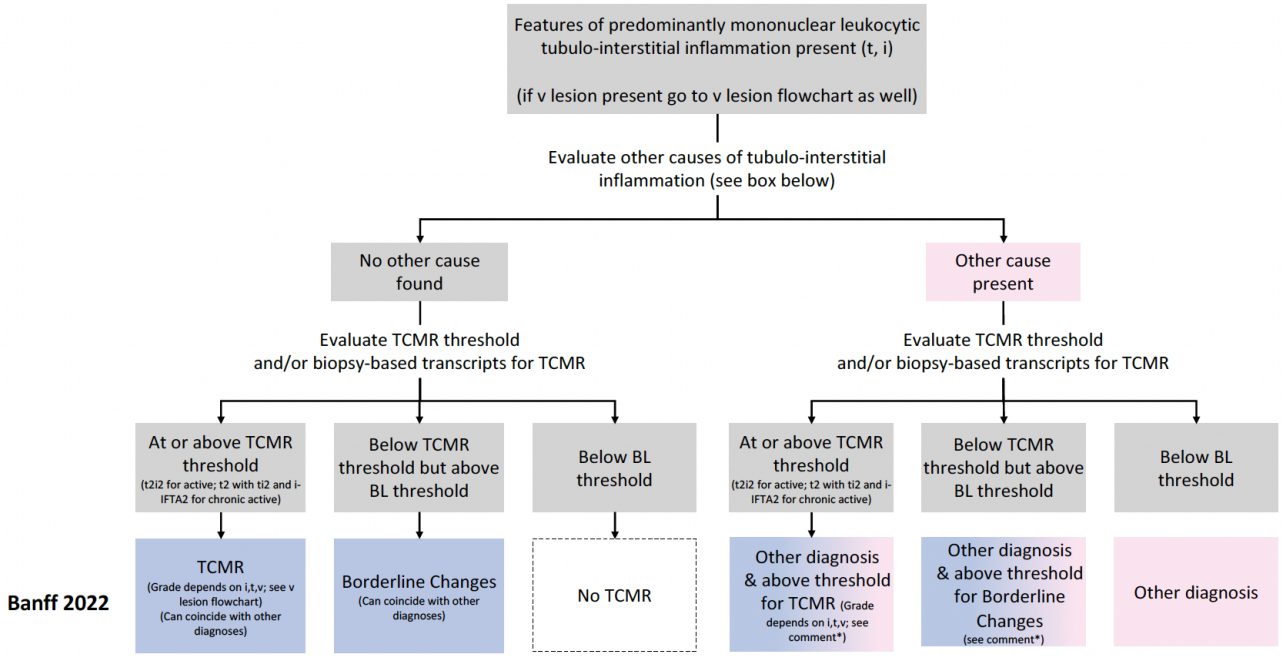
2024 Banff Meeting
September 16-20, 2024

ACADÉMIE NATIONALE DE MÉDECINE · PITOR · Université Paris Cité

- Mevcut sınıflama kurallarında değişiklik YOK !
- İmmünolojik süreçlerin artan karmaşıklığı !
- Banff 2022 ABMR akış şeması ✓, *i&t* ve *v* lezyonları için yeni akış şeması önerisi ➡
- *v* lezyonunun önemi ve izole *v* tartışması – Banff 2026
- Aktivite ve kronisite indekslerinin veri eksikliği nedeniyle sınıflamaya entegrasyonu ?
- Glomerüler hastalık raporlama önerileri
- Dijital ve moleküler araçların gelecekte sınıflamayı dönüştürme potansiyeli

https://amjtransplant.wixsite.com/ajtbanffblog/2022-banff-meeting-reports/kidney-2/2024-banff-meeting-report-kidney

Figure 1



Banff 2022

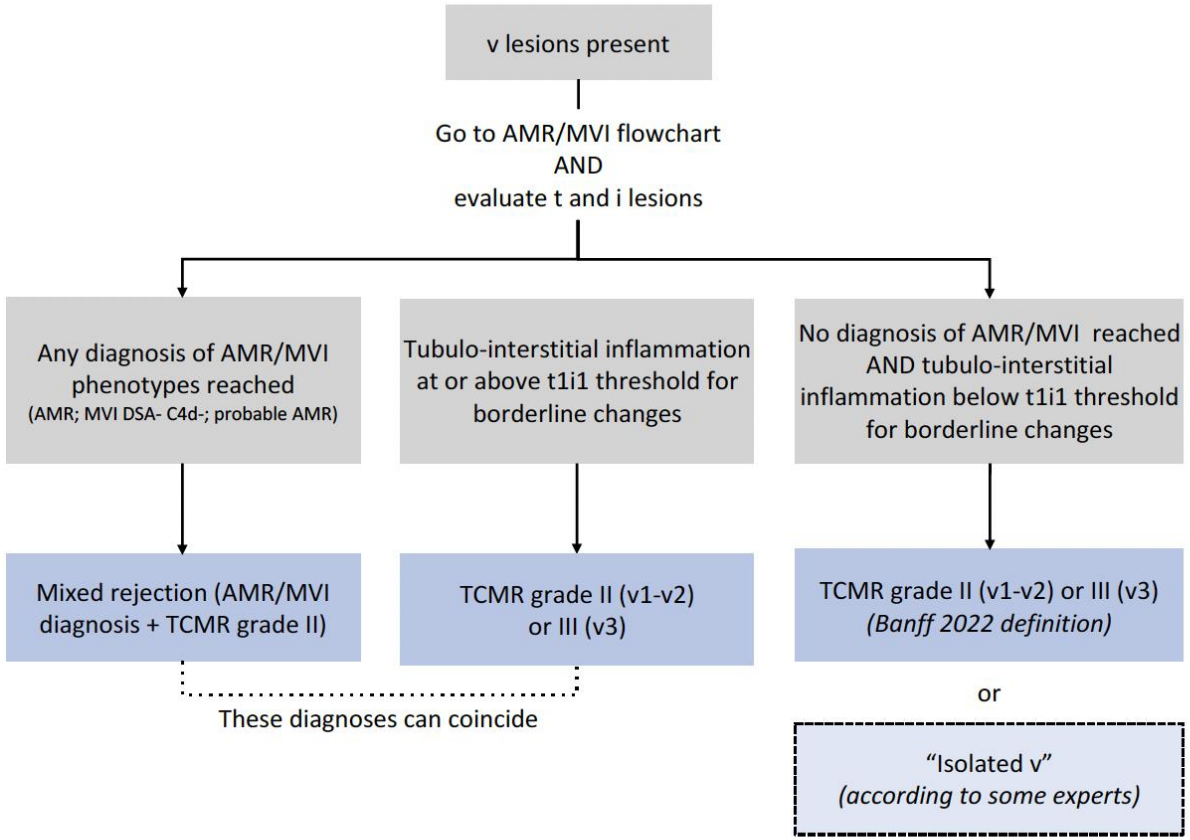
Non-exhaustive list of features potentially suggestive of alternative or concomitant causes of tubulo-interstitial inflammation, non-alloimmune:

- Histological features:
  - Suggestive of infectious disease: viral cytopathic changes; positive viral IHC (SV40, CMV, adeno etc); granulomas; neutrophil casts; abundant plasma cells
  - Suggestive of PTLD: monomorphic, plasma cell-rich or atypical lymphoid cells; architectural effacement; supportive IHC for B cell and T cell markers
  - Raises the possibility of drug reaction: abundant eosinophils
  - Suggestive of recurrent glomerular or tubulointerstitial disease: prior biopsy-proven native diagnosis; glomerular or tubular basement membrane immune complex deposition (IF/EM)
- Additional diagnostic features:
  - Polyomavirus and other viral PCR testing (CMV, adeno…)
  - Urological complications (reflux, obstruction, retention etc.)
  - Urine evaluation (leukocyturia, culture etc).
  - CRP testing,other clinical/laboratory features suggestive of infection or drug reaction….

*Comment: tubulo-interstitial infiltrate could be related to [other cause] but is also above threshold for T cell-mediated alloimmune process; consider treatment depending on clinical evolution/consider re-biopsy

https://amjtransplant.wixsite.com/ajtbanffblog/2022-banff-meeting-reports/kidney-2/2024-banff-meeting-report-kidney

Figure 2



Banff 2022

https://amjtransplant.wixsite.com/ajtbanffblog/2022-banff-meeting-reports/kidney-2/2024-banff-meeting-report-kidney

Banff 2024 raporu

BANFF FOUNDATION FOR ALLOGRAFT PATHOLOGY

2024 Banff Meeting
September 16-20, 2024

ACADÉMIE NATIONALE DE MÉDECINE · PITOR · Université Paris Cité

- Mevcut sınıflama kurallarında değişiklik YOK !
- İmmünolojik süreçlerin artan karmaşıklığı !
- Banff 2022 AMR akış şeması ✓, *i&t* ve *v* lezyonları için yeni akış şeması önerisi
- *v* lezyonunun önemi ve izole *v* tartışması – Banff 2026
- Aktivite ve kronisite indekslerinin veri eksikliği nedeniyle sınıflamaya entegrasyonu ?
- Glomerüler hastalık raporlama önerileri
- Dijital ve moleküler araçların gelecekte sınıflamayı dönüştürme potansiyeli

https://amjtransplant.wixsite.com/ajtbanffblog/2022-banff-meeting-reports/kidney-2/2024-banff-meeting-report-kidney

Özet

- Rejeksiyon tanı kriterleri aynı
- DSA mutlaka bakılmalı, tanının önemli bir bileşeni
- DSA ve C4d-neg MVI ve caTCMR klinik önemi tartışmalı
- Moleküler testlerin validasyonu
- Dijital ve yapay zeka araçlarının entegrasyonu
- Klinikopatolojik korelasyon

BANFF FOUNDATION
FOR ALLOGRAFT PATHOLOGY

Banff 2026
Oct. 5.-9.
Banff, Canada

ALBERTA
TRANSPLANT
INSTITUTE

SAVE THE DATE

Dinlediğiniz için teşekkürler...

yasozluk@istanbul.edu.tr

@yasozluk