

THE BIG PICTURE

CAR T-cell toxicities can move fast. Recognition must move faster.

A concise guide to recognizing and managing key toxicities after CAR T-cell therapy

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The One Thing to Know



Start with the pattern—not the label



CRS: fever is the starting signal



CRS grading at a glance



ICANS: small neurologic changes matter



ICE score: 10 points, five tasks



Other toxicities can still be high stakes



Infection can mimic—and compound—toxicity



The outpatient response



Quick Myth Check

① The One Thing to Know



THE ONE THING TO KNOW

A new fever or neurologic change is never “just a symptom.”

After chimeric antigen receptor (CAR) T-cell therapy, fever may signal cytokine release syndrome (CRS), infection, or both. Confusion, language or writing changes, tremor, weakness, somnolence, or seizure may signal immune effector cell-associated neurotoxicity syndrome (ICANS). Both demand immediate assessment.

Why it matters

Cytokine release syndrome (CRS) and immune effector cell-associated neurotoxicity syndrome (ICANS) are often reversible with vigilant monitoring, supportive care, and prompt treatment, but both can become severe or fatal. Early recognition and intervention are central to reducing morbidity and preventing avoidable deterioration.¹

Think in two time horizons

EARLY: HOURS TO WEEKS	CRS, ICANS, infection, immune effector cell-associated hemophagocytic lymphohistiocytosis-like syndrome (IEC-HS), coagulopathy, organ dysfunction, and acute cytopenias.
LATER: WEEKS TO YEARS	Prolonged or recurrent cytopenias, humoral immune dysfunction/hypogammaglobulinemia, recurrent infection, delayed product-specific neurologic or gastrointestinal toxicities, and secondary malignancies.

In 20 seconds

- Check vital signs and neurologic status.
- Use the applicable grading framework and reassess as the clinical picture evolves.
- Evaluate competing diagnoses—especially infection.
- Start supportive and syndrome-directed care without avoidable delay.
- Escalate to inpatient or intensive care when severity or trajectory demands it.



Start with the pattern —not the label



FIRST MOVE

Stabilize. Assess. Differentiate. Treat.

Do not wait for diagnostic certainty when the patient is deteriorating. Address airway, breathing, circulation, oxygenation, hemodynamics, access, and seizure safety while the team evaluates CRS, ICANS, infection, and other causes.

The rapid assessment

1. IDENTIFY	CAR T-cell product, infusion date/day, diagnosis, recent CRS/ICANS, recent tocilizumab or corticosteroids, neutropenia, and current medications.
2. MEASURE	Temperature, blood pressure, pulse, oxygen saturation, respiratory status, level of consciousness, and full neurologic/immune effector cell-associated encephalopathy (ICE) assessment when applicable.
3. LOOK FOR OVERLAP	CRS and infection can coexist. ICANS and CRS can coexist. Medication effects, metabolic abnormalities, stroke, seizure, and disease progression may mimic toxicity.
4. GRADE	Use the institution-approved grading framework—commonly the American Society for Transplantation and Cellular Therapy (ASTCT) criteria for CRS and ICANS—and follow applicable product-label and protocol requirements. Re-grade whenever clinical status changes.
5. ACT	Begin supportive care and the institution-approved toxicity pathway. Do not delay higher-level care while waiting for all tests.
6. REASSESS	Trend vital signs, grade, neurologic findings, organ function, response to treatment, and disposition needs.

Always ask

- Is the patient unstable now?
- Could this be infection or sepsis?
- Is there concurrent neurotoxicity?
- Has the syndrome progressed despite initial care?
- Is the current setting still safe?

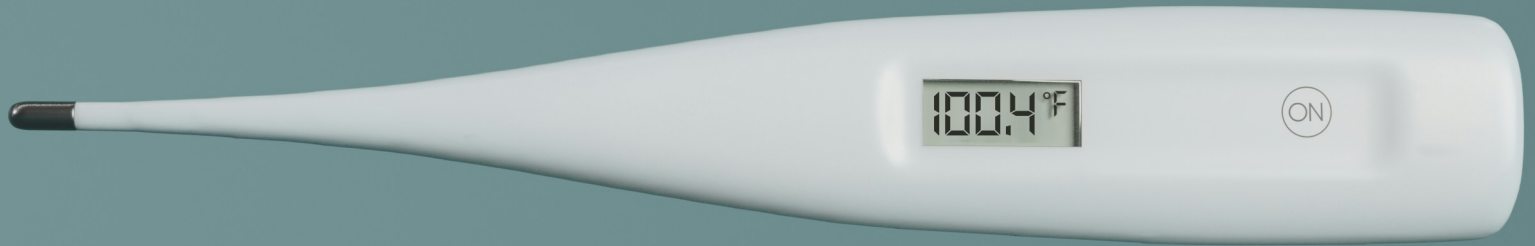
OUTPATIENT REALITY

The escalation pathway is part of the treatment.

A prompt admission is not a failure. Delayed recognition, delayed treatment, a failed handoff, or an unsafe transfer are the failures to prevent.



CRS: fever is the starting signal



RECOGNIZE

Suspect CRS when fever of 38°C or higher occurs after CAR T-cell infusion while evaluating infection and other causes in parallel. Under ASTCT criteria, fever at CRS onset must not be attributable to another cause; hypotension and hypoxia determine subsequent severity.

CRS severity is driven by the support required for hypotension and hypoxia, not by fever height alone. Infection and other causes still require evaluation.

What it can look like

- Fever, chills, fatigue, myalgias, malaise
- Tachycardia, hypotension, dizziness, syncope
- Hypoxia, dyspnea, increasing oxygen requirement
- Organ dysfunction, capillary leak, coagulopathy, or rapid deterioration

What to do

- **Support first:** Oxygen, intravenous access, fluids and hemodynamic support, diagnostics, and antimicrobials when indicated.
- **Grade repeatedly:** The patient can progress quickly; disposition should follow the current grade and trajectory.
- **Treat by grade and trajectory:** Tocilizumab, corticosteroids, or both may be indicated based on severity, persistence or progression, product labeling, and the institution-approved pathway.
- **Escalate early:** Use explicit admission and intensive care unit (ICU) criteria; match transport and monitoring to acuity.

Supportive care is foundational for most early CAR T-cell toxicities. Persistent, progressive, or severe CRS may require tocilizumab, corticosteroids, and higher-level supportive care according to current product labeling and the institution-approved toxicity algorithm.¹

Do not miss

- Neutropenic fever or sepsis
- Cardiac or pulmonary complications
- Bleeding or coagulopathy
- Concurrent ICANS
- Hyperinflammatory syndrome when fever and organ dysfunction persist or recur

CLINICAL PEARL

Tocilizumab treats CRS—not isolated ICANS.

If CRS and ICANS occur together, address each syndrome with the appropriate pathway. Repeated tocilizumab does not substitute for neurotoxicity treatment.



CRS grading at a glance



HOW TO GRADE

Use the more severe finding: hypotension or hypoxia.

Fever of 38°C or higher starts the syndrome when not attributable to another cause. Once CRS is established and antipyretic or anticytokine therapy has been administered, fever is no longer required to grade subsequent CRS severity.

Parameter	Grade 1	Grade 2	Grade 3	Grade 4
Fever	≥38°C	≥38°C	≥38°C	≥38°C
Hypotension	None	No vasopressor required	One vasopressor ± vasopressin	Multiple vasopressors, excluding vasopressin
Hypoxia	None	Low-flow nasal cannula ≤6 L/min or blow-by	High-flow nasal cannula >6 L/min, facemask, nonrebreather, or Venturi mask	Positive-pressure support: continuous positive airway pressure (CPAP), bilevel positive airway pressure (BiPAP), or intubation/mechanical ventilation

Grading notes

- Hypotension and hypoxia must not be attributable to another cause.
- Organ toxicities may be graded with Common Terminology Criteria for Adverse Events (CTCAE) criteria, but they do not determine the CRS grade.
- A patient with low-flow oxygen but one vasopressor is grade 3 because the more severe event determines grade.
- Always reassess and re-grade when support requirements change.

ASTCT consensus CRS grading is based on fever at onset plus the degree of hypotension, hypoxia, or both.² CTCAE v6.0 contains a CRS adverse-event term for toxicity reporting; it should not replace the ASTCT framework when the institution or protocol specifies ASTCT grading.³

CRS management recommendations

Grade	Core recommendations
All grades	Perform serial clinical assessment and institution-defined laboratory monitoring. Evaluation commonly includes a complete blood count (CBC), metabolic panel, inflammatory markers, coagulation testing, and infectious workup when indicated. Do not delay sepsis evaluation or treatment in an unstable patient. For grade 2 or higher CRS, use continuous cardiorespiratory monitoring as clinically appropriate; consider echocardiography or additional hemodynamic assessment for severe CRS.
Grade 1	Provide supportive care with antipyretics, judicious intravenous hydration, and management of symptoms or organ toxicities. Evaluate and treat infection according to neutropenic-fever/sepsis guidance when applicable. Escalate treatment if fever persists, symptoms progress, or the product-specific/institutional algorithm recommends earlier intervention.
Grade 2	Continue supportive care; provide intravenous fluids, supplemental oxygen, or both as indicated. Tocilizumab dosing should follow current product labeling and the institution-approved pathway. A commonly used dose is 8 mg/kg intravenously (maximum 800 mg/dose) for patients weighing 30 kg or more; dosing differs for patients under 30 kg. Repeat dosing and corticosteroid use are product- and pathway-specific. Escalate promptly for persistent hypotension, increasing oxygen needs, or lack of clinical improvement.
Grade 3	Provide ICU-level evaluation/care, with vasopressor and respiratory support as indicated. Use tocilizumab, including repeat dosing as permitted by current labeling and the institutional pathway, with corticosteroids according to the product-specific and institution-approved severe-CRS pathway. Reassess frequently and escalate refractory toxicity with cellular-therapy/critical-care input.
Grade 4	Provide ICU care with advanced hemodynamic and respiratory support as needed. Use tocilizumab when appropriate and high-dose corticosteroids according to the current severe-CRS pathway. Refractory CRS requires urgent expert-guided escalation and evaluation for overlapping syndromes such as infection or IEC-HS.

SAFETY NOTE

These recommendations are a reference—not a standing order.

Confirm current US Food and Drug Administration (FDA)-approved product labeling and the institution-approved CRS algorithm before treatment. Product labels differ in when to begin tocilizumab or corticosteroids, monitoring expectations, and management of refractory toxicity.



ICANS: small neurologic changes matter



RECOGNIZE

Writing and language changes may appear before dramatic decline.

Watch for impaired attention, disorientation, word-finding difficulty, handwriting change, tremor, weakness, somnolence, agitation, severe headache, seizure, or reduced consciousness.

Assess with discipline

- Compare with the patient's documented neurologic and handwriting baseline.
- Complete the ICE assessment when applicable—and assess consciousness, motor findings, and seizures beyond the ICE score.
- Review sedating medications and metabolic, infectious, vascular, and structural causes.
- Use seizure precautions, fall/aspiration precautions, and airway support when indicated.
- Use neurology consultation, electroencephalography (EEG), neuroimaging, lumbar puncture, rapid response, or ICU evaluation as indicated by the institution-approved pathway.

What the guidance says

Because patients with moderate-to-severe ICANS can deteriorate rapidly, guidance supports prompt corticosteroids plus supportive care, followed by tapering after sustained improvement. Tocilizumab should be reserved for concurrent CRS; it has not been shown to treat isolated ICANS.¹

Emergency neurologic signs

- Seizure
- Inability to awaken or protect the airway
- Rapidly worsening mental status
- New focal weakness or severe motor deficit
- Signs of increased intracranial pressure

DO NOT ANCHOR

Not every neurologic change is ICANS.

Evaluate stroke, infection, metabolic encephalopathy, medication effects, seizure/postictal state, disease involvement, and other urgent causes while treating suspected ICANS.



ICE score: 10 points, five tasks



USE IT WELL

Trend the score—and compare it with baseline.

ICE screens cognition. ICANS grading also requires assessment of consciousness, seizures, motor findings, and raised intracranial pressure/cerebral edema.

ICE task	How to assess	Points
Orientation	Correctly states year, month, city, and hospital	4
Naming	Names 3 objects, such as a clock, pen, and button	3
Following commands	Performs a simple command, such as showing 2 fingers	1
Writing	Writes a standard sentence	1
Attention	Counts backward from 100 by 10	1
Total	Maximum score	10

ICE result	Interpretation
10	No impairment on the ICE screen
7-9	Compatible with grade 1 ICANS if no more severe domain is present
3-6	Compatible with grade 2 ICANS if no more severe domain is present
0-2	Compatible with grade 3 ICANS if awake; assess all other domains
0 (unarousable)	Grade 4 ICANS

The ICE tool assigns 10 points across orientation, naming, commands, writing, and attention.²

Pediatric note: For children younger than 12 years and for older patients who cannot reliably complete ICE because of developmental status, ASTCT recommends the Cornell Assessment of Pediatric Delirium (CAPD) to aid ICANS grading. Other ICANS domains—level of consciousness, seizures, motor findings, and raised intracranial pressure/cerebral edema—still apply.²

Adult ICANS grading

Domain	Grade 1	Grade 2	Grade 3	Grade 4
ICE score	7-9	3-6	0-2	0; unarousable and unable to perform ICE
Consciousness	Awakens spontaneously	Awakens to voice	Awakens only to tactile stimulus	Unarousable or requires vigorous/repetitive tactile stimulation; stupor/coma
Seizure	Not applicable	Not applicable	Any focal/generalized seizure resolving rapidly; or nonconvulsive EEG seizure resolving with intervention	>5-minute life-threatening seizure; or repetitive seizures without return to baseline
Motor finding	Not applicable	Not applicable	Not applicable	Deep focal weakness, such as hemiparesis or paraparesis
Raised intracranial pressure / cerebral edema	Not applicable	Not applicable	Focal/local edema on imaging	Diffuse cerebral edema; decerebrate/decorticate posturing; cranial nerve VI palsy; papilledema; or Cushing triad

Grade by the most severe domain not attributable to another cause. ICE 0 can be grade 3 if the patient is awake with global aphasia; it is grade 4 when the patient is unarousable.

ICANS management recommendations

Grade	Core recommendations
All grades	Perform serial neurologic assessments, including ICE when applicable, and evaluate for competing causes. Consider neurology consultation, seizure precautions, aspiration/fall precautions, and head elevation. EEG, neuroimaging, lumbar puncture, and antiseizure prophylaxis should be individualized according to symptoms, grade, product labeling, and the institutional neurotoxicity pathway.
Grade 1	Provide supportive care and close reassessment. If concurrent CRS is present, treat CRS according to its grade. Corticosteroid use for grade 1 ICANS varies by product and patient risk; follow current product labeling and the institution-approved neurotoxicity algorithm.
Grade 2	Continue supportive care and initiate corticosteroids according to current product labeling and the institution-approved ICANS pathway (commonly dexamethasone in adults). Reassess frequently and escalate the level of care for worsening mental status, seizure, airway risk, hemodynamic instability, or concurrent severe CRS.
Grade 3	Higher-level or ICU care is generally appropriate. Use corticosteroids per the severe-ICANS pathway and continue until clear improvement, followed by an individualized taper. Treat concurrent CRS separately with tocilizumab when indicated.
Grade 4	Provide ICU care; protect the airway and treat seizures/status epilepticus promptly. High-dose methylprednisolone is commonly used for grade 4 ICANS according to guideline-, product-, and institution-specific pathways. Persistent or worsening toxicity requires urgent multidisciplinary evaluation and expert-guided alternative therapy.



Other toxicities can still be high stakes



KEY DISTINCTION

Tocilizumab is directed at concurrent CRS.

It has not been shown to mitigate isolated ICANS. Monitor for ICANS relapse during corticosteroid taper and follow the product- and institution-specific neurotoxicity pathway.

Immune effector cell-associated hemophagocytic lymphohistiocytosis-like syndrome (IEC-HS)

- Suspect IEC-HS when a new or worsening hyperinflammatory syndrome develops with elevated or rapidly rising ferritin plus features such as cytopenias, coagulopathy/hypofibrinogenemia, transaminitis, or organ dysfunction. It typically emerges as CRS is resolving or after an initial CRS course, but infection, malignancy progression, and other causes must be evaluated in parallel.
- IEC-HS is a specialist emergency. Escalate to the cellular therapy team and higher-level care while completing a parallel evaluation for infection and other causes of hyperinflammation.
- Consensus-based first-line therapy may include anakinra with or without corticosteroids, with individualized escalation for refractory disease; follow the institution-approved IEC-HS pathway.

ASTCT consensus guidance defines IEC-HS as a distinct hyperinflammatory toxicity after immune effector cell therapy. Current product labels may instead use hemophagocytic lymphohistiocytosis/macrophage activation syndrome (HLH/MAS) terminology.⁴

Cytopenias / immune effector cell-associated hematotoxicity (ICAHT)

- Anemia, neutropenia, and thrombocytopenia may be early, prolonged, or biphasic. The European Hematology Association/European Society for Blood and Marrow Transplantation consensus framework defines early ICAHT as occurring from day 0 through day 30 and late ICAHT as occurring after day 30.⁵
- Assess bleeding, infection, transfusion needs, marrow recovery, medication effects, nutritional factors, disease, and other causes.
- Use transfusion support, growth-factor strategies, antimicrobial prophylaxis, and additional diagnostic evaluation according to severity, timing, marrow recovery, concurrent toxicities, and institutional guidance.

Humoral immune dysfunction / hypogammaglobulinemia

- B-cell aplasia is most directly associated with CD19-directed CAR T-cell therapy, while hypogammaglobulinemia and impaired humoral immunity can occur after both CD19- and B-cell maturation antigen (BCMA)-directed products.^{6,7}
- Monitor immunoglobulins, clinically relevant immune recovery, and recurrent or severe infections.
- Vaccination, antimicrobial prophylaxis, and immunoglobulin replacement decisions should follow product-, patient-, and institution-specific guidance.

Coagulopathy / disseminated intravascular coagulation (DIC)

- Look for bleeding, bruising, abnormal coagulation results, decreasing fibrinogen levels or platelet counts, thrombosis, and organ dysfunction.
- Treat the underlying inflammatory syndrome and provide urgent blood-product and critical-care support as indicated.⁸

Delayed or product-specific toxicities

Non-ICANS neurologic toxicities can occur outside the classic ICANS window, particularly after BCMA-directed CAR T-cell therapy, and may include cranial nerve palsies, movement/neurocognitive syndromes, peripheral neuropathy, Guillain-Barré syndrome, or myelitis. New neurologic symptoms warrant prompt product-specific evaluation even after CRS or ICANS has resolved.^{7,9,10}

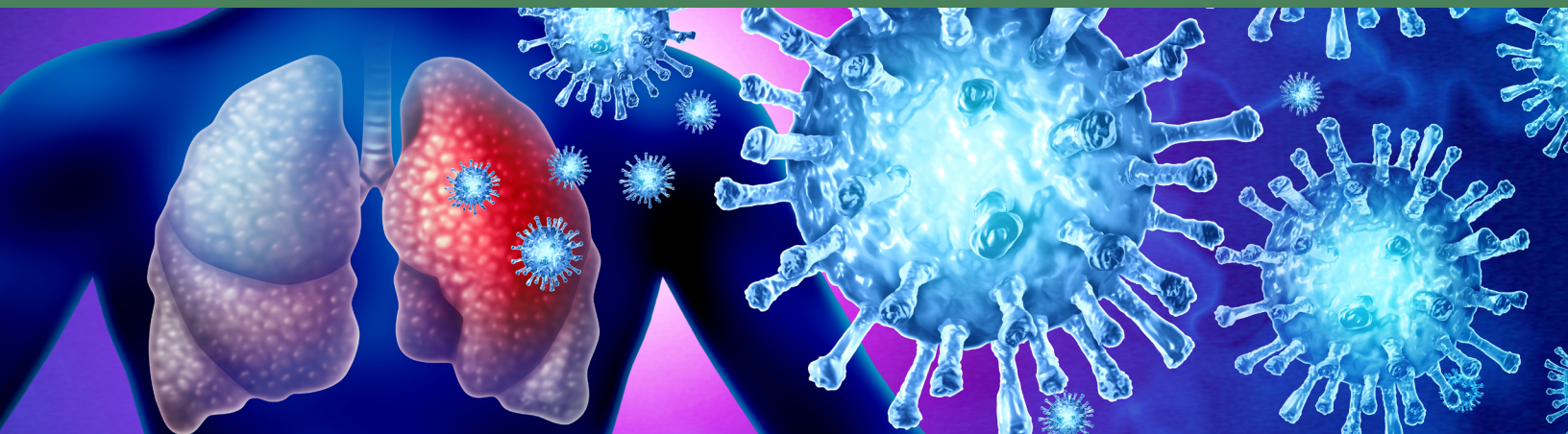
Immune effector cell-associated enterocolitis (IEC-EC) is a rare, potentially life-threatening delayed toxicity reported predominantly after ciltacabtagene autoleucel. Severe or prolonged diarrhea, abdominal pain, and weight loss weeks to months after infusion require urgent infectious and gastrointestinal evaluation; FDA added IEC-EC to the CARVYKTI Boxed Warning in October 2025.^{7,11,12}

Secondary malignancies

Secondary hematologic malignancies, including T-cell malignancies, are a class safety concern after BCMA- and CD19-directed genetically modified autologous CAR T-cell immunotherapies. FDA recommends lifelong monitoring for secondary malignancies; if a new malignancy occurs, follow the product-specific reporting and testing instructions.^{8,13,14}



Infection can mimic—and compound—toxicity



THE RULE

CRS is not a reason to dismiss infection.

CAR T-cell recipients may have neutropenia, mucosal injury, prior treatment exposure, central access, corticosteroid exposure, and humoral immune dysfunction. Fever deserves parallel infectious and inflammatory evaluation.

Early infection risk

- Bacterial infection and neutropenic fever
- Viral reactivation or respiratory infection
- Fungal infection in higher-risk or heavily immunosuppressed patients
- Line, pulmonary, urinary, gastrointestinal, skin, or other focal sources

Later infection risk

- Prolonged cytopenias
- Humoral immune dysfunction / hypogammaglobulinemia
- Corticosteroid or other immunosuppressive exposure
- Incomplete immune reconstitution

The management pattern

- **Culture and evaluate:** Obtain institution-defined laboratory tests, cultures, imaging, and source evaluation without delaying treatment in unstable patients.
- **Treat promptly:** In febrile neutropenia, sepsis, or clinical instability, start empiric antibacterial therapy

according to institutional febrile-neutropenia/sepsis pathways. Tailor route and spectrum to acuity, neutropenia, organ function, local epidemiology, and infectious disease guidance.

- **Reassess prophylaxis:** Align antibacterial, antiviral, antifungal, and Pneumocystis prophylaxis with current institutional and product-specific standards.
- **Track recovery:** Monitor blood counts, immunoglobulins, immune recovery, and recurrent infections over time.

Infection risk evolves over time after CAR T-cell therapy and is influenced by lymphodepletion, cytopenias, B-cell/plasma-cell dysfunction, corticosteroid exposure, and delayed immune reconstitution. Prevention and management should follow current CAR T-specific infectious-disease guidance.⁶

OUTPATIENT TRIGGER

Fever requires immediate contact.

Do not wait for the next visit or rely on a portal message. Follow the 24/7 CAR T-cell pathway and destination instructions.



The outpatient response: make the next move obvious



DESIGN PRINCIPLE

Every symptom needs an owner, a clock, and a destination.

The pathway should identify who answers, who grades, who orders, who prepares medication, who accepts admission, where the patient goes, and what happens when the usual route fails.

Call now

- Fever at or above the institution-provided threshold
- New confusion, speech or writing change, tremor, weakness, somnolence, or severe headache
- New supplemental oxygen requirement, dyspnea, dizziness, hypotension, syncope, or rapid heartbeat
- Uncontrolled vomiting/diarrhea, poor intake, bleeding, or any rapid change
- Caregiver or monitoring failure that makes outpatient care unsafe

Activate emergency response

- Seizure, loss of consciousness, or inability to awaken
- Severe breathing difficulty, cyanosis, collapse, or severe chest pain
- Any immediately life-threatening deterioration

Track what matters

- Symptom onset to first call
- First call to trained clinician and in-person assessment
- Order tocilizumab, corticosteroid, antimicrobial, or other urgent treatment
- Bed request to admission; high-grade toxicity to ICU transfer
- Protocol deviations, failed contacts, transport delay, and medication delay

Outpatient expert recommendations emphasize 24/7 access to a trained clinical pathway, frequent early assessment, rapid access to toxicity-directed medications, predefined escalation/admission criteria, and reliable transfer processes. Exact monitoring frequency and location should follow the CAR T-cell product, patient risk, and institutional model.¹⁵⁻¹⁸

THE TAKEAWAY

Fast recognition is treatment.

The best toxicity plan does not merely describe what to give. It ensures that the right patient reaches the right clinician and level of care and receives the right medication before the syndrome outruns the system.



Quick Myth Check



MYTH: Fever after CAR T-cell is probably CRS.	REALITY: Fever may be CRS, infection, or both. Evaluate in parallel.
MYTH: A normal ICE score rules out neurotoxicity.	REALITY: Consciousness, seizures, motor findings, and other neurologic changes also affect ICANS severity.
MYTH: Tocilizumab treats all CAR T-cell immune toxicity.	REALITY: Tocilizumab is used for CRS; it does not treat isolated ICANS.
MYTH: Toxicity is only an early problem.	REALITY: Cytopenias, immune dysfunction, infections, delayed product-specific toxicities, and secondary malignancies may persist or emerge later.
MYTH: Admission means outpatient care failed.	REALITY: Prompt admission can show that recognition and escalation worked.
MYTH: The toxicity algorithm is enough.	REALITY: Safe care also requires staffing, medication access, caregiver education, transport, handoff, and bed capacity.

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