ERN-EuroBloodNet Topic on Focus on Inherited Platelet Function Disorders (IPFD)

webinar

HEALTH PROFESSIONALS

Glanzmann Thrombasthenia

Session 10 · General Aspects of Treatment

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Educational Session ·
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Agenda

1 **Background** — What is Glanzmann thrombasthenia?

2 **Current treatment** — Tranexamic acid, platelet transfusion, recombinant FVIIa

3 **Potentially curative** — Haematopoietic stem cell transplant — the published case experience

4 **Innovative therapies** — Sutacimig in Glanzmann thrombasthenia — mechanism and Phase 1/2 data

5 **Emerging strategies** — Concizumab (anti-TFPI) and gene therapy approaches in GT

6 **Extending the approach** — Sutacimig in factor VII deficiency

7 **Close** — Summary and take-home messages

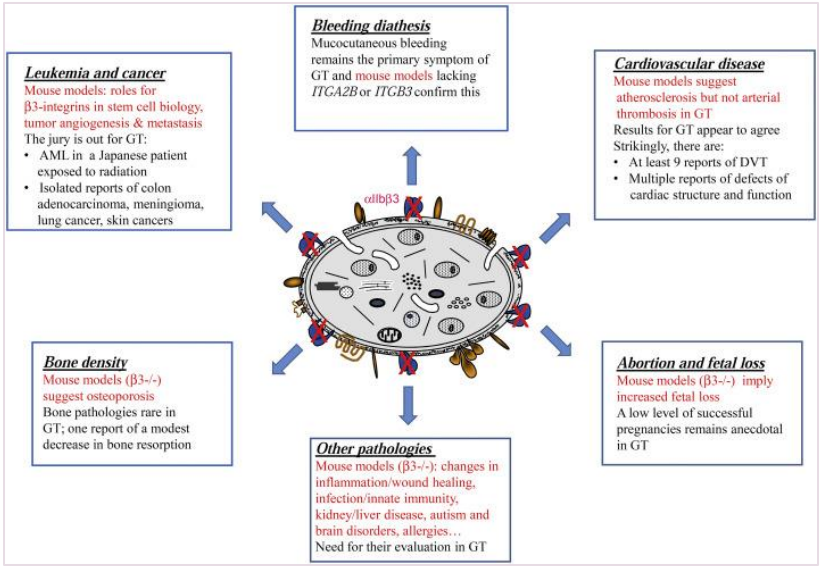
01

BACKGROUND

What is Glanzmann thrombasthenia?

What Is Glanzmann Thrombasthenia?

- **Autosomal recessive bleeding disorder:** quantitative or qualitative deficiency of platelet integrin αIIbβ3 (GPIIb/IIIa) — platelets fail to aggregate via fibrinogen despite a normal platelet count.
- Estimated prevalence **~1 in 1,000,000** worldwide — one of the rarest bleeding disorders.
- **Core phenotype:** mucocutaneous bleeding — purpura, epistaxis, gingival bleeding, menorrhagia, GI haemorrhage; major risk with trauma, surgery and childbirth.
- **Diagnosis:** absent aggregation to all agonists except ristocetin (light transmission aggregometry), confirmed by flow cytometry and/or genetic testing.
- **αIIbβ3 is not platelet-exclusive:** an emerging literature links GT to a broader phenotype beyond bleeding — clinicians should stay alert to it (right).



Beyond bleeding: the extended GT phenotype under active study — cardiovascular disease, fetal loss, bone density, cancer risk and more (Nurden AT. Blood Rev. 2017;31(5):287–299).

02

CURRENT TREATMENT

Tranexamic acid, platelet transfusion, recombinant FVIIa

Tranexamic Acid

- **Mechanism:** antifibrinolytic — competitively binds lysine-binding sites on plasminogen, blocking its interaction with fibrin and preventing plasmin-mediated clot breakdown; stabilises the fibrin clot at sites of mucosal injury.
- **Role in GT:** first-line adjunct for mild mucocutaneous bleeding (epistaxis, gingival bleeding, menorrhagia) and for dental/minor procedures, where it is often sufficient alone.
- **Combination use:** for major bleeding or surgery, used alongside platelet transfusion or recombinant FVIIa rather than as monotherapy.
- **Practical dosing:** oral 15–25 mg/kg 2–3×/day, or IV 10 mg/kg; a topical/mouthwash formulation is particularly useful for gingival and dental bleeding.
- **Caution:** avoid in frank upper urinary tract bleeding (risk of ureteric clot colic/obstruction); use with care in patients with a personal history of thrombosis.

Platelet Transfusion — Efficacy and the Alloimmunisation Challenge

- Mainstay for moderate–severe bleeding and peri-operative cover when TXA (± rFVIIa) is insufficient; leukoreduced platelets used throughout to reduce HLA sensitisation.
- **Central challenge — alloimmunisation:** patients can develop antibodies to HLA class I antigens and, specifically to GT, to the GPIIb/IIIa complex itself on donor platelets.
- **Risk tracks genotype:** biallelic null (type I) variants — including the ITGA2B c.1544+1G>A founder mutation in the French Manouche population — carry the highest risk (an earlier French cohort found ~81% vs ~25% antibody positivity in null vs non-null genotypes).
- **Mechanistically reassuring:** anti-GPIIb/IIIa sera reduce fibrinogen binding in vitro but do not trigger complement- or CD62P-mediated platelet

36% of 55 GT patients developed anti-αIIbβ3 and/or anti-HLA antibodies on prospective follow-up

Mostly transient anti-αIIbβ3 antibodies typically resolve by 18 months; anti-HLA can persist longer

Desprez D, et al. J Thromb Haemost. 2026;24:2627–2639 (French multicentre TAAS cohort, n=55).

Monitoring is unreliable: in a retrospective cohort of 4 GT patients (Rutten et al., Platelets 2026), light-transmission aggregometry stayed reduced for almost every agonist after transfusion despite satisfactory clinical haemostasis — haemostatic benefit should be judged clinically, not by a normalised aggregometry trace.

Recombinant Factor VIIa (rFVIIa)

- **Mechanism:** bypasses the need for platelet GPIIb/IIIa–fibrinogen bridging — rFVIIa binds directly to the activated platelet surface and generates a burst of thrombin sufficient to form a stable, fibrin-rich plug without platelet aggregation.
- **Role in GT:** preferred in patients refractory to, or at risk from, platelet transfusion — particularly those with anti-GPIIb/IIIa or anti-HLA alloantibodies. Used for on-demand bleeding and surgical/procedural prophylaxis.
- **Dosing:** typically 90 mcg/kg IV, repeated every 2 hours for at least 3 doses in significant bleeding or surgery.
- **Pharmacokinetics:** short elimination half-life (~2.3–2.6 hours) drives the need for frequent re-dosing.
- **Safety — thrombosis is rare:** pooled safety data across 30 years of trials, registries and postmarketing surveillance (12,288 bleeding/surgical episodes) found an overall thrombotic event rate of 0.17%, and just 0.19% in GT specifically (1 event in 518 GT episodes) — versus 0.11% in haemophilia with inhibitors, 0.82% in FVII deficiency, and 1.77% in acquired haemophilia.
- **Who is at risk:** among reported thrombotic events, risk tracked older age (≥65y in 28.6%), cardiovascular disease (17.8%), and concomitant activated prothrombin complex concentrate use (17.8%) — factors uncommon in the typically younger GT population.

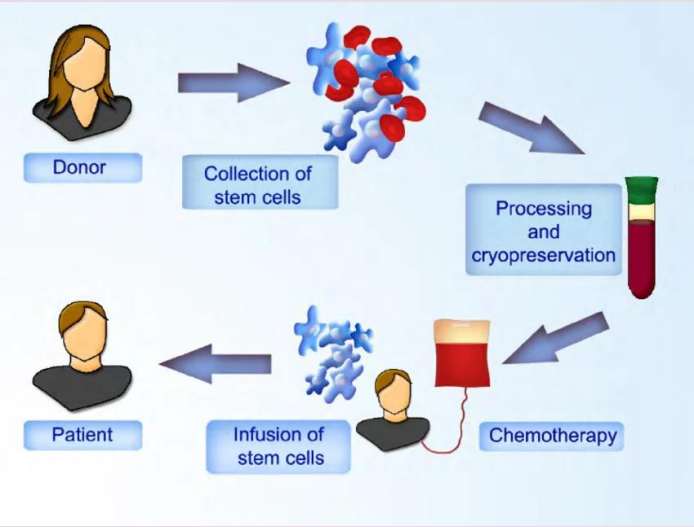
Rajpurkar M, Croteau SE, Boggio L, Cooper DL. J Blood Med. 2019;10:335–340.

03

POTENTIALLY CURATIVE

Haematopoietic stem cell transplant — the published case experience

Haematopoietic Stem Cell Transplant — The Case Report Evidence



Allogeneic HSCT process (generic schematic).

- Evidence base is limited to isolated case reports and small series — no prospective trial, registry, or comparative study of HSCT in GT exists. The largest published tally (2019) identified 19 allogeneic transplants across 18 patients since 1985.
- Reserved for severe, transfusion-refractory bleeding and/or significant alloimmunisation precluding ongoing platelet or rFVIIa therapy.
- Matched-sibling, matched-unrelated, and — more recently — haploidentical (T-depleted) donors have all been used successfully, with reduced-toxicity conditioning increasingly favoured.

ENGRAFTMENT

Rapid, durable

Full donor chimerism by ~day 28 in reported cases; neutrophil engraftment ~day 11–16.

COMPLICATIONS

Mostly mild

GvHD (usually mild, gut/skin), CMV/viral reactivation, immunosuppression toxicity; graft failure rare.

DURABILITY

Months–years

Bleeding-free follow-up reported to 18–24 months and beyond; long-term (>10y) data remain sparse.

MORTALITY

No pooled rate

No GT-specific mortality/TRM rate has been published; reported cases (~19 transplants) describe no deaths, but numbers are too small to estimate one reliably.

HSCT in GT — The Published Literature in Depth

STUDY	DONOR & CONDITIONING	OUTCOME REPORTED
Crone & James 2019 — Clin Med; UK, n=2	Haploidentical (T-depleted PB) + 10/10 MSD (BM); flu/thiotepa/treosulfan/ATG	Full donor chimerism by day 28 both patients; mild gut GvHD + CMV reactivation (pt1), ciclosporin-related toxicity (pt2); symptom-free at 18–24 months.
Li et al. 2022 — Front Pediatr; China, n=1	10/10 matched-sibling PBSCT, 2-year-old boy	"Successfully cured"; no significant transplant-related complications reported.
Pooled tally, 2019 — cited in Crone & James	Mixed — matched-sibling, matched-unrelated and haploidentical donors across reports since 1985	19 allogeneic HSCT reported in 18 patients in total; outcomes generally favourable but not systematically pooled.
Mixed-chimerism sibling series, 2025 — J Pediatr Hematol Oncol	2 siblings + literature review	Specifically examines mixed donor chimerism as a feature to monitor even when patients remain clinically bleeding-free.
Additional case reports/series — adult & paediatric	Donor types and conditioning vary across reports — see References	No registry or comparative-trial data exist; each report stands alone — individualise expectations accordingly.

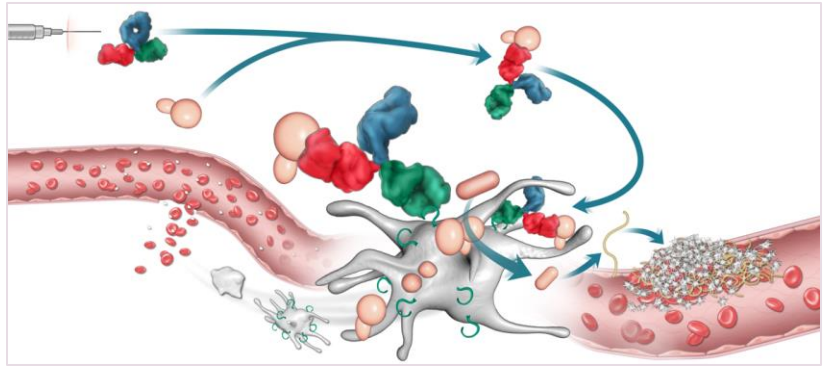
04

INNOVATIVE THERAPIES

Sutacimig in Glanzmann thrombasthenia — mechanism and Phase 1/2 data

Sutacimig: Targeting FVIIa to the Activated Platelet Surface

- **Subcutaneous bispecific antibody:** low-volume (<1 mL) SQ dose; one arm binds endogenous FVIIa, the other binds TLT-1, a receptor expressed only on activated platelets.
- **Extends FVIIa half-life:** endogenous FVIIa normally has a short half-life and weak affinity for activated platelets — sutacimig binds and stabilises it.
- **Targeted delivery:** because TLT-1 is expressed only on activated platelets, sutacimig localises FVIIa specifically to the site of vessel injury.
- **Net effect:** enhanced thrombin generation and fibrin formation at the bleeding site — independent of the defective GPIIb/IIIa receptor itself.



Sutacimig binds endogenous FVIIa and, following vessel injury, localises it to the activated-platelet surface via TLT-1. Gandhi PS, et al. Nat Cardiovasc Res. 2024.

Phase 1/2 Study: Multiple Ascending Dose + Long-Term Extension

- **Primary objective:** safety and tolerability. Other objectives: PK/PD, and efficacy based on annualised treated bleed rate (ATBR) reduction.
- **Key entry criteria:** confirmed GT diagnosis, history of bleeding requiring treatment, adults 18–67 years.
- **Design:** ≥ 6-week run-in → Part A single ascending dose → Part B 12-week treatment across 4 dose cohorts → Part C long-term extension, up to 20 months.
- **Dose cohorts (Part B, N=34):** 0.3 mg/kg Q1W (n=5); 0.3 mg/kg Q2W (n=11); 0.6 mg/kg Q2W (n=13); 0.9 mg/kg Q2W (n=5).

N=34 treated in Part B; 28 continued into the long-term extension

7.3 mo mean treatment duration (median 6.9; range 1.9–15.9); 25 participants exposed ≥ 6 months

Data as of interim cut-off 14 Oct 2025. NCT06211634.

Study Population: Demographics and Baseline Bleeding Burden

CHARACTERISTIC	N=34
Age, years	Median (range): 40.5 (18–66)
Sex, n (%)	Female 16 (47); Male 18 (53)
Weight, kg	Median (range): 75 (52–134)
Total FVII(a), %	Median (range): 99.5 (51.1, 164.5)
Baseline ATBR during run-in	Mean (SEM) 40.2 (11.8); range 0–324.7; median (Q1, Q3) 17.7 (4.0, 47.6)
Cause of treated bleeds during run-in, %	Spontaneous 75.0; Traumatic 19.3; Iatrogenic 4.7; Not categorised 1.0

Data as of interim cut-off 14 Oct 2025. Efficacy set for run-in bleeding excludes 3 participants (run-in <6 weeks or diary noncompliance).

Generally Manageable Safety and Tolerability Profile

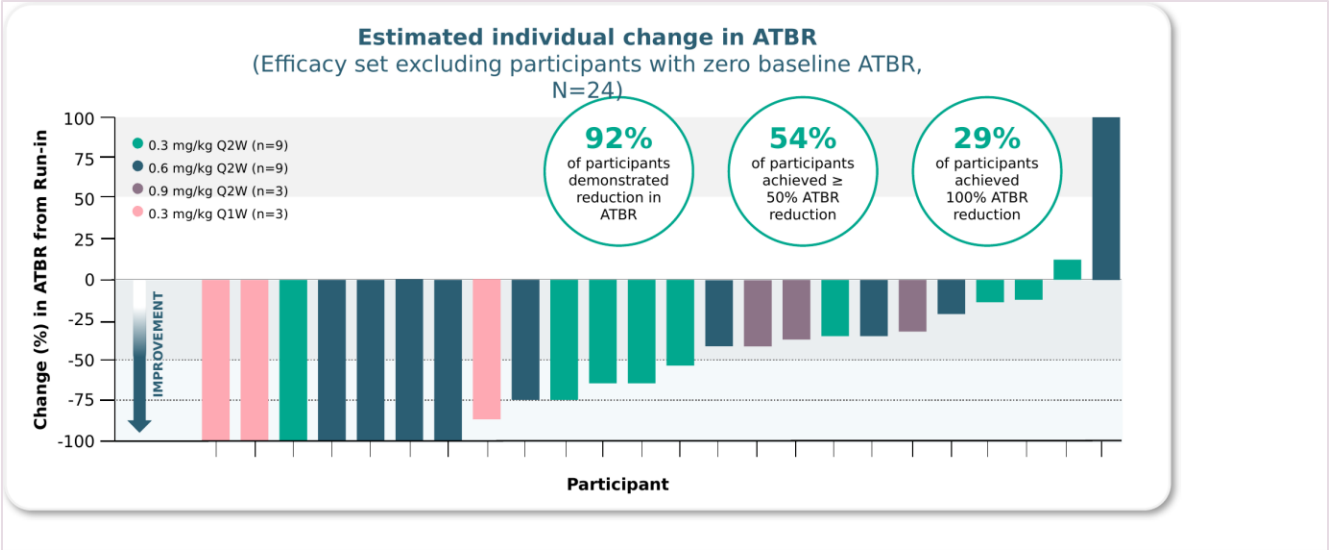
12-week treatment + extension — median exposure 6.9 months (range 1.9, 15.9)

All TEAE	**32 (94.1%)**
Related TEAEs	**15 (44.1%)**
Grade 3+ TEAEs	**7 (20.6%)**
Serious AEs	**5 (14.7%)**
Related SAEs	**2 (5.9%)**
Discontinuations (AE)	**4 (11.8%)**
Deaths	**0**

Treatment + Extension Period, Part B + C (N=34).

- **Most common TEAEs:** headache (23.5%), fibrin D-dimer increased (23.5%), nasopharyngitis (20.6%), back pain (17.6%) — mostly mild to moderate. No related TEAE ≥ Grade 3.
- **Venous thromboembolism:** 3 Grade 2 VTEs (related), all at higher dose levels and/or with ≥ 3 concurrent VTE risk factors; drug discontinued, anticoagulation given, all resolved or resolving at last visit.
- **Anti-drug antibodies:** 6/34 developed ADA titres (2 after a single Part A dose); 3/6 resolved/resolving at cut-off; no associated safety events.

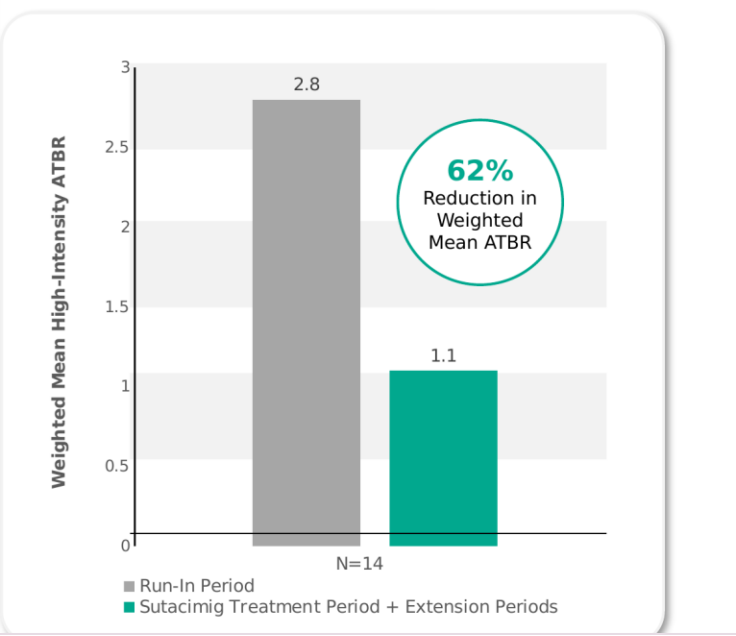
Clinically Meaningful, Persistent Reduction in Bleeding Rate



Estimated individual change in ATBR, efficacy set excluding zero-baseline participants (N=24). Data cut-off 14 Oct 2025.

- **Reduction observed across all dose cohorts:** efficacy-set mean ATBR reduction 47% (24, 63) over the 12-week treatment period and 47% (26, 62) with extension (N=31).
- **Enhanced response with weekly dosing:** 0.3 mg/kg Q1W reached 84–87% reduction, versus 38–45% for 0.3–0.6 mg/kg Q2W and 11–19% for 0.9 mg/kg Q2W — a dose-frequency, not just dose-level, effect.
- **Effect was persistent,** not just an early-treatment phenomenon — comparable reduction was maintained through the extension period (median exposure 6.9 months).

Reduced Need for High-Intensity Treatment; Successful Procedures



- **High-intensity treatment =** systemic haemostatic intervention (rFVIIa, platelet transfusion, plasma, cryoprecipitate) or a medical/surgical/radiologic procedure for haemostasis.

Subpopulation with prior high-intensity treatment use, N=14. Data cut-off 14 Oct 2025.

PARTICIPANTS WITH PROCEDURES ON SUTACIMIG

Partial mastectomy & lymph node biopsy

Dose: 0.3 mg/kg Q1W • IV TXA 1g + rFVIIa 7 mcg/kg pre-procedure; oral TXA post-discharge

✓ **Successful haemostasis**

Moh's surgery (basal cell carcinoma, scalp)

Dose: 0.9 mg/kg Q2W • Oral TXA, peri-procedural only

✓ **Successful haemostasis**

Gingival plaque removal

Dose: 0.6 mg/kg Q2W • rFVIIa 5 mcg/kg IV + topical rFVIIa

✓ **Successful haemostasis**

Conclusions: Proof of Concept Confirmed in GT

- **At > 6 months of treatment, sutacimig data support advancing to Phase 3:** safety profile remained stable with continued dosing and was overall generally well tolerated.
- **Proof of concept confirmed:** clinically meaningful mean ATBR reduction across dose levels, a marked reduction in bleeding events requiring high-intensity treatment, and successful management of surgeries with minimal concurrent haemostatic agent use.
- **Exposure range identified** to mitigate the risk of thromboembolic events observed at higher/more frequent dosing.
- **Phase 3 dose identified:** FDA alignment reached to enter Phase 3 with 0.2 mg/kg once weekly. Next step: a global Phase 3 study, proceeding in 2026.

05

EMERGING STRATEGIES

Concizumab (anti-TFPI) and gene therapy approaches in GT

Concizumab: Repurposing Anti-TFPI to Boost Thrombin Generation in GT

- **Mechanism:** concizumab is a monoclonal antibody against tissue factor pathway inhibitor (TFPI); blocking this natural brake on coagulation enhances thrombin generation — a route to better haemostasis that does not depend on the defective GPIIb/IIIa receptor.
- **Bordeaux in vitro study (Dubut et al. 2024):** blood/plasma from 10 patients with type I GT and 10 healthy controls, tested across thrombin generation, ROTEM, flow-chamber thrombus formation, and clot-lysis assays.
- **Concizumab improved every parameter tested:** shortened thrombin lag time by 33–42% (P<.0001), improved ROTEM clotting time, initiated flow-chamber thrombus formation in 67% of GT samples (P<.047), and extended clot-lysis resistance from 23 to 40–48 minutes (P=.005).
- **Important caveat:** TFPI inhibition improved but did not normalise haemostatic reactions in GT samples — in vitro proof of concept only; no clinical trial in GT patients yet.

Dubut J, Goin V, Derray C, et al. J Thromb Haemost. 2024;22(9):2589–2600.

Gene Therapy for Glanzmann Thrombasthenia — A Preclinical Field

- **Goal:** restore functional αIIbβ3 expression on megakaryocytes/platelets by introducing a corrective ITGA2B or ITGB3 transgene — in principle a one-time, curative alternative to lifelong on-demand therapy or HSCT.
- **Lentiviral vector gene transfer:** in a murine ITGB3-mutant GT model, lentiviral transduction of G-CSF-mobilised peripheral blood stem cells corrected platelet function (Wilcox et al.); an oncoretroviral β3 vector generated viable αIIbβ3 complex on megakaryocytes from human GT patients.
- **Large-animal proof of concept:** in a canine GT model (Great Pyrenees, ITGA2B mutation), lentiviral αIIb gene transfer stabilised at ~10% αIIbβ3-expressing platelets over 2–5 years' follow-up, with significantly decreased cutaneous bruising and shortened mucosal bleeding time.
- **iPSC approach:** monocytes from 2 type I GT patients were reprogrammed to induced pluripotent stem cells and corrected with a GP1ba promoter-driven αIIb transgene, achieving high expression in iPSC-derived megakaryocytes (Sullivan et al.).
- **Status:** entirely preclinical — no human gene-therapy trial in GT has been reported. Open challenges include achieving stable, high-level transgene expression, avoiding insertional mutagenesis, and managing host immune responses to the vector or gene product.

Poon MC, Di Minno G, d'Oiron R, Zotz R. Transfus Med Rev. 2016;30(2):92–99.

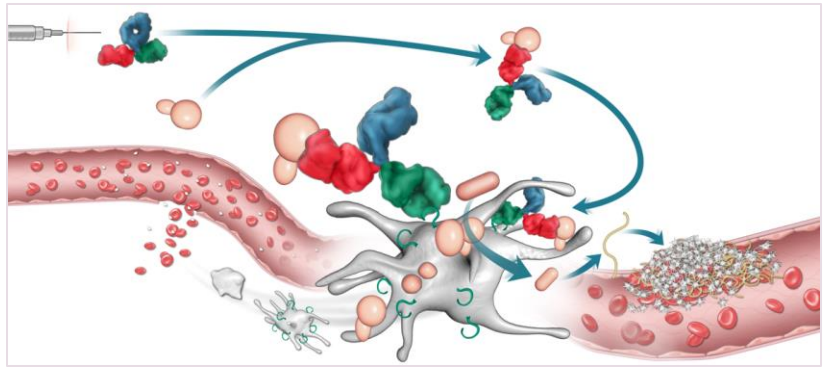
06

EXTENDING
THE APPROACH

Sutacimig in factor VII deficiency

Sutacimig: Mechanism and Rationale in FVII Deficiency

- **Sutacimig (HMB-001)** is a subcutaneously administered bispecific antibody: one arm binds and stabilises endogenous factor VIIa, the other binds TLT-1 on activated platelets — concentrating FVIIa at the platelet surface to amplify local thrombin generation.
- **Same mechanism, new indication:** the identical molecular approach developed for GT is being extended to congenital FVII deficiency, where insufficient circulating FVIIa — rather than a platelet receptor defect — limits thrombin generation.
- **Preclinical variant coverage (ISTH 2026):** confirmed binding across 22 of 25 tested FVII variants (88%), supporting predicted retained binding for >90% of variants in the severe-to-moderate FVIID mutation database — broad applicability across the FVIID population.
- **Status:** these preclinical findings underpin an ongoing Phase 2 study in Factor VII deficiency. No FVIID-specific clinical efficacy/safety data have been reported — the Phase 2 long-term extension data presented at ISTH 2026 remain GT-specific (34 patients).



Sutacimig bispecific mechanism — FVIIa-stabilising arm + TLT-1-binding arm on activated platelets (as characterised in GT).

Sutacimig in FVII Deficiency: Where This Stands

- **Conceptually attractive:** converts a bispecific-antibody platform validated in GT into a subcutaneous, non-replacement approach for a second rare bleeding disorder driven by FVII/FVIIa insufficiency.
- **Broad genetic reach:** >90% predicted variant coverage suggests the approach should not be restricted to a narrow genetic subgroup of FVIID.
- **Early stage:** clinical development is ongoing (Phase 2). Efficacy, safety and dosing in FVIID patients are not yet established and should not be extrapolated from the GT data presented to date.
- **Relevance for ERN practice:** worth tracking as a potential future subcutaneous prophylactic option for FVIID, alongside existing plasma-derived FVII concentrate and recombinant FVIIa on-demand therapy.

07

CLOSE

Summary and take-home messages

Summary & Take-Home Messages

GT management is stepwise — tranexamic acid for minor bleeding, platelet transfusion or rFVIIa for moderate–severe bleeding (chosen partly on alloimmunisation status), and HSCT reserved as the only curative option for severe, refractory disease.
Alloimmunisation is common but often transient; a pipeline beyond HSCT is emerging — sutacimig, concizumab and gene therapy — each targeting GT through a different mechanism.

Individualise treatment in specialist ERN centres, balancing bleeding severity, alloimmunisation risk, fertility/pregnancy plans and patient preference.

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