Trinity Health

Hypercoagulable states

Vladimir-Ducarmel Joseph, MD, MHA
Regional Provider Education Lead
Clinical Documentation Improvement
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About me

Vladimir-Ducarmel Joseph, MD, MHA

- CDI experience in both For-profit and Not-For-profit organizations.
- Regional Physician CDI Education Lead for Trinity Health Of New England.
- Special areas of interest include Physician Leadership, Team Dynamics, Clinical Documentation Improvement and Education.

Objectives

1. Provide clinical overview of hypercoagulable states and related clinical indicators
2. Understand and describe atrial fibrillation as a hypercoagulable state
3. Understand and describe the CDI and coding implications

Plan

1. Introduction & overview
2. Overview of hemostasis & Virchow's triad
3. Primary hypercoagulable state: common examples
4. Secondary hypercoagulable state: common examples
5. Atrial fibrillation: a hypercoagulable state
6. Diagnosis & treatment
7. CDI, coding implications & clinical indicators
8. Key takeaways

How it usually begins…

Provider: My favorite CDS! How are you?

CDIS: Good morning Dr. X. I saw that you answered the query on Mrs. B. Thank you.

Provider: Yeah. That HCS query? You know that the patient has afib, right? That's why she is on Coumadin.

CDIS: You are treating the afib with the Coumadin?

Provider: Not the afib per se. I am preventing DVT in this patient given her elevated risk of developing a clot from her persistent afib combined with her DM and very low ejection fraction heart failure.

CDIS: Do you think all these risk factors present in this patient put her in a state where she becomes prone to developing clots?

Provider: Well, you are rephrasing what I just said.

CDIS: Does that mean this patient is in a hypercoagulable state?

Provider: Come to think of it, you may have a point. Tell me more about HCS in this context.

Objective #1

1. **Provide clinical overview of hypercoagulable states and related clinical indicators**
2. Understand and describe atrial fibrillation as a hypercoagulable state
3. Understand and describe the CDI and coding implications

Introduction & overview

Patients are considered to have a hypercoagulable state if they have laboratory abnormalities or clinical conditions that are associated with increased risk of thrombosis.

The hypercoagulable states are a group of acquired and inherited disorders that increase the risk of abnormal development of blood clots.

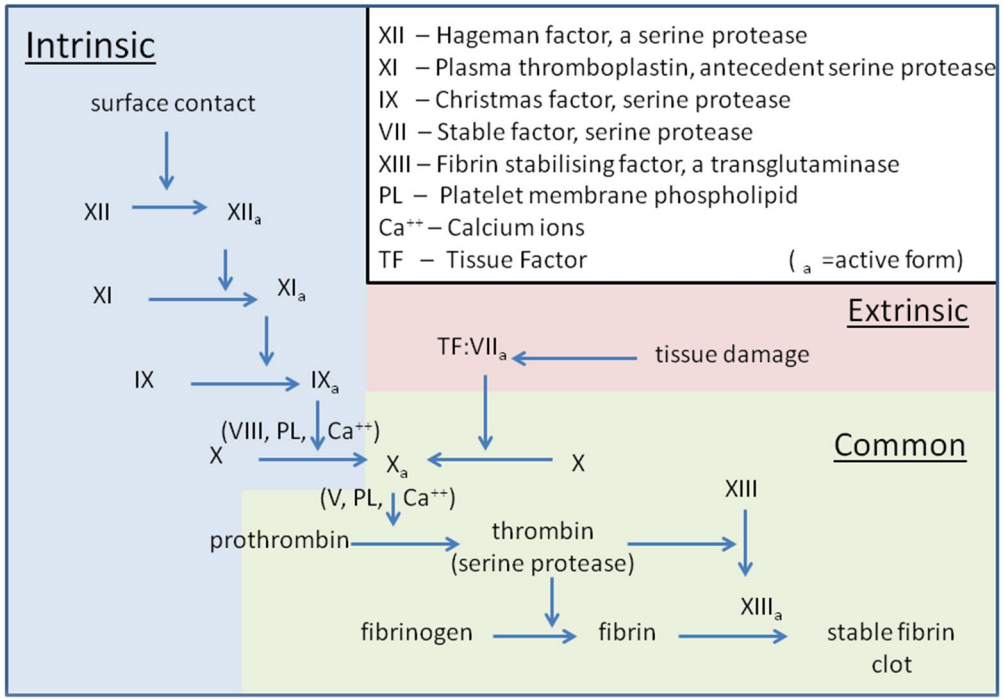
They are divided into:

- primary or inherited hypercoagulable states
- secondary or acquired hypercoagulable states.

Reference: The Hypercoagulable States by Schafer AI. https://www.ncbi.nlm.nih.gov/pubmed/3158262

Normal hemostasis

The three pathways that makeup the classical blood coagulation pathway



Normal hemostasis

3 pathways:

- Tissue factor or extrinsic pathway
- Contact activation or intrinsic pathway
- Common pathway

Cofactors:

- Calcium & phospholipid
- Vitamin K

Normal hemostasis

Regulators:

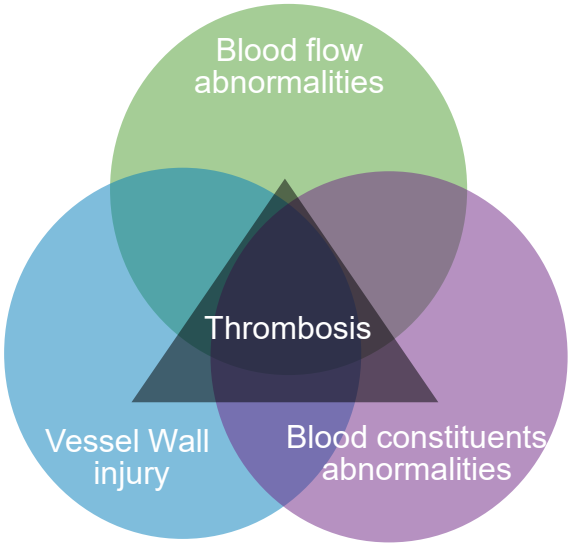
- Protein C
- Antithrombin
- Tissue factor pathway inhibitor
- Plasmin
- Prostacyclin (PGI2)

Virchow's triad

A major theory delineating the pathogenesis of venous thromboembolism.

Pathogenesis of VTE:

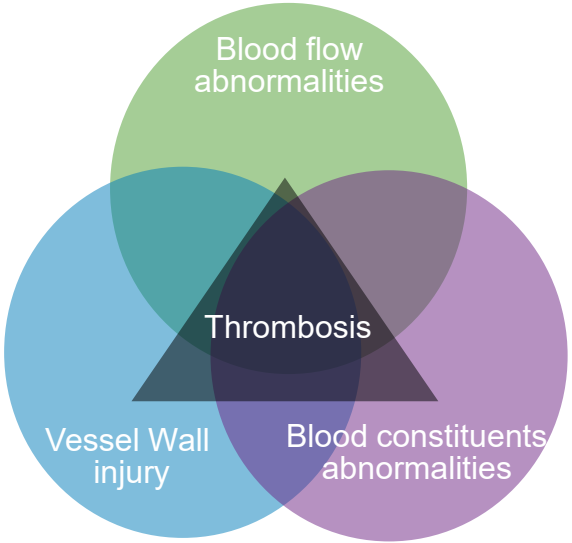
- Alterations in blood flow (i.e. stasis)
- Vascular endothelial injury
- Alterations in the constituents of the blood
  (i.e. inherited or acquired hypercoagulable state)



Reference: Overview of the causes of venous thrombosis by Bauer & Lip, 2019

Virchow's triad

- A risk factor for thrombosis can now be identified in over 80 percent of patients with venous thrombosis.
- There is often more than one factor at play in a given patient.
- Many patients with VTE fulfill most or all of Virchow's triad of stasis, endothelial injury, and hypercoagulability.



Reference: Overview of the causes of venous thrombosis by Bauer & Lip, 2019

Primary Hypercoagulable States

Primary hypercoagulable states are inherited disorders of specific anticoagulant factors. The most frequent causes of an inherited (primary) hypercoagulable state are:

- The factor V Leiden mutation
- The prothrombin gene mutation
- Together, they account for 50 to 60 percent of cases.

Primary Hypercoagulable States

The following three account for most of the remaining cases:

- Defects in protein S
- Defects in protein C
- Defects in antithrombin (formerly known as antithrombin III)

Primary Hypercoagulable States

Other alleged inherited thrombophilia. It remains unclear whether some of these disorders are actually associated with an increased risk of venous thrombosis:

- Heparin cofactor II deficiency
- Plasminogen deficiency
- Dysfibrinogenemia
- Factor XII deficiency

Secondary Hypercoagulable States

Secondary hypercoagulable states are primarily acquired disorders that predispose to thrombosis through complex and multifactorial mechanisms. These involve blood flow abnormalities or defects in blood composition and of vessel walls. Common examples include:

- **Previous thromboembolism.** Previous thrombotic events are a major risk factor for recurrent VTE. In an outpatient prospective cohort study, the risk of recurrence after an acute episode of venous thrombosis was 18, 25, and 30 percent at two, five, and eight years, respectively. In a community-based epidemiologic study, a previous history of VTE conferred a relative risk (RR) of 7.9 for VTE recurrence.
- **Malignancy.** Patients with cancer often have a hypercoagulable state due to the production of substances with procoagulant activity (e.g. tissue factor and cancer procoagulant). Clinical VTE occurs in approximately 15 percent of such patients and is common cause of serious clinical outcomes. Approximately 20 percent of patients with symptomatic deep venous thrombosis have a known active malignancy

Reference: Overview of the causes of venous thrombosis by Bauer & Lip, 2019

Secondary Hypercoagulable States

More examples:

- **Pregnancy.** Pregnancy is associated with an increased risk of thrombosis that may be due in part to obstruction of venous return by the enlarged uterus, as well as the hypercoagulable state associated with pregnancy.
- **Certain Drugs:**
  - Oral and transdermal contraceptives
  - HRT
  - Testosterone
  - Tamoxifen
  - Bevacizumab
  - Glucocorticoids
  - Antidepressants
- **Prolonged Immobilization** (prolonged sitting, extended travel)
- **Surgery.** Thrombotic risk is greatly increased during surgery, particularly orthopedic, major vascular, neurosurgery, and cancer surgery.

Reference: Overview of the causes of venous thrombosis by Bauer & Lip, 2019

Secondary Hypercoagulable States

More examples:

- **Major trauma.** The mechanisms of activation of the coagulation system following surgery or trauma are incompletely understood as of this presentation, but may include decreased venous blood flow in the lower extremities, diminished fibrinolysis, immobilization, the release or exposure of tissue factor, and depletion of endogenous anticoagulants such as antithrombin.
- **Antiphospholipid antibodies.**
- **Renal diseases** (ESRD, nephrotic syndrome, renal transplantation). To better illustrate, using the Healthcare Cost and Utilization Project's Nationwide Inpatient sample, the annual frequency of pulmonary embolism in adults with end-stage renal disease, chronic kidney disease, and normal renal function was 527, 204, and 66 per 100,000 persons.
- **Liver disease**
- **Cardiovascular disease** (heart failure with very low EF, atrial fibrillation etc.)

Reference: Overview of the causes of venous thrombosis by Bauer & Lip, 2019

Secondary Hypercoagulable States

Many patients with an episode of VTE have more than one acquired risk factor for thrombosis. This was shown in a population-based study of the incidence of VTE in residents of Worcester, MA during 1999. The six most prevalent pre-existing medical characteristics of patients in this study were:

- More than 48 hours of immobility in the preceding month (45%)
- Hospital admission in the past three months (39%)
- Surgery in the past three months (34%)
- Malignancy in the past three months (34%)
- Infection in the past three months (34%)
- Current hospitalization (26%)

Only 11 percent of the 587 episodes of VTE had none of these six characteristics present, while 36 and 53 percent had 1 to 2 and ≥3 risk factors, respectively.

Reference: Overview of the causes of venous thrombosis by Bauer & Lip, 2019

Objective #2

1. Provide clinical overview of hypercoagulable states and related clinical indicators
2. **Understand and describe atrial fibrillation as a hypercoagulable state**
3. Understand and describe the CDI and coding implications

Atrial fibrillation: a hypercoagulable state

Several studies that measured indices of coagulability suggest that atrial fibrillation confers a hypercoagulable state, increasing the risk of thromboembolism and stroke.

The pathophysiology of thromboembolism in atrial fibrillation is multi-factorial but increasing evidence points to the fulfillment of Virchow's triad in this arrhythmia, leading to a prothrombotic or hypercoagulable state.

Consideration must be given to the patient's age, the presence of structural heart disease, and clinical risk factors such as previous hypertension, diabetes, various biomarkers, or heart failure (particularly depressed left ventricular systolic function).

Reference: Mechanisms of thrombogenesis in atrial fibrillation by Lip, 2017

Atrial fibrillation: a hypercoagulable state

Evidence of flow abnormalities is provided by the loss of atrial systole in atrial fibrillation that results in increased stasis of blood within the left atrium. Fast ventricular rate, which is often a consequence of that arrhythmia, reduces effective ventricular filling and further worsens intra-atrial stasis.

Evidence of blood constituents abnormalities is provided by increased platelet activation in atrial fibrillation has been provided by numerous studies.

Evidence of vessel wall abnormalities in AF is provided by the following observations:

Reference: Atrial Fibrillation and the Hypercoagulable State: From Basic Science to Clinical Practice

Atrial fibrillation: a hypercoagulable state

- Endocardial damage and disorganization of the left atrial appendage endocardium has been described in the setting of mitral valve disease, especially where atrial fibrillation is present.
- Abnormal plasma indices of endothelial damage/dysfunction, such as vWf, which have been related to thrombogenesis, stroke risk, and adverse prognosis.
- Increased levels of circulating endothelial cells (CECs), an index of endothelial damage in the setting of AF and target organ damage (heart failure, stroke, myocardial infarction).

Reference: Atrial Fibrillation and the Hypercoagulable State: From Basic Science to Clinical Practice

Diagnosis

The diagnosis of hypercoagulable state is based on clinical judgement and evaluation of the patient. The tests that are ordered based on the history and examination of the patient. These tests can include:

- CBC
- PT
- PTT
- Thrombodynamics test
- Thrombin time
- Lupus anticoagulant
- Anti-cardiolipin antibody
- Anti-B2 glycoprotein-1 antibody
- Protein C resistance
- Fibrinogen tests
- Factor V Leiden
- Homocysteine levels
- Prothrombin mutations

Treatment

The treatment depends on the underlying cause. Regardless of the cause, the treatment will involve the use of anticoagulants such as heparin, warfarin, direct thrombin inhibitors (Argatroban), Xarelto, etc.

- In primary cases, the treatment is usually a maintenance therapy as there is no cure as of this presentation.
- In secondary cases, an effective correction of the underlying cause (nephrotic syndrome, atrial fibrillation, etc.) will reduce the associated risks.

Objective #3

1. Provide clinical overview of hypercoagulable states and related clinical indicators
2. Understand and describe atrial fibrillation as a hypercoagulable state
3. **Understand and describe the CDI and coding implications**

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CDI & coding implications

Hypercoagulable state (primary or secondary), when documented in the medical record is a CC and can, therefore, impact the length of stay, reimbursement, severity of illness and risk of mortality.

Secondary hypercoagulable state is often under documented and underreported. Provider education is key in dealing with this issue.

CDI & coding implications

Remember

Documentation of secondary hypercoagulable state must meet the definition of a secondary diagnosis. In other words, it must, at least, include one of the following:

- Clinical evaluation
- Treatment
- Diagnostic tests & procedures
- Prolonged length of stay
- Increase in nursing care and/or monitoring

CDI & coding implications

What to look for:

- History (personal and/or family)
- Presence of risk factors
- Abnormal lab values (i.e. abnormal coagulation profile)
- Specific workup (i.e. hypercoagulable workup)
- Anticoagulant therapy

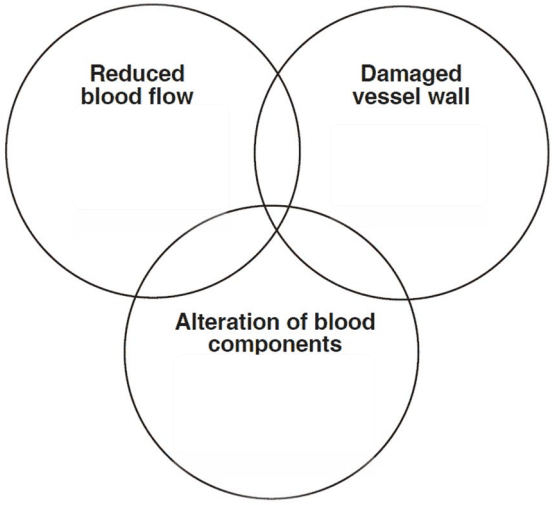
FAQs

How come afib is a hypercoagulable state?

The pathophysiology of thromboembolism in atrial fibrillation is multi-factorial but increasing evidence points to the fulfillment of Virchow's triad in this arrhythmia, leading to a prothrombotic or hypercoagulable state.

In afib, we have:

1. Stasis in the atrium
2. Evidence of damage to the endothelium
3. Evidence of increased platelet activation



FAQs

The anticoagulant was given as a prophylactic intervention. We cannot code hypercoagulable state, can we?

The prophylactic intervention is regarding VTE. You will not code DVT for example since the patient has not developed it (yet.) You can, however, code the HCS if documented and supported by the provider since the patient has already developed it.

Key takeaways

At this point, you should know that:

- Hypercoagulable states can be either primary/inherited or secondary/acquired
- Secondary hypercoagulable state is underdocumented and underreported
- Atrial fibrillation confers a reversible secondary hypercoagulable state
- The documentation of secondary hypercoagulable state must meet the definition of a secondary diagnosis
- In the presence of convincing clinical indicators, a query can be generated

Questions & comments

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Reference

- Overview of the causes of venous thrombosis by Bauer, MD and Lip, MD available here: https://www.uptodate.com/contents/overview-of-the-causes-of-venous-thrombosis?search=virchow%20triad&source=search_result&selectedTitle=1~13&usage_type=default&display_rank=1
- “Hypercoagulability syndromes” available here: https://jamanetwork.com/journals/jamainternalmedicine/fullarticle/649434
- Mechanisms of thrombogenesis in atrial fibrillation by Gregory YH Lip, MD. Taken from: https://www.uptodate.com/contents/mechanisms-of-thrombogenesis-in-atrial-fibrillation#H9
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- Is the hypercoagulable state in atrial fibrillation mediated by vascular endothelial growth factor? by Chung N. et al. Found in Stroke September 2002, Volume 33, Issue 9.

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- Persistence of Hypercoagulable State after Resection of Intra-Abdominal Malignancies by Thorson, MD; Van Haren, MD; Ryan, MD; Curia, MD; Sleeman, MD,FACS; Levi, MD,FACS, Livingstone, MD, FACS; and Proctor, PhD. Found in Journal of the American College of Surgeons, Volume 216, Issue 4, April 2013, Page 589-590.
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- The Hypercoagulable States by Schafer AI; Ann. Intern. Med. June1985. Found here https://www.ncbi.nlm.nih.gov/pubmed/3158262

Thank you

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