

**Global Thrombosis  
Forum  
High School Scholars'  
Day  
Friday, July 25th,  
2025**

12:00pm - 5:00pm CST  
Loyola University Chicago  
Health Science Division  
Stritch School of Medicine  
Room #160  
2160 S 1st Ave, Maywood, IL 60153  
[https://us02web.zoom.us/j/555633853  
?omn=82674393341](https://us02web.zoom.us/j/555633853?omn=82674393341)



We are pleased to report that, consistent with the objectives of the International Union of Angiology and Loyola University Chicago, as well as its training and educational initiatives, the Global Thrombosis Forum, under the leadership of Dr.'s Jawed Fareed and Atul Laddu have made a significant impact in inspiring the public sector, particularly younger students, to promote awareness of thrombosis and its management at various levels. Today, we are celebrating the 10th annual High School Scholars' Day. Over the past 13 years, such programs have recruited talented younger scholars who have participated in educational and translational research programs in an exemplary manner. Since its inception, the Global Thrombosis Forum has collaborated with Loyola University Chicago on various initiatives aimed at promoting these activities. The summer research scholars' programs at various institutions, including Loyola University Chicago, have provided a significant platform for younger students to conduct translational research projects, resulting in participation in national and international meetings as well as publications. I thank the faculty members associated with hemostasis and thrombosis research at Loyola University Chicago for their continuous support in managing the High School Scholars program. Under the strong leadership of Professor Pier Luigi Antignani, Honorary President of the IUA, and Dr. Atul Laddu, President and CEO of GTF, these projects will continue to expand and will provide opportunities for younger students, which have already helped our scholars in achieving their goals in becoming MDs and scientists and have been helpful in their career planning and education to become physicians and scientists to serve healthcare and biomedical research programs. The Global Thrombosis Forum has facilitated interaction among ACCP, AC Forum, ASH, AVF, EVF, ISTH PERT, THSNA, and the

Brazilian Society of Vascular Medicine, and expanded the training program for scholars. We are thankful to Dr. Ramacciotti, Professor Komlos, and Professor Mansilha for their support. The parents of the scholars have supported their children's participation and should be commended for their efforts. We thank Dean Marzo for the support, Vice Provost Dr. Singh for facilitating, and Prof. Eva Wojcik for promoting the activities of this High School Scholars program.

## **AGENDA**

### **Poster Presentations**

**12:00 – 1:00 pm – Lunch and  
Poster presentation SSOM 160**

#### ***Women in Science***

Presenters: Naina Manoj, Yukta Borse  
Mentor: Sonika Tatipalli

#### ***CAT***

Presenters: Aahana Borkar, Aarav Gupta  
Mentor: Atul Laddu, MD

#### ***d-Dimer***

Presenters: Harshika Lekkala  
Mentor: Atul Laddu, MD

### **Welcome Addresses:**

**1:00 pm - 1:10 pm**

Pier Luigi Antignani, MD  
(Honorary President IUA)

**1:10 pm - 1:20 pm** - Welcome to GTF Interns-Dr. Eva Wojcik, Helen M. and Raymond M. Galvin Professor of Pathology and Laboratory Medicine and Urology, Chair, Department of Pathology and Laboratory Medicine.

**1:20 pm - 1:35 pm** - Dr. Tent Reed: Action & Attitude: The Power of Your Plus

### **Medical Student Presentation:**

**1:35 pm - 1:50 pm** - Dysregulation of fibrinolysis and its relationship with blood cellular indices in acute pulmonary embolism.

Presenter - Mira Nigudkar

**1:50 pm – 2:05 pm** - Inflammation-Induced Endothelial Dysfunction and Anti-PF4 Antibodies in Thromboinflammation of Acute Pulmonary Embolism.

Presenter - Marty Lundy

**2:05 pm – 2:15 pm** - Relevance of Annexin V with Coagulation Factors X and V in Pulmonary Embolism Patients.

Presenter - Aryan Mathur (Undergraduate Student)

**2:15 pm – 2:20 pm – Discussion**

**2:20 pm – 2:40 pm – Coffee Break**

### **Internship Research Presentations:**

**2:40 pm – 2:50 pm** Thrombogenic Mechanisms in Thalassemia

Presenter - Preston Reed

**2:50 pm – 3:00 pm** - Hypercoagulable state in Thalassemia.

Presenter - Divyanka Kavadikar (GTF Intern)

**3:00 pm – 3:10 pm** - Thrombotic complications in sickle cell anemia.

Presenter - Bhakti Singh (GTF Intern)

**3:10 pm – 3:20 pm** - Factor Xa Inhibitors: Mechanisms, Clinical Complications, and Advances in Reversal Strategies.

Presenter - Keerti Chakravarthy (GTF Intern)

**3:20 pm – 3:30 pm** - Factor IIa Inhibitors: Mechanisms, Clinical Complications, and Advances in Reversal Strategies.

Presenter - Parnika Tanguturu (GTF Intern)

**PowerPoint Presentations:**

**3:30 pm – 3:40 pm** - Elevation of d-Dimer without evidence of Venous Thromboembolism.

Presenters: Shravani Vedak, Siddarth Ranjan

Mentor: Atul Laddu, MD

**3:40 pm – 3:50 pm** - Worldwide Heparin Shortage

Presenters: Aditi Jadhav, Parnika Tanguturu

Mentor: Gitika Aggarwal, MD

**3:50 pm – 4:00 pm** - Immune

Thrombocytopenic Purpura

Presenters: Riddhi Surve, Riya Daftari

Mentor: Ashok Nahar, MD

**High School Student Presentations**

**4:00 pm – 4:05 pm** - Studies on comparison of bovine and porcine heparin for mediation

Presenter: Gia Kapur

**4:05pm – 4:10pm** – Comparative Studies on a Novel Collagen Peptide (CRPa) with other agonists

Presenter: Yash Gupta

**4:10 – 4:20pm - Invited Discussion**

**Closing Remarks**

**4:20 pm - 4:30 pm** - Closing Remarks by Dr. Jawed Fareed and Dr. Atul Laddu.

# **ABSTRACTS**

## **GTF Posters:**

### **1. Women in Science: Naina Manoj, Yukta Borse**

Throughout history, women have struggled and faced much opposition in holding positions in scientific or mathematical fields. They were denied the same opportunities and resources as men. Many believed women were not capable of understanding or working in these areas. Women in science have made significant contributions throughout history. However, less than 30% of the world's researchers are women. Celebrating the successes of women in science can serve as an inspiration to both women and men. The scientists profiled here have found different areas in which to focus their talents. Celebrating the successes of women in science can serve as an inspiration to both women and men. Our goal was to research the data on women in science in industry, conferences, publications, and leadership, identify reasons for barriers to women in science since we felt that this is a significant issue that does not often receive adequate attention, and develop innovative strategies to promote the entry, recruitment, retention, and sustained advancement of women in biomedical and research careers. We have also added impressive data from our experience over the past 12 years in GTF, and we're seeing a strong representation of female participation in all areas.

### **2. CAT: Aahana Borkar, Aarav Gupta**

Cancer-associated thrombosis (CAT) is the development of blood clots in cancer patients and is a leading cause of morbidity and mortality in oncology patients. Approximately 20% of all VTE events occur in individuals with cancer, and patients with malignancy have a four- to seven-fold increased risk of thrombosis compared to the general public. The heightened risk stems from factors including tumor cell secretion of procoagulant substances (e.g., tissue factor), chemotherapy, immobility, and inflammatory responses that promote a higher tendency to create clots. VTE risk is particularly elevated within the first 6 months following a cancer diagnosis. Despite advances in therapy, CAT remains a major clinical challenge due to its recurrence, bleeding risks with anticoagulation, and its contribution to early cancer-related death. CAT commonly occurs in patients with clinically active malignancy; however, there is a subset of patients in whom thrombosis can be the first manifestation of their cancer. The correlation between malignancy and thrombosis has been reported as early as 1823. The relationship between cancer and Thrombosis was first reported by Bouilland in 1823 and Armand Trousseau later in 1865. Overall, cancer patients comprise 15-20% of patients who are diagnosed with VTE, and this percentage has risen steadily over the years. Thrombosis has become the second leading cause of death in cancer patients.

### **3. d-Dimer: Harshika Lekkala**

D-dimer (DD) is a protein fragment in every person's blood. It is a soluble fibrin degradation product resulting from the fibrinolytic system's breakdown of thrombi. DD serves as a valuable marker of coagulation and fibrinolysis activation. Elevated DD levels may indicate an increased risk of blood clotting or a recent clot. A degradation product of cross-linked fibrin, which is the protein that forms the meshwork of a blood clot. Normal DD level is considered less than 0.50 µg/mL. DD has also been evaluated to determine the optimal duration of anticoagulation in VTE patients, to diagnose and monitor disseminated intravascular coagulation, and to aid in the identification of medical patients at high risk for VTE. The quantification of DD levels thus serves a vital role in guiding therapy.

## **GTF Presentations:**

### **1. Elevation of d-Dimer without evidence of Venous Thromboembolism: Shravani Vedak, Siddarth Ranjan**

Venous Thromboembolism (VTE) is the leading cause of morbidity and mortality among hospitalized patients. Several factors, including the extent of thrombosis and fibrinolytic activity, play a crucial role in influencing the sensitivity and specificity of D-dimer testing. A typical D-dimer level is less than 0.50 ng/L. The D-dimer test is highly sensitive (>95%) in DVT and PE. Upon presentation, patients with high D-dimer levels should prompt a more intense diagnostic approach, irrespective of pretest probability.

We present here a case of an 84-year-old male who suffered from pneumonia and significantly elevated D-dimer levels but with no evidence of DVT or PE to find the relationship between elevated D-dimer levels and the presence of evidence of VTE, such as DVT or PE.

### **2. Worldwide Heparin Shortage: Aditi Jadhav, Parnika Tanguturu**

Heparin is a hundred-plus-year-old anticoagulant still used in therapy. It is an effective and relatively safe drug. Despite the development of several other anticoagulants in recent years, heparin has held its place in hospitals and pharmacies. In recent years, disturbing news has emerged about a global heparin shortage due to African Swine Fever, which has caused physicians and pharmacists to be naturally scared. In this presentation, the authors researched the various aspects of the global heparin shortage. Each party involved, such as the FDA, manufacturers, physicians, and pharmacists, must play a synergistic role in preventing and combating this global shortage of a life-saving anticoagulant.

### **3. ITP: Riddhi Surve, Riya Daftari**

ITP is an immune disorder marked by low platelet count due to destruction of platelets or impairment in platelet production. ITP can be acute or chronic, occurs in all ages, and ranges in severity. Adult cases are more likely to be chronic. The patient may present with petechiae or mucocutaneous bleeding. The authors present a literature review with a focus on pathophysiology, mechanism of action, clinical presentation, treatment (current and investigational), and prognosis of ITP.

## **1. Hypercoagulable state in Thalassemia: Divyanka Kavadihar, GTF Intern**

Thalassemia is an inherited hemolytic anemia that affects the production of hemoglobin, and is prevalent typically in people of Southeast Asian, Mediterranean, and African American descent. Thalassemia is a condition that affects the production of hemoglobin, causing defective alpha or beta globin chains, resulting in either alpha or beta thalassemia.

Thalassemia patients are at higher risk for contracting thrombotic complications, due to damage in their red blood cells activating the intrinsic pathway of the coagulation cascade. Biomarkers such as thromboxane metabolites, platelet life spans, coagulation inhibitors, and coagulation factors confirm the existence of a chronic hypercoagulable state in thalassemic patients. While treatments such as chronic blood transfusions and chelation therapy do exist, more modern therapies are coming to light, such as fetal hemoglobin agents and autologous gene therapy which target thalassemia in different ways, especially focusing on erythropoiesis.

Over the last decade, life expectancy of thalassemia sufferers has markedly improved. However, some complexities such as thalassemia-associated thrombosis have been identified in patients presenting with a hypercoagulable state. Increased platelet activation, expression of P-selectin and CD63, increased levels of endothelial adhesion proteins, decreased levels of natural anticoagulants (protein C, protein S, antithrombin III) all contribute to the increased hypercoagulability. The high risk of thrombotic complications in thalassemia cases is well accepted, however the precise mechanisms responsible for this remain largely unknown. It is important to elucidate the exact mechanisms of thrombotic state in thalassemia to help improve therapeutic measures and healthcare.

## **2. Thrombotic complications in sickle cell anemia: Bhakti Singh, GTF Intern**

Sickle cell anemia (SCA) is an inherited blood disorder producing abnormal hemoglobin S (HbS) that distorts red blood cells into rigid, sickle shapes. These cells block vessels, shorten cell lifespan, and cause chronic anemia, pain crises, and increased thrombotic risk. Several thrombotic complications occurring in these patients have been reported.

Thrombosis in SCA represents a complex pathological process contributing to the disease's

morbidity and mortality. SCA patients suffer from hypercoagulable state driven by chronic hemolysis, which releases free hemoglobin and heme, consuming nitric oxide (NO) and promoting oxidative stress that impairs endothelial function. TNF-alpha and other inflammatory mediators enhance adhesion and clot formation.

Despite this known information, the precise mechanisms leading to hypercoagulable state in SCA patients remains largely under established. Hence, it is crucial to understand these complex mechanisms leading to thrombotic complications in SCA, in order to help develop optimal treatment regimes. This presentation reviews the complex mechanisms promoting thrombotic complications in SCA and discusses the pathophysiology, complications, and

advances in targeted therapies to reduce thrombotic complications and improve patient outcomes.

### **3. Thrombogenic Mechanisms in Thalassemia: Preston Reed**

Thalassemia is one of the most common hereditary hemolytic anemias and is a fatal disease, typically occurring in populations from Southeast Asia, Mediterranean regions. Thalassemia is a disease characterized by the production of insufficient hemoglobin, caused by missing alpha or beta globin genes. One of the most profound complications for patients with thalassemia is thrombosis. Thalassemia-associated thrombosis have been identified in patients presenting with thrombotic complications, which has indeed caused a major burden on clinicians and patients. Even though this high risk of thrombotic complications in patients with intravascular haemolysis is well accepted, the exact mechanisms that are responsible for this hypercoagulable state remain under established.

This presentation will cover the pathophysiology of the hypercoagulable state. Specifically, exploring the impact that protein deficiencies, elevated tissue factor, phosphatidylserine, floating microparticles, platelet activation and changes in signaling pathways have on hypercoagulation. Uncovering the mechanisms of this hypercoagulable state is essential to unlocking therapies to combat this disease. This is critical to improving the lives of the millions of people living worldwide with this condition with an optimal therapeutic approach.

### **4. Studies on Comparison of Bovine and Porcine heparin for Mediation- Gia Kapur**

Heparin is a drug that prevents the formation of clots in blood. However, 3-5% of patients who receive heparin develop Heparin Induced Thrombocytopenia (HIT), a potentially fatal disease that coagulates blood in the presence of heparin. Currently, porcine heparin is the main source of this drug. However, due to an outbreak of African swine flu in pig herds, the US has been unable to attain enough batches of this drug as the herds are dying. The FDA is currently looking into other alternatives such as bovine heparin. The present experiment compares the level of aggregation that porcine and bovine heparin induce at different concentrations in HIT antibodies. The experiment used PAP8 machines which use light transmission aggregometry to detect the level of aggregation in each sample. The results of this experiment yielded that bovine heparin behaves similarly to porcine heparin at concentrations, 10, 1, and 0 ug/mL, but not at 0.1 ug/mL concentration. This suggests that bovine heparin is an acceptable candidate for FDA approval due to no added risk from using bovine over porcine heparin.

### **5. Relevance of Annexin V with Coagulation Factors X and V in Pulmonary Embolism Patients: Aryan Mathur**

Pulmonary embolism (PE) is a life-threatening condition caused by thrombi obstructing the pulmonary arteries. Annexin V, a marker of apoptosis and endothelial injury, has been associated with coagulation and vascular dysfunction, but its role in PE is not well

defined. Factor V and Factor X are key coagulation proteins, though their relationship with Annexin V in PE remains unclear. This study aimed to measure and compare plasma levels of Annexin V, Factor V, and Factor X in 65 PE patients and 15 healthy controls, and to assess possible relationships. Biomarker levels were measured using sandwich ELISAs and analyzed with GraphPad Prism. All markers, Annexin V, Factor V, and Factor X were significantly elevated in PE patients compared to controls. However, only minimal correlation was found between Annexin V and the coagulation factors, suggesting they may reflect distinct thrombo-inflammatory pathways. Notably, varying levels of these biomarkers were observed across different degrees of PE severity. These results suggest independent roles for Annexin V, Factor V, and Factor X in PE pathophysiology and support further investigation into their potential use as diagnostic or prognostic biomarkers.

## **6. Factor Xa Inhibitors: Mechanisms, Clinical Complications, and Advances in Reversal Strategies: Keerti Chakravarthy, GTF Intern**

Factor Xa (FXa) inhibitors, such as apixaban, rivaroxaban, edoxaban, and betrixaban, are direct oral anticoagulants (DOACs) that selectively inhibit FXa, a crucial enzyme in the coagulation cascade responsible for thrombin generation. These agents are widely used for stroke prevention in atrial fibrillation and treatment of venous thromboembolism, for which they provide effective and convenient anticoagulation. Despite their benefits, they carry a risk of serious bleeding. For reversal of FXa inhibitor-associated bleeding, andexanet alfa (AA)—a recombinant modified FXa decoy protein—is the only FDA-approved agent specifically for apixaban and rivaroxaban reversal. AA works by binding and neutralizing FXa inhibitors, rapidly restoring coagulation, but may increase the risk of thrombosis and is associated with high costs. Alternatively, 4-factor prothrombin complex concentrates (4F-PCCs), used off-label, supply vitamin K-dependent clotting factors to support hemostasis, with variable efficacy. Additionally, ciraparantag, a small synthetic molecule under investigation, shows promise as a broad-spectrum reversal agent for multiple anticoagulants, including factor Xa (FXa) inhibitors. Prompt and appropriate reversal strategy selection is essential to optimize outcomes in FXa inhibitor-associated bleeding.

## **7. Factor IIa Inhibitors: Mechanisms, Clinical Complications, and Advances in Reversal Strategies: Parnika Tanguturu, GTF Intern**

Factor IIa Inhibitors: Direct thrombin (Factor IIa) inhibitors, including dabigatran argatroban, and bivalirudin (Angiomax), directly inhibit thrombin, a central enzyme in the coagulation cascade responsible for converting fibrinogen to fibrin, activating platelets, and stabilizing clots. Dabigatran is an oral prodrug that is converted to its active form in the liver and selectively inhibits both free and clot-bound thrombin. It is commonly used for stroke prevention in atrial fibrillation and the treatment of venous thromboembolism. Argatroban is a synthetic, reversible parenteral thrombin inhibitor primarily used in patients with heparin-induced thrombocytopenia (HIT), while bivalirudin, a hirudin analog, is often used as an anticoagulant during percutaneous coronary intervention (PCI). While effective, these agents pose a risk of serious bleeding. For dabigatran-associated bleeding, idarucizumab, a monoclonal antibody

fragment, is FDA-approved for the rapid and specific reversal. In the absence of targeted reversal for parenteral agents, 4-factor prothrombin complex concentrates (4F-PCCs) may be used off-label to support hemostasis. Early recognition and prompt reversal are crucial for improving outcomes in direct thrombin inhibitor-associated bleeding.

## **8. Comparative Studies on a Novel Collagen Related Peptide Platelet Agonist: Yash Gupta**

Platelet function tests assess platelet traits to diagnose bleeding disorders and monitor antiplatelet therapy. Agonists like Collagen Related Peptide-A (CRP-A) are necessary for platelet aggregation in these tests. The present study compares the aggregation response of CRP-A (Pplus Medical Limited, Ireland) to standard agonists. Using platelet-rich plasma from healthy donors, maximum aggregation, primary slope, and onset time in response to different concentrations of CRP-A were compared to those achieved using standard agonists. Aggregation assays were performed using BioData's PAP-8 aggregometer. CRP-A at a concentration of 10µg/mL induced a comparable level of aggregation as 20µM ADP and 190µg/mL collagen. While the rate of aggregation was comparable among agonists, CRP-A's lag time was shorter than collagen. Solutions of CRP-A showed no loss of activity when stored at 4°C for 2 weeks. The study demonstrates that CRP-A is an effective platelet agonist with stability. Further studies to confirm its clinical utility are warranted