22nd Annual Vanderbilt APP Symposium

The Last 5 Years of Cellular & Antibody-Mediated Rejection

Immunology, diagnostics, and therapeutics across kidney, heart, lung, liver, and pancreas transplantation

Yasmin Khan, MD
Associate Professor of Pediatrics, Division of Pediatric Allergy, Immunology and Pulmonary Medicine
Monroe Carrell Jr Children's Hospital at Vanderbilt

INTRODUCTION

Today's Objectives

1. **Review the immunology** of T-cell mediated rejection (TCMR) and antibody-mediated rejection (AMR) — the shared vocabulary for everything that follows.

2. **Summarize diagnostic advances** of the last 5 years: Banff reclassification, DSA-negative AMR, molecular diagnostics, and donor-derived cell-free DNA.

3. **Summarize therapeutic advances** — and failures — including anti-CD38 therapy, IL-6 blockade, complement inhibition, and imlifidase.

4. **Apply organ-specific and pediatric specific nuance** across kidney, heart, lung, liver, and pancreas transplantation.

01

Basic Immunology of Rejection

The shared biology behind T-cell and antibody-mediated rejection

BASIC IMMUNOLOGY

The Three-Signal Model of T-Cell Activation

Every major immunosuppressant blocks one of these three signals — the mechanistic backbone of the whole talk.

Signal 1

Antigen recognition

TCR/CD3 engages peptide-MHC on an antigen-presenting cell (donor or host)

Blocked by: calcineurin inhibitors (tacrolimus, cyclosporine)

Signal 2

Costimulation

CD80/CD86 on the APC engage CD28 on the T cell (plus CD40–CD40L)

Blocked by: belatacept (CTLA4-Ig)

Signal 3

Proliferation

IL-2 (and IL-15) signal through the PI3K/mTOR pathway to drive cell-cycle entry

Blocked by: mTOR inhibitors, IL-2R blockers

Allorecognition: Three Ways a T Cell "Sees" the Graft

Direct	Recipient T cell recognizes intact donor MHC displayed on a donor antigen-presenting cell (passenger leukocyte).	*Dominant early — drives early acute rejection; wanes as donor APCs are cleared*
Indirect	Recipient APC processes and presents donor alloantigen as peptide on self-MHC.	*Drives chronic rejection AND is required for B-cell/antibody help (T follicular helper cells)*
Semi-Direct	Recipient dendritic cell acquires intact donor MHC and presents to both pathways at once.	*Adds a layer of cross-regulation between the direct and indirect routes*

CD8 T cells bind MHC-I (all nucleated cells) and act as cytotoxic killers — CD4 T cells bind MHC-II (professional APCs) and provide help via cytokines and B-cell support.

TCMR: From Activated T Cell to Tissue Injury

1. Chemokine-guided homing across activated endothelium
2. Reactivation against APCs presenting antigen in the interstitium
3. IFN-γ release → the molecular hallmark of ALL rejection (CXCL9/10/11)
4. Direct cytotoxicity (perforin, granzyme, FasL) + macrophage activation

Histology Signature

Kidney: Tubulitis (T cells invade tubular epithelium) ± endarteritis/intimal arteritis

Heart: Lymphocytic myocyte injury and necrosis

Trend over time: TCMR becomes progressively rarer beyond 5–10 years post-transplant (clonal T-cell exhaustion) — unlike AMR, which can present decades out

BASIC IMMUNOLOGY

AMR: Building the Antibody Factory

A naïve B cell encounters donor antigen and, with T-follicular-helper support, differentiates three ways:

FAST BUT WEAK

Short-lived plasmablasts

Early, low-affinity antibody — transient

THE REAL TARGET

Long-lived plasma cells

Somatic hypermutation in the germinal center → high-affinity IgG produced indefinitely from BONE MARROW — without further T-cell help

THE RELAPSE RISK

Memory B cells

Rapid anamnestic response on re-exposure to donor antigen

Clinical implication: mature IgG comes from marrow plasma cells — this is why plasma-cell-directed therapy (bortezomib, anti-CD38) is rational, and why rituximab (targets CD20, which mature plasma cells lose) has disappointed as AMR monotherapy.

BASIC IMMUNOLOGY

How Antibody Actually Damages the Graft

Complement-Dependent

1. IgG/IgM binds donor endothelial antigen
2. C1q/r/s activation → C4 cleavage
3. C4d fragment binds tissue covalently and PERSISTS — the diagnostic "footprint" of antibody activity
4. C3 convertase → C3a/C5a recruit neutrophils, monocytes, mast cells
5. Membrane attack complex (C5b-9) → endothelial cell lysis

Complement-Independent

1. NK cells engage antibody-coated endothelium via Fc receptors (CD16)
2. Antibody-dependent cellular cytotoxicity (ADCC) using granzyme/perforin
3. Direct antibody–endothelial signaling promotes endothelial activation/proliferation
4. Explains C4d-NEGATIVE AMR — especially important in lung transplantation

Both pathways converge on IFN-γ-driven endothelial activation — the same final common pathway seen in TCMR, which is part of why "mixed rejection" exists.

TCMR vs. AMR at a Glance

Feature	TCMR	AMR
Effector	T cells, macrophages	DSA + complement + NK cells (ADCC)
Typical course	Early; becomes rare after 5–10 yrs (T-cell exhaustion)	Can present at ANY time — including decades out
Key histology	Tubulitis / interstitial inflammation (kidney); lymphocytic myocyte injury (heart)	Microvascular inflammation, capillaritis, C4d
Biomarker	Biopsy; IFN-γ-induced transcripts	DSA (MFI), C4d, dd-cfDNA, molecular transcripts
First-line treatment	Pulse steroids ± T-cell-depleting therapy	No FDA-approved therapy; PLEX/IVIG/rituximab ± newer agents
Frequency trend	Falling for 30 years (kidney: 30–40% → 6–11% at 1 yr)	Now the LEADING cause of late graft loss

02

The Last 5 Years: Diagnostics

Banff reclassification, DSA-negative AMR, molecular diagnostics, and dd-cfDNA

DIAGNOSTICS — THE BIG SHIFT

AMR Classification Has Gotten More Granular

Hyperacute ABMR Preformed antibody — now rare given crossmatch testing

Early acute ABMR (Type 1) In previously sensitized patients

ABMR independent of sensitization (Type 2) Newly recognized entity — arises without classic preformed sensitization

Late-stage ABMR (LABMR) Chronic; can present years to decades post-transplant

Mixed rejection TCMR + AMR features together — increasingly recognized as common, not the exception

Sub-threshold ABMR-like changes Histology suggestive but not meeting full criteria

DSA-NEGATIVE AMR

up to ~66%

of molecular/histologic AMR in one study occurred WITHOUT detectable circulating DSA

Clinical pearl: a negative DSA does NOT rule out AMR.

The Banff Classification Keeps Evolving

2016	Liver	AMR criteria formally introduced for liver — a decade-plus after kidney. “Probable chronic AMR” requires exclusion of alternates + compatible histology/fibrosis + recent HLA-DSA + focal C4d+.
2019	Kidney	Molecular diagnostics incorporated as adjunct; early “probable/suspicious” categories introduced.
2022	Kidney	Major reappraisal: microvascular inflammation ALONE no longer sufficient for AMR; new “probable AMR” category; ATI and new-onset arterial fibrosis removed as criteria; non-HLA antibodies excluded from formal DSA definition.
2022 / 2024	Pancreas	Refined TCMR/AMR/islet-pathology criteria; duodenal cuff biopsy formalized; chronic active TCMR and the “indeterminate” category further clarified in 2024.
2024	Kidney	Rejection reframed as a spectrum of phenotypes, not discrete boxes — emphasis on differential diagnostic reasoning integrating molecular data.

Eplet Matching: Beyond A / B / DR

The old way

Antigen-level HLA matching at A, B, and DR loci — a blunt instrument that treats each HLA molecule as a single unit of mismatch.

The refinement

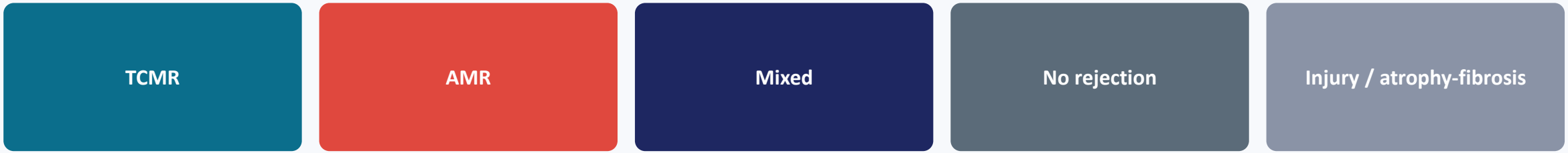
Eplets = the functional, antibody-binding subunits of an HLA epitope — a much finer-grained way to assess mismatch.

Class II eplet mismatch has been shown to be an independent — and in some studies STRONGER — risk factor for de novo DSA and AMR than conventional HLA mismatch counts.

DIAGNOSTICS

Molecular Diagnostics: The Molecular Microscope (MMDx)

Gene-expression / transcript-based analysis of biopsy tissue — detects rejection-associated IFN-γ-induced transcripts even when histology and C4d are ambiguous.



MMDx resolves biopsies into these molecular phenotypes — a second read alongside histology, not a replacement for it.

Where it stands, organ by organ

Kidney: most mature, largest validation base. **Heart:** expanding steadily alongside biopsy programs. **Lung:** newest entrant — commercial platforms extended to transbronchial/mucosal biopsies only in the last 1–2 years.

Donor-Derived Cell-Free DNA (dd-cfDNA)

A blood test measuring donor DNA fragments released from injured graft cells — rises with active rejection, particularly microvascular/AMR-type injury.

Kidney & Heart — furthest along

- Commercial assays: AlloSure, Prospera, AlloSure Heart / AlloMap Heartcare
- Validated for surveillance and biopsy triage
- Newer use: serial-TREND monitoring of treatment response, not just a single value
- Practical rule: rising/persistent dd-cfDNA + clinical concern → lower threshold for biopsy; falling trend during treatment is reassuring

Liver — promising, not yet primary-diagnostic

- dd-cfDNA rises BEFORE liver enzyme elevation and before clinical graft dysfunction
- Levels fall with successful rejection treatment
- NOT yet recommended as a stand-alone diagnostic tool — low positive predictive value, awaiting multicenter validation
- An honest “promising but not there yet” data point to share with patients

03

The Last 5 Years: Therapeutics

What's worked, what hasn't, and what's coming

STILL TRUE IN 2026

No therapy is FDA-approved specifically for antibody-mediated rejection — in ANY organ.

The last 5 years have been about testing mechanistically-targeted agents against the biology from Section 1 — with genuinely mixed results. Real-world treatment remains largely empiric: steroids, plasma exchange, IVIG, and rituximab, layered with newer agents as trial data matures.

Anti-CD38: Felzartamab

What it does

Anti-CD38 monoclonal antibody (also used as daratumumab in multiple myeloma) that depletes plasma cells and plasmablasts — targeting the actual long-lived antibody factories described earlier.

What the phase 2 RCT showed (NEJM, 2024)

Reduced molecular AND histologic AMR activity and DSA vs. placebo in late AMR — the first agent with a clear randomized mechanistic + histologic signal. Phase 3 programs now underway.

MOST PROMISING RECENT DATA POINT

Also named among promising novel heart-transplant immunosuppressants in the 2024 literature (as daratumumab).

THERAPEUTICS — A CAUTIONARY TALE

IL-6 Blockade: The IMAGINE Trial

IL-6 drives T-follicular-helper differentiation, plasmablast survival, and endothelial activation — a strong biologic rationale that looked promising in early open-label data.

-8.0

vs -5.2 mL/min/1.73m²

eGFR decline at 52 weeks (clazakizumab vs placebo) — not significant

28.3%

vs 22.2%

allograft loss / irreversible dysfunction — numerically WORSE on drug

Stopped early

after interim analysis

trial terminated — would not meet its primary endpoint

A strong biologic rationale plus encouraging phase 2 data does not guarantee phase 3 success. Tocilizumab (INTERCEPT trial) is still ongoing — don't assume the whole IL-6 pathway is dead — but IMAGINE is essential context for managing patient expectations about “the new drug I read about.”

Complement Inhibition & Imlifidase

Complement inhibition

Eculizumab (C5/terminal blockade) and C1-esterase inhibitor target the classical-pathway cascade described earlier.

More evidence in prevention/desensitization and catastrophic AMR than in established chronic AMR.

Imlifidase

An IgG-cleaving enzyme that inactivates ALL IgG subclasses within hours — a desensitization tool to cross a positive crossmatch, NOT a maintenance AMR treatment.

- 5-year outcome data show durable graft survival despite DSA rebound after cleavage
- Case report of successful use in acute LIVER AMR (2023)
- Named among heart transplantation's novel therapies as “IgG endopeptidase”

Belatacept & Regulatory T Cells: What's Next

Belatacept (costimulation blockade)

Blocks CD28–B7 (Signal 2) — therefore also reduces T-cell help to B cells. Primarily used for maintenance / CNI-sparing regimens, with growing interest as an AMR-prevention adjunct. Also named among novel heart-transplant immunosuppressants.

Regulatory T cells (Tregs)

A genuinely different strategy: harnessing natural immune tolerance rather than blunting the whole immune system. Injected Treg therapy has demonstrated SAFETY and potential in kidney transplant recipients (the ONE Study — 7 harmonized phase 1/2A trials).

A preview of cellular therapy as a future tolerance strategy — conceptually different from everything else in this section.

The Pipeline at a Glance

Agent / Class	Target	Where it stands (2026)
Felzartamab (anti-CD38)	Plasma cells / plasmablasts	Positive phase 2 RCT; phase 3 underway — most promising
Clazakizumab (IL-6)	IL-6 signaling	Phase 3 IMAGINE trial NEGATIVE, stopped early
Tocilizumab (IL-6R)	IL-6 signaling	INTERCEPT trial ongoing
Eculizumab / C1-INH	Complement cascade	Best evidence in prevention/desensitization, not chronic AMR
Imlifidase	Circulating IgG	Desensitization tool; not a maintenance therapy
Belatacept	T-cell costimulation (Signal 2)	Maintenance/CNI-sparing; AMR-prevention interest growing
Bortezomib	Proteasome / plasma cells	Largely disappointing in RCTs
Regulatory T cells	Immune tolerance	Phase 1/2A safety demonstrated — early days

04

Organ-Specific Rejection

Kidney, heart, lung, liver, and pancreas — what's different and what's new

ORGAN-SPECIFIC

Kidney: The Reference Organ

Most mature Banff system; every agent in the pipeline slide was tested here first.

30–40%	10–20%	6–11%
1990s	Early 2000s	By 2020

1-year acute rejection rate — steadily falling with better immunosuppression and monitoring, not disease disappearing

- **AMR now the bigger contributor to late graft loss** In one protocol-biopsy cohort, ALL rejection-related graft failures had AMR evidence by the time of failure; TCMR alone did not cause allograft loss.
- **Subclinical rejection is common early** Histology-positive, function-stable biopsies occur in 5–50% of cases in the first 3 months — stable creatinine doesn't fully exclude rejection.
- **DSA nuance matters** Complement-binding (C1q+) DSA carries worse prognosis than non-complement-binding DSA at the same MFI; de novo DSA risk rises with nonadherence, HLA-DRB1 mismatch, and delayed graft function.

ORGAN-SPECIFIC

Heart: pAMR Grading & Cardiac Allograft Vasculopathy

pAMR Grading Scale

0	1 (H+)	1 (I+)	2	3
Negative	Histopath only	Immunopath only	Both positive	Severe / combined

Cardiac Allograft Vasculopathy (CAV) — the chronic rejection analog

- A leading cause of death after year 1; ISHLT grades 0–III by angiographic stenosis + graft dysfunction
- Grafts are denervated — angina is rarely a symptom, so silent ischemia is a real risk
- Intravascular ultrasound (IVUS) improves diagnostic accuracy over angiography alone
- Biopsy remains the surveillance gold standard at regular intervals up to 5 years; MRI is being explored to reduce biopsy burden

ORGAN-SPECIFIC

Heart: Novel Therapies & Non-Invasive Monitoring

Novel immunosuppression — 2024 literature

Explicitly highlighted as "showing promising results":

- Daratumumab (anti-CD38)
- Belatacept
- IL-6-directed therapy
- IgG endopeptidase (imlifidase)

Liquid biopsy paradigm shift

• Cell-free DNA, gene-expression profiling, and microRNA-based "molecular microscopy" are reducing reliance on endomyocardial biopsy
• Omics (proteomics, metabolomics, pharmacogenomics) increasingly used for biomarker discovery

Horizon context: the first genetically-modified porcine-to-human cardiac xenotransplants (2022) specifically targeted overcoming hyperacute rejection through donor genetic modification — illustrating how far immune-engineering has advanced.

ORGAN-SPECIFIC

Lung: The Hardest Organ for AMR

C4d is frequently NEGATIVE in lung AMR — complement-independent Fc-receptor/NK-cell injury dominates here, so a negative C4d does not exclude the diagnosis.

- **No single validated gold standard** ISHLT consensus requires allograft dysfunction + DSA + compatible pathology + C4d + exclusion of other causes — but each element can be absent or ambiguous.
- **Clinical spectrum is wide** From fulminant AMR (severe graft failure, alveolar hemorrhage) to subclinical AMR (histology positive, no dysfunction — significance unclear).
- **Treatment is extrapolated, outcomes remain poor** Steroids, PLEX, IVIG, rituximab, bortezomib, eculizumab are used, but most patients progress to chronic lung allograft dysfunction (CLAD) despite treatment.
- **CLAD has two phenotypes** Bronchiolitis obliterans syndrome (BOS, obstructive) vs. restrictive allograft syndrome (RAS) — AMR is one of several contributors to either.

ORGAN-SPECIFIC

Liver: TCMR, the RAI, & Plasma-Cell-Rich Rejection

The liver is relatively tolerogenic (dual blood supply, large sinusoidal endothelial surface, soluble HLA that absorbs circulating antibody) — but rejection still happens.

Rejection Activity Index (RAI)

Each scored 0–3: portal inflammation, bile duct damage, venous endothelial inflammation (total 0–9)

Mild	RAI < 4
Moderate	RAI 4–6
Severe	RAI 7–9

Early TCMR (<90 days, more common): ductulitis, endotheliitis. Late TCMR (>90 days): interface/lobular hepatitis, plasma cell infiltrates — WORSE graft survival than early.

Plasma-Cell-Rich Rejection

An atypical, often LATE (>6 months) TCMR variant — histologically mimics autoimmune hepatitis (plasma cells >30% of infiltrate).

First described in pediatrics; now recognized in adults — especially PBC, PSC, ALD, cryptogenic cirrhosis. Also emerging AFTER hepatitis C antiviral therapy.

A distinct, often-missed entity that we should recognize when late liver-test abnormalities appear.

ORGAN-SPECIFIC

Liver: AMR & Two Genuinely New Risk Factors

AMR incidence in liver is unknown, likely lower than other organs, often subclinical. Diagnosis is complicated: concurrent TCMR can also elevate DSA/C4d, and C4d-negative AMR is well described here too.

Established risk factors: autoimmune liver disease (single greatest risk), young age, inadequate immunosuppression, prior rejection, female recipient/male donor, fewer HLA-DR matches, and nonadherence (biggest driver of LATE rejection specifically).

Two genuinely new (last ~5 years) findings to know:

1 COVID-19 vaccination

Rarely reported to trigger rejection in solid organ recipients — but the benefit of vaccination still clearly outweighs this small risk.

2 Checkpoint inhibitors (anti-PD-1)

Increase rejection RATE but were NOT associated with increased graft LOSS in a recent review — increasingly relevant as more recipients survive to need cancer immunotherapy.

ORGAN-SPECIFIC

Pancreas: Least-Studied, Most Reliant on Surrogates

- **Smallest evidence base** Diagnosis is hampered by biopsy difficulty and overlap with pancreatitis / technical complications.
- **Banff 2022 multidisciplinary report** Refined TCMR / AMR / islet-pathology criteria; formalized duodenal cuff biopsy as a technically easier surrogate site; reviewed noninvasive diagnostics (amylase/lipase trends, DSA).
- **Banff 2024 report** Further refined diagnosis of chronic active TCMR and reworked the “indeterminate” category using leukocyte immunostains.
- **Practical point** Pancreas surveillance leans more on enzyme trends + DSA + imaging alongside biopsy than any other organ in this talk.

05

Pediatric Considerations

Why pediatric rejection isn't just “adult rejection, smaller doses”

PEDIATRIC CONSIDERATIONS

Why Pediatric Immunology Is Genuinely Different

What's the same

Same Banff/ISHLT diagnostic criteria applied across all ages; same core drug classes (CNIs, antimetabolites, steroids, biologics); same TCMR/AMR mechanistic framework from Section 1.

What's genuinely different

At the extremes of pediatric age, immune maturity itself changes the biology — with consequences for what's possible, and what's necessary, that adult protocols never have to weigh.

ABO-INCOMPATIBLE HEART TRANSPLANTATION — POSSIBLE ONLY IN INFANTS

< 12–24 months

Infants in this age range haven't yet produced isohemagglutinins (anti-A/anti-B antibodies) or mature T-cell-independent antibody responses.

Result: ABO-incompatible infant heart transplant achieves graft/patient survival equivalent to ABO-compatible transplant — the same mismatch would cause hyperacute rejection in an older child or adult.

PEDIATRIC CONSIDERATIONS

EBV-Naive Status & the PTLD Balancing Act

Adults

~90–95%

already EBV-seropositive at time of transplant

Children

6× higher PTLD risk

Far more often EBV-naive — a D+/R- mismatch (EBV+ donor, EBV-naive recipient) carries roughly 6× the PTLD risk

The pediatric-specific tension

Reduction of immunosuppression is the cornerstone of PTLD management — but that directly conflicts with rejection prevention. This tradeoff exists in adults too, but is far sharper in pediatrics given how much more common EBV-naive status is. Enhanced EBV viral-load surveillance in D+/R- pediatric recipients, especially early post-transplant, is a mainstay of practice with little adult analog.

PEDIATRIC CONSIDERATIONS

The “High-Risk Age Window”: Adolescent Nonadherence

Rejection epidemiology in pediatrics has its own age-shaped curve — distinct from both younger children and adults transplanted later in life.

Younger children

Lower late-rejection risk — adherence largely managed by parents/caregivers

Adolescent / young-adult transition

The “high-risk age window” — a distinct spike in late acute rejection and graft loss, driven predominantly by nonadherence as autonomy increases

Adults transplanted later in life

Different risk profile — nonadherence still matters, but doesn't cluster the same way around a single age band

Practical point: this is essentially its own rejection-risk category, not just “adults but less compliant.” Transition-of-care programs and structured adherence screening are increasingly standard practice for this age band.

PEDIATRIC CONSIDERATIONS

Steroid Minimization for Growth — A Pediatric-Only Tradeoff

Why minimize

Chronic corticosteroid exposure impairs linear growth and pubertal development in children — a consideration with essentially no adult analog. Steroid-sparing and steroid-avoidance protocols have been developed specifically to protect growth trajectories in pediatric kidney transplant recipients.

The tradeoff

This directly shapes baseline maintenance immunosuppression — and therefore baseline rejection risk — in a way adult protocols don't need to weigh. Described in the literature as “a balancing act in progress”: steroid-minimizing regimens can preserve growth and reduce metabolic morbidity, but evidence on long-term rejection risk is still maturing and center practice varies widely.

PEDIATRIC CONSIDERATIONS — WHAT'S NEW

Sex Differences & Classification Limits (2025–2026)

Sex-based differences in rejection (2025, multi-organ pediatric cohort)

Male recipients had significantly lower rejection odds in liver (OR 0.85) and lung (OR 0.61) transplant; a smaller effect in kidney; no difference in heart. Raises the question of sex-informed immunosuppression dosing in children — not an established adult concept.

Classification limits (2026, 535 pediatric kidney recipients)

Traditional rejection categories (AMR vs. TCMR) did NOT predict long-term graft survival — a chronicity index (CI ≥4) did, predicting markedly worse 3-year eGFR (35.9 vs. 62.8 mL/min/1.73m^2). Suggests adult-derived Banff categories may need pediatric-specific validation.

06

Clinical Practice Takeaways

What this means for your day-to-day practice

CLINICAL TAKEAWAYS

What This Means for Your Practice

- **A negative DSA does not rule out AMR** Molecular/histologic AMR without detectable DSA is common — don't let a clean antibody panel close the door.
- **“Microvascular inflammation” ≠ automatic AMR** Since Banff 2022, read the full assigned category on the biopsy report, not just the MVI score.
- **DSA quality matters, not just presence** Complement-binding (C1q+) DSA carries worse prognosis than non-complement-binding DSA at the same MFI.
- **Use dd-cfDNA trends, not single values** Rising/persistent levels + clinical concern lower the threshold for biopsy; falling trend during treatment is reassuring — except liver, where it's not yet a stand-alone tool.
- **Set realistic expectations about “new drugs”** Felzartamab is genuinely promising; clazakizumab/IMAGINE shows phase 2 promise doesn't guarantee phase 3 benefit.
- **Know your organ's blind spots** Lung AMR is often C4d-negative; liver AMR/late-TCMR/plasma-cell-rich rejection are real and evolving; pancreas relies on surrogate markers.
- **Pediatrics plays by some different rules** Immune immaturity, EBV-naive status, adolescent nonadherence, and growth-driven steroid minimization all shift the risk calculus in ways adult protocols don't face.

REFERENCES

Selected References

- Tamargo CL, Kant S. Pathophysiology of Rejection in Kidney Transplantation. J Clin Med. 2023;12(12):4130.
- Chrysakis N, Magouliotis DE, Spiliopoulos K, et al. Heart Transplantation. J Clin Med. 2024;13(2):558.
- Maddur H, Wilson N, Patil P, Asrani S. Rejection in Liver Transplantation Recipients. J Clin Exp Hepatol. 2024;14(4):101363.
- Halloran PF, Einecke G, Sikosana MLN, Madill-Thomsen K. The Biology and Molecular Basis of Organ Transplant Rejection. In: Eisen HJ, ed. Pharmacology of Immunosuppression. Handbook of Experimental Pharmacology, vol 272. Springer; 2021.
- Banff 2022 & 2024 Kidney Meeting Reports; Banff 2022 & 2024 Pancreas Transplantation Reports (Am J Transplant).
- ISHLT Consensus Conference on Antibodies and AMR in Heart Transplantation, summary. J Heart Lung Transplant. 2025.
- Felzartamab Phase 2 RCT. N Engl J Med. 2024 (NEJMoa2400763). IMAGINE Phase 3 Clazakizumab Trial results, 2025–2026.
- Imlifidase 5-year outcomes and Delphi International Consensus on Imlifidase Desensitization, 2024.
- Antibody-Mediated Rejection in Lung Transplantation: 21st-Century Perspective. Transplant International. 2024.
- Translating B Cell Immunology to Antibody-Mediated Rejection Treatment. Nat Rev Nephrol. 2023.

Pediatric-specific literature

- Sex-Based Differences in Rejection Among Pediatric Solid Organ Transplant Recipients. Pediatr Transplant. 2025.
- Prognostic impact of rejection and chronicity index on long-term graft outcomes in pediatric kidney transplant recipients. Front Immunol. 2026.
- ABO-incompatible heart transplantation in infants — immunologic basis and outcomes (multiple sources incl. NEJM 2001; JHLT 2024).
- Post-transplant lymphoproliferative disease after pediatric kidney transplant. (PMC review.)
- Steroids in Pediatric Kidney Transplantation: A Balancing Act in Progress. Am J Transplant.
- Age-specific risk of renal graft loss from late acute rejection or non-compliance in the adolescent and young adult period.

Thank You

Questions & Discussion