EAHAD 2026

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Congress Review

Review of the European Association for Haemophilia and Allied Disorders (EAHAD) 19th Annual Congress

Location:	Dublin, Ireland
Date:	3rd–6th February 2026
Citation:	EMJ Hematol. 2026;14[Suppl 1]:6-16. https://doi.org/10.33590/emjhematol/QY481M5A

IN the vibrant city of Dublin, Ireland, over 2,000 delegates from over 77 countries gathered for the 19th annual European Association for Haemophilia and Allied Disorders (EAHAD) Congress, to recognise the history, advancements, and persisting challenges in the field of haemophilia and associated disorders.

Niamh O'Connell, Consultant Haematologist, National Coagulation Centre (NCC), St James's Hospital, Dublin, and the EAHAD 2026 Congress President, opened the event by highlighting the society's missions and aims: to deliver high-quality clinical care, elevate education, and progress scientific research. Referencing the Irish word 'meitheal', meaning a collective voluntary effort by a community for the common good, based on reciprocity and trust, O'Connell drew similarities between this mindset and the approach of EAHAD: a unified community brought together with the aim of advancing haemophilia care. Closing her welcome, she encouraged everyone to make the most of the event: "Create networks, make connections, go home with heads full of plans, and computers full of ideas."

Following the welcome, a comprehensive summary of the 'Allied Healthcare Professionals' Day' was offered. This day, which took place on 3rd February, was a multidisciplinary event aimed to showcase the role of nurses, physiotherapists, and psychosocial professionals (PP) in the care and management of bleeding disorders. It featured both joint sessions, designed for all specialities, as well as streamed sessions tailored for each speciality. The joint session this year focused on the theme of family consultations, tackling a range of topics from intergenerational guilt, to navigating complex family dynamics and supporting young people with haemophilia.

Christina Burgess, Haemophilia and Bleeding Disorders Counselling Association (HBDCA), specifically summarised the PP stream, which were two sessions specifically aimed at PPs, recognising the emerging mental and social issues in patients, and offering guidance on psychosocial care offered at important transition points, such as trauma-sensitive care for a new diagnosis, the EAHAD European psychosocial principles for gene therapy, and the use of eye movement desensitisation and reprocessing therapy in treating anxiety and adjustment disorders in patients with haemophilia.

Maj Friberg Birkedal, Rigshospitalet, Copenhagen, Denmark, then took the

stage to offer more detail on the nursing stream sessions, starting with the classic SLAM session, which, as noted by Birkedal, "showcased the power of engaging nurse-led research projects." The SLAM session, a highlight of each EAHAD Congress, is an opportunity for healthcare professionals and researchers to present top-selected abstracts. Whilst topics did vary, the nurse stream SLAM session focused on the development of new care strategies and evaluating the impact of implementation. The winner of the SLAM session was Cristina Benedicto Moreno, Sant Joan de Déu Barcelona Hospital, Spain, with their abstract titled 'Implementation of a Nursing-led Remote Evaluation for the Assessment and Prioritization of Adolescents with Heavy Menstrual Bleeding'. The remaining sessions touched on the procedure-related anxiety in children and a masterclass on mucosal bleeds.

"The SLAM session is an opportunity for healthcare professionals and researchers to present top-selected abstracts"

Finally, Ruth Elise Dybvik Matlary, Oslo University Hospital, Norway, summarised the physiotherapists' stream. It began with a SLAM session, with topics covering blood-induced joint damage in Glanzmann thrombasthenia, patterns of habitual exercise among children with haemophilia, and long-term survival of hip and total knee arthroplasty in people with haemophilia, among others. The winner of the SLAM session was Paula Loughnane, Trinity College Dublin, with their abstract titled 'Patterns of Habitual Physical Activity among Irish Children and Adolescents with Haemophilia'. Following this, there was an informative session centred around joint health, including prehabilitation, intervention, and recovery, and finally, a debate that 'all people with bleeding disorders should have an annual musculoskeletal review'.

Rose Anne Kenny, Chair of Medical Gerontology, Trinity College and St James Hospital, subsequently took the stage, giving a talk on evidence-based strategies for successful ageing. She began summarising the following terms: 'lifespan', which is the maximum number of years an individual can live; 'life expectancy', which is the average age an individual can expect to live at different stages of life; and 'health span', which is the number of years an individual is healthy without chronic and debilitating disease.

However, as highlighted by Kenny, the world's population is ageing. Between 2023–2100, the population of people aged 65 years or over is set to increase by 49% in Albania, 41% in China, 34% in France, and 30% in the USA.[1] She highlighted the disparity between life expectancy and health-adjusted life expectancy, citing data showing that the gap between the two widened by 13% from 2000 to 2019. Delving into this further, there is also a reported sex difference, with women presenting a mean health span–life span gap of 2.4 (0.5 years) wider than men ($p<0.001$). This gap was found to be positively associated with the burden of noncommunicable diseases and total morbidity, and negatively with mortality. Finally, she shared data that showed that mental and substance use disorders, musculoskeletal disease, and unintentional injuries contributed the most to years lived with disability in the USA.[2]

Kenny then touched on the issue of multimorbidities, the presence of multiple medical conditions, which is known to increase with age.[3] She referenced data from a 2019 study, which investigated the disease associations in 6,101 Irish adults aged over 50 years.

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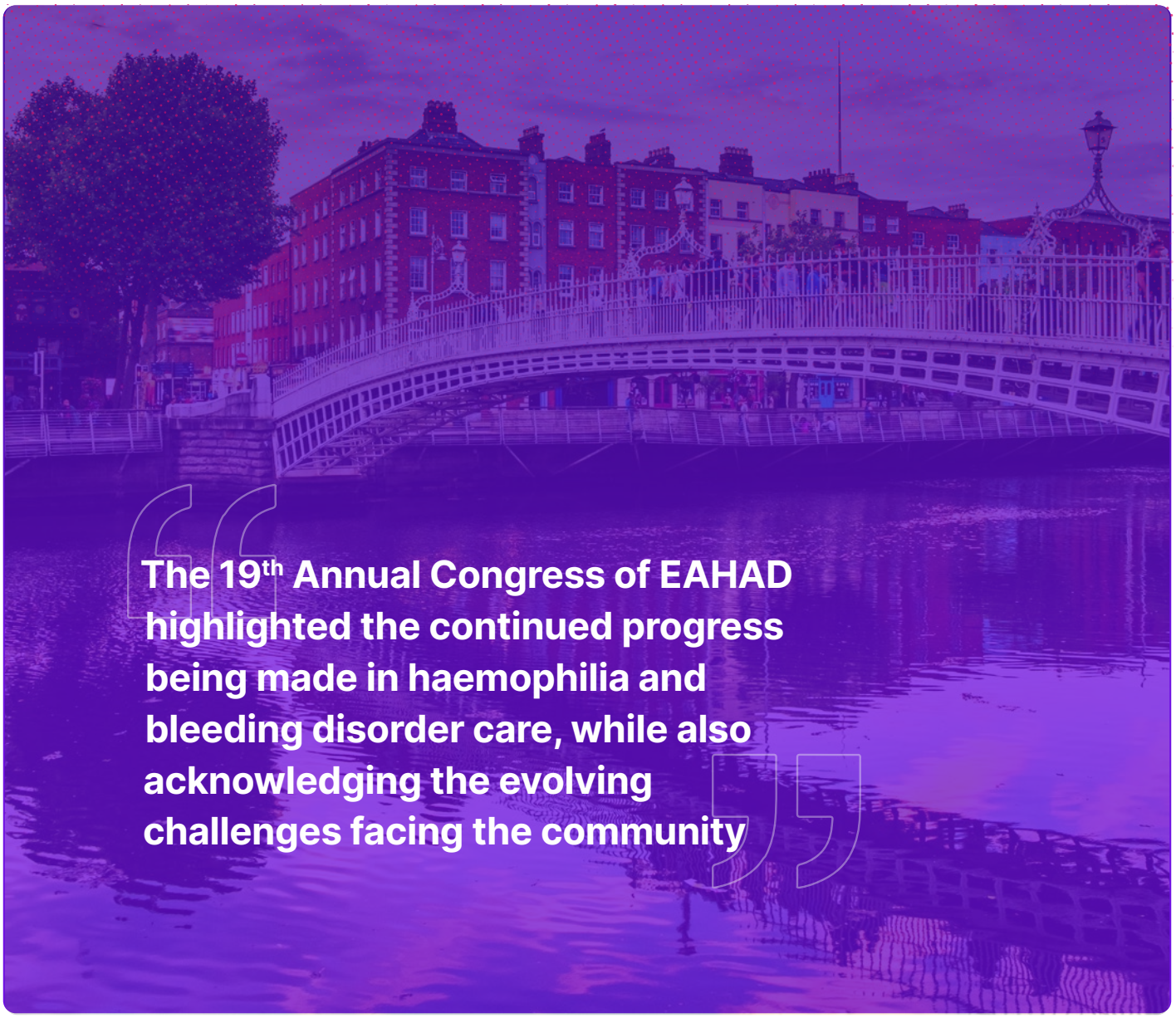
Results found the estimated lifetime prevalence of multimorbidity for older Irish adults was 73.25%, with only 9.08% not expressing any of the 31 diseases studied.[3] As emphasised by Kenny, this is important as these pre-existing conditions should be factored by healthcare professionals when assessing a patient's care plan. The researchers of this study then set out to investigate the likelihood of these comorbidities occurring more than by random chance.

A network of co-morbidities found that high cholesterol, arthritis, and hypertension were among some of the most highly prevalent morbidities among males.

In conclusion, the 19th Annual Congress of EAHAD highlighted the continued progress being made in haemophilia and bleeding disorder care, while also acknowledging the evolving challenges facing the community. Through multidisciplinary collaboration, the meeting showcased the vital contributions of clinicians, nurses, physiotherapists, PPs, and researchers in improving patient outcomes.



European Study Reveals Burden of Glanzmann Thrombasthenia

A EUROPEAN prospective study presented at EAHAD 2026 provided new insights into the clinical burden of Glanzmann thrombasthenia, highlighting significant gaps in the evidence guiding its management. The study, which is ongoing across multiple European centres, systematically collects clinical, laboratory, and patient-reported data to improve understanding of this rare bleeding disorder.[4]

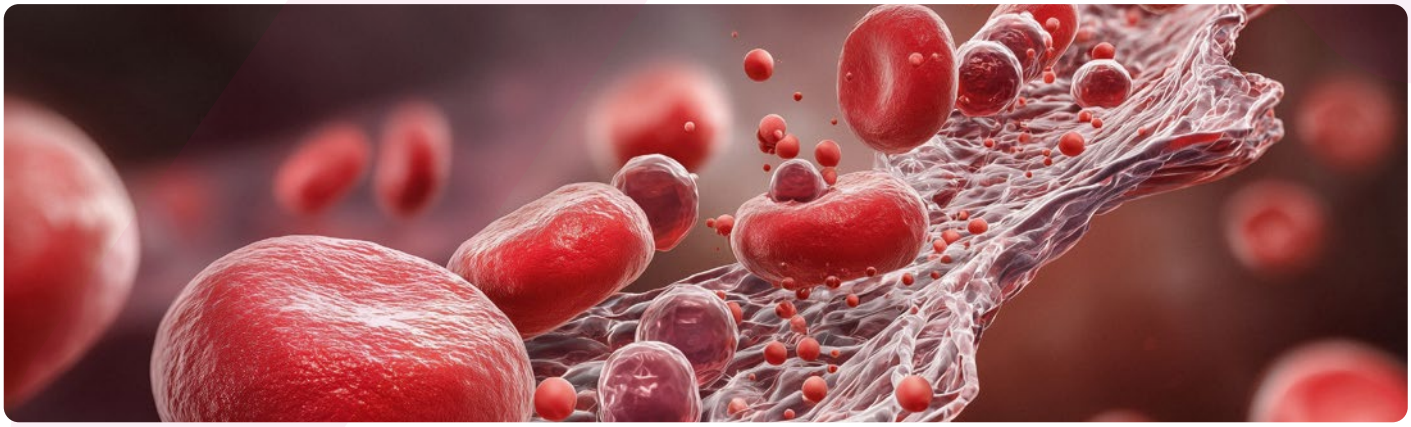
Glanzmann thrombasthenia is a rare inherited platelet disorder caused by the absence or dysfunction of the αIIbβ3 integrin receptor, which is essential for platelet aggregation. Patients experience lifelong mucocutaneous bleeding, including epistaxis, menorrhagia, and gastrointestinal bleeding. Although treatments such as tranexamic acid, recombinant activated Factor VII, and platelet transfusions are widely used, there is no consensus on optimal dosing or timing, particularly in high-risk scenarios, such as major surgery.

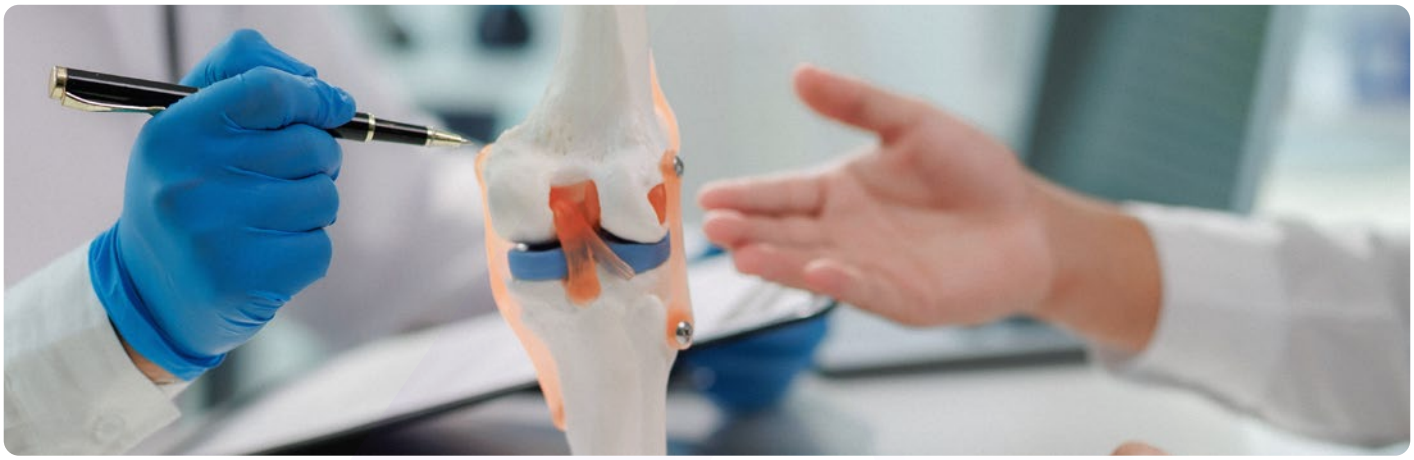
The Glanzmann Natural History Study (GNHS) is a prospective, multinational observational study including both paediatric and adult patients. It collects baseline clinical characteristics, bleeding history, comorbidities, patient-reported outcome measures, and quality-of-life data. Participants are followed longitudinally to document bleeding episodes, surgical interventions, and treatment responses. An additional 'plus' component includes genetic analyses to explore genotype–phenotype correlations and screening for alloantibodies against platelet antigens and human leukocyte antigens, reflecting the high lifetime exposure to platelet transfusions in this population.

As of early 2026, 32 patients had been enrolled, with 50–100 additional participants expected by the end of the year, aiming for a total accrual of 200 patients. Among the enrolled cohort, 50% were female, and patients represented all major Glanzmann thrombasthenia types (Types 1, 2, and 3). Significant comorbidities were observed, including two intracranial bleeds, four peripheral venous thrombotic events, one myocardial infarction, and one ischaemic stroke. Almost half of patients required continuous iron supplementation, indicating ongoing blood loss from mucocutaneous or gastrointestinal bleeding. Over a recent 4-week period, 40% of patients required medical treatment for bleeding.

The study also evaluated bleeding assessment tools. Traditional lifetime bleeding scores were found to be limited for detecting short-term changes following interventions. A short-term, patient-reported Immune Thrombocytopenia Purpura Bleeding Assessment Tool (ITP-BAT) demonstrated correlation with lifetime scores and sensitivity to recent changes, supporting its potential use for monitoring responses to therapy, including platelet transfusions, recombinant Factor VIIa, and emerging treatments.

Despite significant regulatory and logistical challenges, the GNHS has successfully engaged multiple European centres, providing a robust platform for ongoing data collection. The study continues to track clinical outcomes, interventions, and antibody prevalence, representing a crucial step toward developing evidence-based strategies for managing this rare but serious disorder.





MRI-Detected Joint Improvements with Emicizumab Prophylaxis

THIS prospective, observational, multicentre study presented at EAHAD 2026 evaluated the impact of emicizumab prophylaxis on joint outcomes in children with severe haemophilia A using both clinical measures and MRI-based structural assessment. Children with severe haemophilia A are at high risk of recurrent joint bleeding, which can lead to progressive arthropathy and reduced quality of life. While previous research on emicizumab has largely focused on clinical outcomes, this study aimed to assess its real-world effects on joint structure using MRI alongside established clinical scoring systems.[5]

The mean annual bleeding rate decreased from 50.00 to 0.46 ($p<0.001$), while the mean HJHS improved from 13 to 8 ($p<0.001$)

Male children aged <18 years with severe haemophilia A receiving emicizumab prophylaxis were enrolled. Participants underwent clinical and MRI evaluation of index joints at baseline and after 12 months of treatment. Clinical outcomes included changes in annual bleeding rate and the Haemophilia Joint Health Score (HJHS). Structural joint changes were assessed using MRI according to the International Prophylaxis Study Group (IPSG) MRI Scale Version 1.0.

A total of 39 children with severe haemophilia A were included, with a median age of 8 years (range: 4–17 years). In total, 54 joints were assessed, including 33 knees, 12 ankles, and nine elbows. After 1 year of emicizumab prophylaxis, both clinical and radiological outcomes improved significantly. The mean annual bleeding rate decreased from 50.00 to 0.46 ($p<0.001$), while the mean HJHS improved from 13 to 8 ($p<0.001$).

At baseline, MRI evaluation showed joint effusions in 100% of joints, with 50.0%, 22.2%, and 27.8% classified as small, moderate, and large effusions, respectively. After 12 months, moderate effusions decreased significantly to 11.1%, and no joints presented with large effusions. Synovial hypertrophy declined from 83.3% to 61.1%, and haemosiderin deposition decreased from 66.7% to 16.7% ($p<0.001$). Osteochondral changes improved in 22.2% of joints, while 5.6% showed progression. Overall, 88.9% of joints demonstrated improvement on MRI and 11.1% remained stable.

These findings indicate that emicizumab prophylaxis provides meaningful clinical and structural joint benefits, supporting its role in slowing joint disease progression and improving long-term musculoskeletal outcomes in children with severe haemophilia A.

Reference Values Enhance Interpretation of Haemophilia Joint Health Scores

NEW research presented at EAHAD 2026 introduces age-specific reference values for the Haemophilia Joint Health Score (HJHS), offering clinicians a more contextualised approach to monitoring joint health in patients with severe haemophilia.[6]

The HJHS is a standardised physical examination performed annually to assess joint status in people with haemophilia. Six index joints (both elbows, knees, and ankles) are evaluated to generate a composite score ranging from 0–124, with higher scores indicating poorer joint health. Although widely used in routine practice and research, interpretation of HJHS results can be challenging. Current assessment relies heavily on comparison with a patient's prior scores and the experience of the examining clinician and does not account for expected age-related changes in joint health.

To address this gap, Nguyen and colleagues analysed data from the Canadian Bleeding Disorder Registry (CBDR) collected between 2018–2024. The study included 551 individuals aged 4 years and older with severe haemophilia A or B. The cohort had a mean age of 24 years, with 54% classified as adults. Most participants (87%) had haemophilia A, 64% were receiving factor replacement therapy, and a 81% had negative inhibitor status.

Using Generalised Additive Models for Location, Scale, and Shape (GAMLSS), the investigators generated percentile curves (5th, 10th, 25th, 50th, 75th, 90th, and 95th) across ages 4–70 years. The resulting curves demonstrated a sigmoid trajectory, with joint deterioration accelerating from early adulthood to approximately 40 years of age, followed by a gradual deceleration into older adulthood. Internal validation showed good calibration, with an expected-to-observed ratio of 1.05. The model was also able to significantly discriminate between haemophilia type ($p=0.026$) and inhibitor status ($p<0.001$).

These reference values enable clinicians to compare an individual patient's HJHS with age-matched peers and monitor changes in percentile ranking over time. This approach provides clearer clinical context and may improve early identification of disproportionate joint deterioration.

The authors note that the model reflects therapies available between 2018–2024 and is limited to patients with severe haemophilia in Canada. International validation and expansion to broader patient populations will be important next steps to support wider implementation and global comparability of joint health outcomes.

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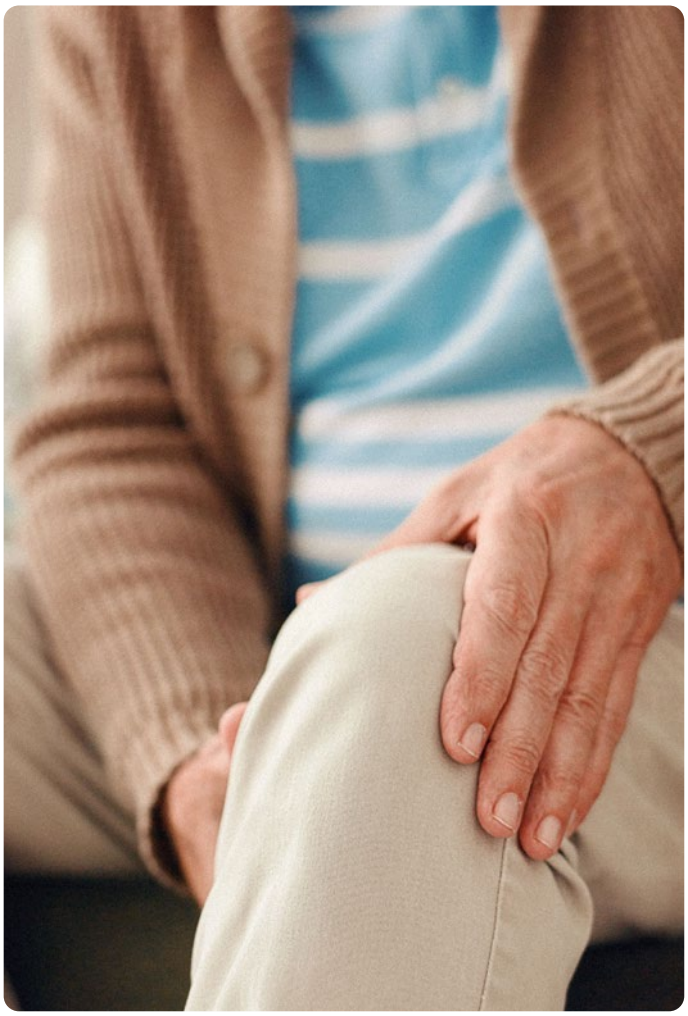

Ribosomal Readthrough Stratifies Inhibitor Risk in F8 Nonsense Mutations

NEW research presented at EAHAD 2026 explored why some patients with haemophilia A who carry factor VIII nonsense mutations develop inhibitors to replacement therapy, while others do not.[7]

Inhibitor formation occurs in around 30% of patients receiving therapeutic Factor VIII and represents one of the most serious complications of treatment. Although large gene deletions are known to carry high inhibitor risk, nonsense mutations have traditionally been considered 'non-conditioned' but show highly variable inhibitor rates. This study aimed to explain that variability.

The investigators focused on premature termination codons in the *factor VIII* gene and the phenomenon of ribosomal readthrough. In some cases, the ribosome can bypass a stop codon and produce a full-length protein. If the authentic (wild-type) amino acid is reinserted at the stop position, endogenous factor VIII may retain normal immunological properties, potentially reducing inhibitor risk. The hypothesis was that patients whose mutations allow wild-type readthrough may develop immune tolerance, whereas those who do not are at higher risk of inhibitors.

Patients were classified according to predicted wild-type readthrough and inhibitor status. The results showed that patients without inhibitors had a higher proportion of mutations predicted to undergo wild-type readthrough. In contrast, those with high-titre or multiple inhibitors had far fewer such mutations. Certain stop codon classes were associated with different risks: some codons (such as UAA-type variants) showed higher rates of wild-type readthrough and lower inhibitor prevalence, while others (such as UGA-type variants) were associated with higher risk. Mutation location also mattered, with highly immunogenic domains (particularly C1 and C2) showing lower wild-type readthrough and higher inhibitor association.

To support these findings, the team expressed 45 different premature termination codon variants *in vitro* using a luciferase-based system to quantify full-length factor VIII production. While overall protein levels did not differ significantly between groups, within the non-wild-type-readthrough group, lower levels of full-length factor VIII were associated with inhibitor development, supporting a protective role for endogenous protein expression.

Finally, *in silico* analyses evaluated *HLA-DRB* allele binding affinity of peptides derived from readthrough-generated variants. Mutations associated with inhibitors showed greater predicted immunogenic differences between therapeutic and endogenous factor VIII. In contrast, mutations linked to inhibitor absence showed more similar predicted human leukocyte antigen (HLA)-binding profiles, suggesting reduced immune activation.

In conclusion, the study proposes a refined classification of factor VIII nonsense mutations based on their likelihood of wild-type readthrough and HLA-binding characteristics. This approach could allow graded prediction of inhibitor risk using both mutation type and HLA genotype, potentially supporting more personalised management strategies in haemophilia A.

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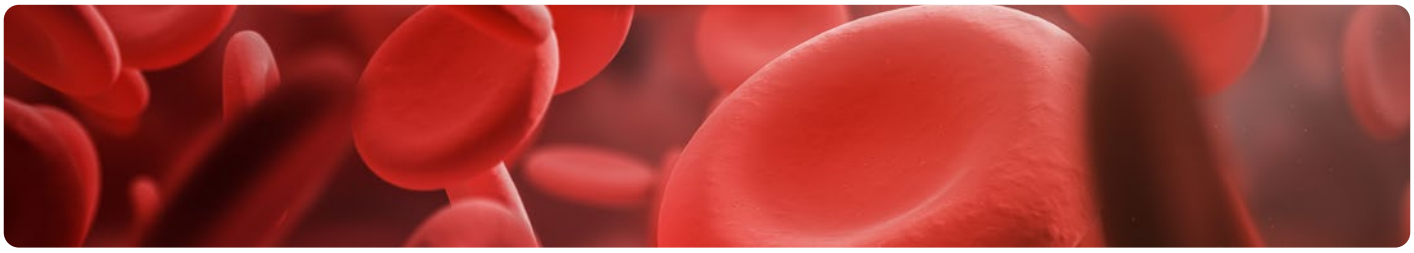


Emicizumab Enables Time-Limited Management in Acquired Haemophilia A

AT EAHAD 2026, investigators presented new real-world data suggesting that emicizumab provides effective, time-limited haemostatic control in patients with acquired haemophilia A (AHA), with treatment duration linked to inhibitor levels at diagnosis.[8]

AHA is a rare autoimmune bleeding disorder caused by inhibitory autoantibodies targeting endogenous factor VIII. Approximately half of cases are associated with an underlying condition, such as cancer, autoimmune disease, or the postpartum state. Standard management has traditionally involved two parallel strategies: haemostatic control with bypassing agents and eradication of the inhibitory autoantibodies using immunosuppressive therapy. However, these approaches are associated with substantial mortality, driven by infection related to immunosuppression and persistent bleeding complications.

The introduction of emicizumab has transformed the therapeutic landscape, offering effective bleed prevention and potentially reducing the need for aggressive early immunosuppression. While its role as first-line haemostatic therapy is increasingly established, questions remain regarding optimal treatment duration and long-term management.

To address this, researchers conducted a retrospective, multicentre observational study including 34 patients diagnosed with AHA between January 2020–October 2025. The median age was 74 years, and 47% of patients presented with major bleeding at diagnosis. Median activated partial thromboplastin time was 83.8 seconds, median factor VIII levels were 1%, and median inhibitor titre was 53 Bethesda units/mL.

An underlying aetiology was identified in 47% of cases, including cancer in 29% of patients.

Over a median follow-up of 2.1 years, no recurrent bleeding events were observed after initiation of emicizumab, confirming its effectiveness as a haemostatic agent in this setting. Importantly, treatment was not indefinite: the median duration of emicizumab therapy was 149 days, reflecting successful inhibitor eradication and treatment discontinuation in most patients.

A key finding was the significant association between inhibitor titre at diagnosis and duration of emicizumab therapy. Higher inhibitor levels at diagnosis were linked to longer treatment duration, suggesting that baseline inhibitor burden may help guide decisions on when to safely discontinue therapy. No significant association was observed between the type of immunosuppressive regimen and emicizumab duration, although heterogeneity in immunosuppressive approaches and the retrospective design may have introduced bias.

In summary, these data reinforce emicizumab as an effective and time-limited haemostatic strategy in AHA. Baseline inhibitor levels may serve as a practical marker to individualise treatment duration, supporting a more tailored and potentially safer long-term management approach in this high-risk population.

Inhibitor Risk Across FVIII Concentrates in Previously Untreated Patients

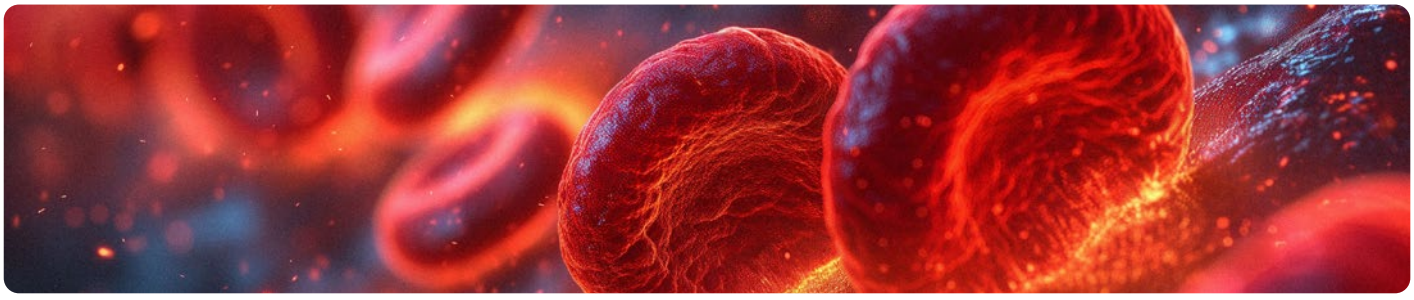
THIS analysis from the PedNet Registry, presented at EAHAD 2026, evaluated the risk of inhibitor development associated with individual factor VIII (FVIII) concentrates in previously untreated patients (PUP) with severe haemophilia A. Replacement therapy with FVIII is essential for the treatment and prevention of bleeding episodes; however, the development of neutralising inhibitors complicates treatment in approximately 30% of patients during the first 50 exposure days (ED). Understanding inhibitor risk across specific FVIII products is therefore important, particularly as FVIII remains the most effective therapy for breakthrough bleeding and surgical procedures.[9]

PUPs with severe haemophilia A born between 2000–2024 were followed until inhibitor development or until 50 EDs. The study compared inhibitor risk across different classes of FVIII concentrates, including plasma-derived FVIII and recombinant FVIII (rFVIII), as well as standard half-life and extended half-life recombinant products. Multivariate Cox regression analysis was performed to compare risks while adjusting for differences in follow-up and other known risk factors for inhibitor development, including prophylaxis with emicizumab. Results were reported as rate ratios (RR) with 95% CI.

A total of 1,503 PUPs were included in the analysis. Inhibitors developed in 444 patients after a median of 12 EDs, corresponding to a cumulative incidence of 31.0% (95% CI: 28.6–33.4%). Compared with standard half-life rFVIII products, the risk of inhibitor development was similar for plasma-derived FVIII (RR: 0.89; 95% CI: 0.69–1.14) and extended half-life rFVIII (RR: 1.10; 95% CI: 0.75–1.61).

Nine individual FVIII concentrates used by at least 40 PUPs were analysed. Advate (Takeda Pharmaceutical Company, Tokyo, Japan; n=392) served as the reference product because it had the largest available dataset. Two concentrates showed a significantly increased inhibitor risk: Kogenate FS (Bayer, Berkeley, California, USA)/Helixate NexGen (CSL Behring, King of Prussia, Pennsylvania, USA; 307 PUPs; RR: 1.40; 95% CI: 1.07–1.82; p=0.013) and Fanhdi (Grifols, Barcelona, Spain; 50 PUPs; RR: 1.72; 95% CI: 1.11–2.68; p=0.024).

Overall, inhibitor incidence was comparable across FVIII product classes, although increased risk was observed for Kogenate FS/Helixate NexGen and, for the first time, the plasma-derived product, Fanhdi. Despite changing treatment patterns in the era of emicizumab, the PedNet registry will continue monitoring inhibitor risk associated with individual FVIII concentrates.



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