BMT MASTERCLASS

ORGANISED BY ISBMT 2025

13th & 14th December 2025

# Engraftment, Graft Failure, and Poor Graft function

## 13/12/2025

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Transplantation and Cellular Therapy 27 (2021) 642–649

ELSEVIER

Transplantation and Cellular Therapy

journal homepage: www.tctjournal.org

ASTCT
American Society for Transplantation and Cellular Therapy

Guideline

# Standardizing Definitions of Hematopoietic Recovery, Graft Rejection, Graft Failure, Poor Graft Function, and Donor Chimerism in Allogeneic Hematopoietic Cell Transplantation: A Report on Behalf of the American Society for Transplantation and Cellular Therapy

# Engraftment

**1. Neutrophil Recovery (Engraftment)**

Defined as:

**“The first of 3 successive days with an absolute neutrophil count (ANC) ≥ 500/mL after post-transplantation nadir.”**

**2. Platelet Recovery (Engraftment)**

Defined as:

**“The first of 3 consecutive days with a platelet count ≥ 20,000/mL in the absence of platelet transfusion for 7 consecutive days.”**

Kharfan-Dabaja MA, et al. . Standardizing Definitions of Hematopoietic Recovery, Graft Rejection, Graft Failure, Poor Graft Function, and Donor Chimerism in Allogeneic Hematopoietic Cell Transplantation: A Report on Behalf of the American Society for Transplantation and Cellular Therapy. Transplant Cell Ther. 2021 Aug;27(8):642-649. doi: 10.1016/j.jtct.2021.04.007. PMID: 34304802.

# Delayed engraftment

**Delayed engraftment** refers to **failure to achieve neutrophil or platelet recovery by the expected time frame for the specific graft source**, *without yet meeting criteria for primary graft failure*.
It represents a **prolonged period of cytopenias before the formal cutoff for graft failure is reached**.

No consensus was reached by either pane

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# Clinically Used Working Definitions

**Delayed Neutrophil Engraftment**
Failure to achieve
**ANC ≥ 0.5 × $10^9$/L by:**
**Day +21** after PBSC transplant
**Day +28** after bone marrow transplant
**Day +35 to +42** after cord blood transplant
*(These timeframes reflect expected median engraftment kinetics.)*

**Delayed Platelet Engraftment**
Failure to achieve
**platelet count ≥ 20 × $10^9$/L unsupported**
by:
**Day +28 to +35** for PBSC/BM
**Day +42 to +60** for cord blood

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# 1. Primary Graft Failure

Primary graft failure = **failure to achieve ANC ≥ 500/mL (≥0.5 × $10^9$/L) by a defined day**, with **associated pancytopenia**, confirmed with donor chimerism testing.

**a. For PBSC or Unstimulated Bone Marrow Grafts**

**"Graft failure was defined as lack of achievement of an ANC ≥ 500/mL by day +30 with associated pancytopenia."**

**b. For Umbilical Cord Blood (UCB) Grafts**

**"Graft failure was defined as lack of achievement of an ANC ≥ 500/mL by day +42 with associated pancytopenia."**

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# 1. Secondary Graft Failure

Secondary graft failure = **loss of previously engrafted donor hematopoiesis**.
ASTCT definition:

**“Secondary graft failure was defined as a decline in hematopoietic function (may involve hemoglobin and/or platelets and/or neutrophils) necessitating blood products or growth factor support, after having met the standard definition of hematopoietic (neutrophil and platelet) recovery.”**

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# Graft Rejection

**Graft rejection** is an **immune-mediated host-versus-graft response** in which the recipient's immune system eliminates donor hematopoietic stem cells, leading to loss of donor chimerism and failure of donor-derived hematopoiesis.

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# Graft Failure

**Graft failure** is the **inability of the donor graft to establish or sustain hematopoiesis**, due to immune or non-immune causes (e.g., inadequate stem cell dose, infections, stromal injury, conditioning toxicity), resulting in persistent cytopenias with low or absent donor chimerism.

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# Aren’t these the same

**Conceptually important**, **clinically meaningful**, and **therapeutically different**, *even though the outward clinical picture may look similar initially*.

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# When does graft rejection usually lead to autologous recovery?

Autologous recovery is more likely when:

**Conditioning is not fully myeloablative**
RIC/NMA regimens
ATG-heavy regimens
PTCy-based haploidentical regimens

**Primary diseases with some residual stem cells**
SAA
Fanconi anemia
**Strong host immune competence**
Young patient
DSA presence
Low conditioning intensity

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# When does graft rejection *not* lead to autologous recovery?

Autologous recovery is unlikely or impossible when:

**A. Conditioning is fully myeloablative**
Busulfan/Cy, TBI/Cy, Melphalan-based regimens
→ Host stem cells eliminated.

**B. Marrow is diseased or fibrotic**
Examples:
Myelofibrosis
MDS/AML with high blast burden
Aplastic anemia with extremely hypocellular marrow

**C. Conditioning toxicity destroyed residual host stem cells**
Chronic viral infections or iron overload also worsen this

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| Term | Mechanism | Treatment Implication |
|---|---|---|
| **Graft rejection** | Immune-mediated host-versus-graft attack | Intensify immunosuppression, consider DSA-directed therapy, second transplant with more immunosuppression |
| **Graft failure** | Multifactorial: cell dose, infection, drugs, stromal issues, conditioning toxicity | Adjust drugs, treat infection, consider CD34+ stem cell boost |

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# Donor Chimerism

**Donor chimerism** is the proportion of hematopoietic cells in the recipient that are derived from the donor, measured using lineage-specific assays (such as CD3 for lymphoid and CD33 for myeloid cells), and expressed as a percentage of donor versus host cells.

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# Categories of Donor Chimerism

**Full donor chimerism:** >95% donor cells
**Mixed (partial) chimerism:** 5%–95% donor cells
**Absent donor chimerism:** <5% donor cells.

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# What is Poor Graft Function?

**Poor graft function (PGF)** is the presence of **persistent multilineage cytopenias** despite **full donor chimerism**, with no evidence of relapse, infection, drug toxicity, or other identifiable causes, leading to continued dependence on transfusions and/or growth factor support.

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| Concept | Definition | Key Features | Chimerism Pattern | Term to Use |
|---|---|---|---|---|
| **Graft Rejection** | Immune-mediated destruction of donor graft | HVG immune attack, rapid aplasia | Donor <5%; host recovery | **Immune-mediated graft rejection** |
| **Primary Graft Failure** | No initial engraftment | No ANC recovery by day +30/+42 | Donor low/absent | **Primary graft failure** |
| **Secondary Graft Failure** | Loss of prior engraftment | Falling counts after initial recovery | Falling donor chimerism | **Secondary graft failure** |
| **Poor Graft Function** | Cytopenias despite engraftment | Normal donor chimerism | Donor >95% | **Poor graft function** |

# What is  Split Chimerism?

**Split chimerism** refers to evaluating donor-versus-recipient chimerism **separately in different hematopoietic lineages**—most commonly **myeloid ($CD33^+$)** and **lymphoid ($CD3^+$)** cells—rather than relying on total (unsorted) chimerism.

# What is  consensus recommendation?

**Routine measurement of donor chimerism using CD3 (lymphoid) and CD33 (myeloid) sorted cells is recommended.”**

**“Use the actual percentages of donor myeloid cells and lymphoid cells, together with blood counts and clinical status, for management decisions.”**

# Why Split Chimerism Is Clinically Important?

**Detects Early Graft Failure vs Immune Escape**

**Falling myeloid (CD33) chimerism** → suggests impending **graft failure** or relapse in myeloid diseases.

**Falling lymphoid (CD3) chimerism** → suggests **immune-mediated graft rejection** or insufficient immune reconstitution.

# Interventions

| Pattern | Likely Problem | Action |
|---|---|---|
| Declining **CD3** | Host T-cell recovery → immune rejection | Withdraw immunosuppression ± DLI |
| Declining **CD33** | Myeloid failure or relapse | Consider stem cell boost; evaluate for relapse |
| Declining both | Global graft failure | Evaluate for second transplant |

Falling **myeloid lineage** chimerism may indicate recurrence of a **myeloid malignancy** even before blood counts change.
Often precedes morphologic relapse by weeks.

# Interventions

**PGF** = full donor chimerism with cytopenias
**Failure** = declining donor chimerism + cytopenias
Split chimerism is essential to make this distinction.

Example:
Cytopenia with CD33 98%, CD3 95% → **Poor Graft Function**

Cytopenia with CD33 30%, CD3 70% → **Mixed chimerism → evolving graft failure**

**Special Role in Non-Malignant Disorders – Sickle cell diseases**

The ASTCT panel highlights:

**“The level of donor chimerism required for disease correction depends on the disease; split chimerism may show adequate myeloid correction even if CD3 chimerism remains mixed.”**

Examples:
**Sickle cell disease**: ~20–30% **myeloid** donor chimerism may be curative.
**Aplastic anemia**: Both CD3 and CD33 are important; early CD3 declines indicate rejection.

# Quiz time

# Thought bubble

**New Resident in a New Institution**

**Description:**
The resident has joined the system; conditioning (counselling) has been completed.

**What do you call a resident who is smoothly and seamlessly integrated into the system?**

**Optimally engrafted Resident**

What do you call a resident who has early friction after joining with nurses and peers, but responsive to discussions, reassurance, mentorship, and clear protocols?

**Acute GVHD Resident.**
**Prognosis:** Excellent. Often becomes a model graft after the first ward round meltdown.

What do you call a resident with persistent maladaptation, selective participation in work, and long-standing interpersonal issues—functional but difficult?

**Chronic GVHD Resident**
Requires structured supervision and clear boundaries.

What do you call a resident who leaves immediately after taking the seat within the first couple of weeks, because they did not like the environment?

**Primary graft failure Resident**
Never engrafted. Counts never came up.

What do you call a resident who has joined for months and decided to leave as he/she thought this is not the specialty they envisaged?

**Secondary graft failure Resident**

**Prognosis:** Emotionally more painful than primary failure—for both graft and transplant team.

What do you call a  resident who left because the environment was toxic even though he/she liked branch?

**Graft rejected resident**. The graft was fine. The host was hostile.

What do you call a  resident who has partial assimilation who may *stabilize—or drift towards optimal integration or may leave the course*

**Mixed Chimera Resident.**

Unpredictable. Requires close chimerism monitoring.

What do you call a resident who continues to be in the course but work output is suboptimal?

**Poor graft function resident** –
Present on duty roster. Absent in productivity. Needs daily transfusions of motivation.
Resource-intensive. Multidisciplinary review advised.

# CASE 1

| Case | Details |
|---|---|
| **Patient** | 42-year-old male, AML in CR1 |
| **Day Post-HSCT** | +28 |
| **Graft Type** | MUD PBSC |
| **Clinical Findings** | Persistent ANC 0.1 ×$10^9$/L, marrow 10% cellular |
| **Split Chimerism** | CD33: **32% donor** (falling) → CD3: **75% donor** |

## What is your interpretation?

| | |
|---|---|
| **Interpretation** | **Early/evolving primary graft failure** (myeloid lineage failing) |
| **Key Indicators** | Falling CD33, persistent aplasia, no infection/drug cause |

## What should be the ideal intervention?

| | |
|---|---|
| **Intervention** | - Stop myelosuppressive drugs - DSA evaluation - **CD34+ stem cell boost** - Plan for second HSCT if needed |
| **Learning Point** | Falling **myeloid chimerism** + aplasia = **graft failure** |

# CASE 2

| Case | Details |
|---|---|
| **Patient** | 19-year-old female, Aplastic Anemia |
| **Day Post-HSCT** | +30 |
| **Graft Type** | Haploidentical (PTCy) |
| **Split Chimerism** | CD3: **18% donor** (falling)CD33: **85% donor** |
| **Clinical Findings** | ANC 0.4 ×$10^9$/L, marrow hypocellular |

## What is your interpretation?

| | |
|---|---|
| **Interpretation** | **Immune-mediated graft rejection** (host T-cell recovery) |
| **Key Indicators** | Isolated CD3 decline ~ host T-cell recovery |

## What should be the ideal intervention?

| | |
|---|---|
| **Intervention** | - **Rapid CNI taper** - **DLI** if no GVHD - Short steroids if immune flare - Plan second HSCT if continues |
| **Learning Point** | Falling **CD3 donor fraction** = **rejection** |

# CASE 3

| Case | Details |
|---|---|
| **Patient** | 54-year-old, MDS |
| **Day Post-HSCT** | +60 |
| **Graft Type** | Matched sibling |
| **Clinical Findings** | Hb 7.2, ANC 0.9, Plt 10 (transfusion-dependent) |
| **Split Chimerism** | CD33: **98% donor**CD3: **97% donor** |
| **Other Findings** | No infections, no relapse, ferritin high |

What is your interpretation?

| | |
|---|---|
| **Interpretation** | **Poor Graft Function** (full donor chimerism + cytopenias) |
| **Key Indicators** | Full donor chimerism rules out failure/rejection |

What should be the ideal intervention?

| | |
|---|---|
| **Intervention** | - Start **eltrombopag** - Reduce CNI - Manage iron overload - **CD34+ boost** if no improvement |
| **Learning Point** | Full donor chimerism + cytopenias = **PGF**, not failure |

# CASE 4

| Case | Details |
|---|---|
| **Patient** | 12-year-old, Sickle Cell Disease |
| **Day Post-HSCT** | +100 |
| **Graft Type** | MSD Bone Marrow |
| **Split Chimerism** | CD33: **28% donor**CD3: **12% donor** |
| **Clinical Findings** | No VOC, HbS ↓ from 90% → 32% |

What is your interpretation?

| Interpretation | **Stable mixed myeloid chimerism is curative in SCD** |
|---|---|
| **Key Indicators** | Myeloid donor ≥20–30% → disease correction |

What should be the ideal intervention?

| Intervention | - Observation only - Avoid unnecessary IST taper |
|---|---|
| **Learning Point** | **Mixed myeloid chimerism** is therapeutic in some non-malignant diseases |

# CASE-5

| Case | Details |
|---|---|
| **Patient** | 48-year-old, AML FLT3-ITD |
| **Day Post-HSCT** | +90 |
| **Graft Type** | MUD PBSC |
| **Split Chimerism** | CD33: **65 → 48 → 30% donor**CD3: **95% donor** |
| **Bone Marrow** | 10% blasts |

What is your interpretation?

| | |
|---|---|
| **Interpretation** | **Relapse**, not graft failure |
| **Key Indicators** | Falling CD33 + blasts = relapse |

What should be the ideal intervention?

| | |
|---|---|
| **Intervention** | - Stop tacrolimus - **DLI** - Start **gilteritinib** - Plan second HSCT in CR2 |
| **Learning Point** | Low CD33 + blasts = **relapse**; low CD33 + empty marrow = **graft failure** |

# CASE 6

| Case | Details |
|---|---|
| **Patient** | 60-year-old, AML |
| **Day Post-HSCT** | +60 |
| **Graft Type** | Haploidentical PTCy |
| **Split Chimerism** | CD3: **30% donor** (expected)CD33: **98% donor** |
| **Clinical Findings** | Counts recovered, no infection/GVHD |

What is your interpretation?

| | |
|---|---|
| **Interpretation** | **Expected slow CD3 recovery** after PTCy, not rejection |
| **Key Indicators** | Normal post-PTCy chimerism pattern |

What should be the ideal intervention?

| | |
|---|---|
| **Intervention** | - Continue slow IST taper - Monitor monthly |
| **Learning Point** | Low CD3 early after PTCy is **physiologic**, not pathologic |

# CASE 7: Haploidentical HSCT with PTCy – Serial Split Chimerism & Intervention

| Parameter | Day +30 | Day +60 | Day +90 | Day +120 |
|---|---|---|---|---|
| **ANC ($\times 10^9$/L)** | 0.9 | 1.4 | 1.2 | 0.6 |
| **Platelets ($\times 10^9$/L)** | 45 | 72 | 60 | 32 |
| **Hb (g/dL)** | 8.6 | 9.4 | 9.2 | 8.1 |
| **CD33 donor chimerism (%)** | 96 | 92 | 78 | 52 |
| **CD3 donor chimerism (%)** | 28 | 55 | 72 | 70 |
| **Clinical status** | Counts just recovering | Doing well, ambulant | Mild fatigue, more transfusions | Increasing transfusion needs, borderline neutropenia |
| **Marrow blasts** | 0% | 0% | 0% | 0% |
| **IST (Tacrolimu** | | | | |

**Expected pattern post-PTCy** (slow T-cell, good myeloid engraftment)
Continue full IST, routine monitoring of CD3/CD33 chimerism and counts.

**Physiological immune reconstitution; no evidence of rejection or failure.**
Begin gradual taper of tacrolimus as per protocol; no DLI indicated

Early signal of **possible evolving myeloid graft dysfunction** vs drug/infection effect
1) Re-check chimerism in 2–3 weeks
2) Screen for viral infections (CMV/HHV-6) 3) Review myelotoxic drugs; hold if possible.

# What should be done by day 120

CD33 falls further to 52% (clear trend), CD3 stable ~70%, increasing transfusion need, ANC 0.6

**Evolving secondary graft failure** (myeloid) with preserved T-cell engraftment; no blasts, so relapse unlikely

1) Stop/avoid myelotoxic drugs
2) Plan $CD34^+$ stem cell boost from same donor (no conditioning)
3) Consider modest IST reduction (if no GVHD) to enhance graft function
4) If CD33 continues to fall or counts crash → discuss second HSCT in MDT.

# Table 2 – Interpretation by Time Point

| Time Point | Key Observations | Interpretation | Planned / Probable Intervention |
|---|---|---|---|
| **Day +30** | High CD33 (96%), low CD3 (28%), neutrophils just recovering | **Expected pattern post-PTCy** (slow T-cell, good myeloid engraftment) | Continue full IST, routine monitoring of CD3/CD33 chimerism and counts. |
| **Day +60** | CD33 92% (stable), CD3 55% (rising), counts improving | **Physiological immune reconstitution**; no evidence of rejection or failure | Begin **gradual taper of tacrolimus** as per protocol; no DLI indicated. |
| **Day +90** | CD33 down to 78%, CD3 up to 72%, mild drop in platelets | Early signal of **possible evolving myeloid graft dysfunction** vs drug/infection effect | 1) Re-check chimerism in 2–3 weeks 2) Screen for viral infections (CMV/HHV-6) 3) Review myelotoxic drugs; hold if possible. |
| **Day +120** | CD33 falls further to 52% (clear trend), CD3 stable ~70%, increasing transfusion need, ANC 0.6 | **Evolving secondary graft failure (myeloid)** with preserved T-cell engraftment; no blasts, so relapse unlikely | 1) Stop/avoid myelotoxic drugs 2) **Plan $CD34^+$ stem cell boost** from same donor (no conditioning) 3) Consider modest IST reduction (if no GVHD) to enhance graft function 4) If CD33 continues to fall or counts crash → discuss **second HSCT** in MDT. |

# Learning points

Early **low CD3 with high CD33** after haplo + PTCy is **expected**, not rejection.

**Trend analysis** is crucial: a *single* CD33 value of 78% may be acceptable, but a **serial fall 96 → 92 → 78 → 52%** with worsening cytopenias signals **evolving secondary graft failure**.

Preserved CD3 donor chimerism (70%) argues **against immune rejection**; this guides you toward **$CD34^+$ stem cell boost** rather than DLI ± aggressive IST taper.

# Thank you