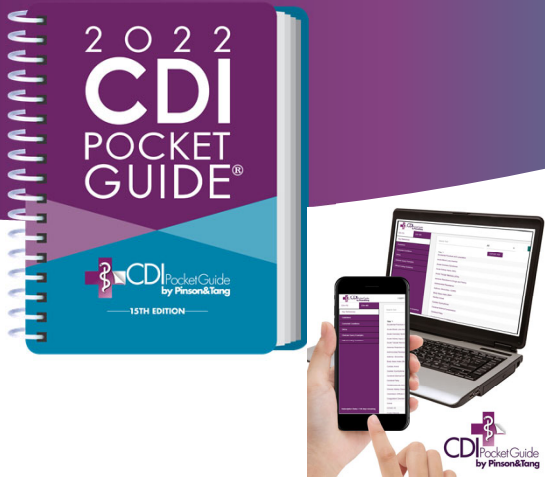


January 27, 2022




CDI Pocket Guide®
Coagulation Disorders

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About Us



Richard Pinson
MD, FACP, CCS, CDIP

Dr. Richard Pinson is a physician, educator, administrator, and healthcare consultant. He practiced Internal Medicine and Emergency Medicine in Tennessee for over 20 years having board certification in both.

Cynthia Tang
RHIA, CCS, CRC

Cynthia brings over 30 years of experience in coding and clinical documentation, health information management, and clinical resource management. For over 25 years she has traveled across the country implementing successful and sustainable coding and CDI programs in hundreds of hospitals.

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Coagulation Disorders

Pinson&Tang Agenda

2022 CDI Pocket Guide
Pages 105-108

Definition of Coagulation and
Coagulation Disorders

ICD-10 Classification

Hypo- and Hyper-coagulopathy

Laboratory Findings

Secondary Hypercoagulable State

Case Examples

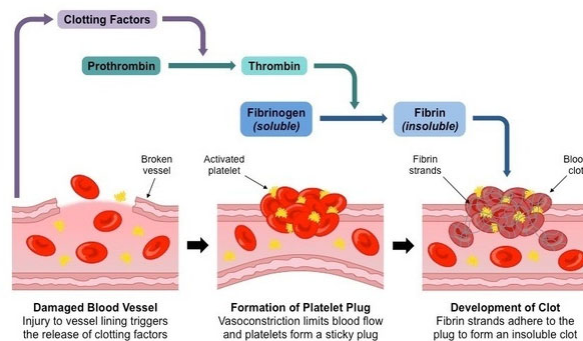
Q&A

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What is Coagulation?

Definition

A complex process whereby blood proteins, i.e., **clotting factors**, plug leaks in blood vessels and stop the loss of blood.



Blood vessels shrink to reduce the blood flow. Blood **proteins** and **platelets** activate around the wound and form a **fibrin** clot, which acts like a mesh to patch the leak and stop the bleeding.

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Coagulation (Clotting) Factors

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| | |
|------|---|
| I | Fibrinogen |
| II | Prothrombin |
| III | Tissue thromboplastin (tissue factor) |
| IV | Ionized calcium (Ca^{++}) |
| V | Labile factor or proaccelerin |
| VI | Unassigned |
| VII | Stable factor or proconvertin |
| VIII | Antihemophilic factor |
| IX | Plasma thromboplastin component, Christmas factor |
| X | Stuart-Prower factor |
| XI | Plasma thromboplastin antecedent |
| XII | Hageman factor |
| XIII | Fibrin-stabilizing factor |

Coagulation Factors are produced by the **liver**, except Factor IV.

Vitamin K is needed for the liver to produce Factors II, VII, IX, X.

Phospholipids are components of the cellular and platelet membranes and provide a surface for the chemical reactions of coagulation to take place.

What can disrupt the coagulation process?

- Low, elevated, or defective clotting factors
- Severe liver disease
- Vitamin K deficiency
- Antibodies from autoimmune disease (e.g., lupus) that inhibit clotting factors, i.e., antiphospholipid syndrome
- Elevated *intrinsic* circulating anticoagulants that inhibit clotting factors (e.g., anti-thrombin, anti-VIIIa)
- Elevated *extrinsic* circulating anti-coagulants (drug-induced).

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Coagulation Disorders

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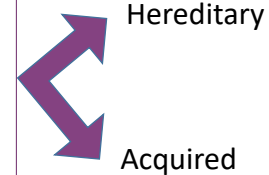
Definition

Disorder of the blood clotting factors and activities which disrupt the body's ability to control blood clotting.

Result: **Bleeding** or **Thrombosis**

Hypo-coagulopathy: Increased tendency toward bleeding (abnormal bleeding)

Hyper-coagulopathy: Increased tendency toward blood clotting (thrombosis)



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ICD-10-CM: Coagulation Defects (D65-D68)

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Hemorrhagic Disorders

Thrombotic Disorders

| Hereditary Hypocoagulopathy | | Hereditary Hypercoagulopathy | |
|-----------------------------|--|------------------------------|---|
| D66 | Hemophilia A (VIII) | D68.5- | Primary thrombophilia |
| D67 | Hemophilia B (IX) | D68.51 | Protein C resistance |
| D68.0 | Von Willebrand's | D68.52 | Prothrombin gene resistance |
| D68.1 | Hemophilia C (XI) | D68.59 | Other: Protein C deficiency, Antithrombin deficiency |
| D68.2 | Other (I, II, V, VII, X, XI...) | | |
| Acquired Hypocoagulopathy | | Acquired Hypercoagulopathy | |
| D68.311 | Acquired hemophilia | D68.61 | Antiphospholipid antibody |
| D68.312 | Lupus anticoagulant | D68.62 | Lupus anticoagulant |
| D68.318 | <i>Intrinsic</i> antibodies, anticoagulants, inhibitors | D68.69 | Other thrombophilia Secondary hypercoagulable state NOS |
| D68.32 | <i>Extrinsic</i> anticoagulants: drug-induced | | |
| D68.4 | Deficiency due to liver disease, Vitamin K | | |

| Other and Unspecified | |
|-----------------------|---|
| D65 | DIC (Disseminated intravascular coagulation) |
| D68.8 | Other specified coagulation defects |
| D68.9 | Coagulation defect, unspecified |

Not Coagulation Disorders

R79.1 Abnormal coagulation
profile

Z79.01 Long term use (current) of
anticoagulants

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Hypo-coagulopathy: Bleeding

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Hereditary

- Von Willebrand disease: VW factor deficiency or abnormality
- Hemophilia A: Clotting Factor VIII deficiency
- Hemophilia B: Clotting Factor IX deficiency (Christmas disease)

Acquired

- Drug-induced:
 - Anticoagulants (Coumadin, Eliquis, etc.) interfere with normal **clotting factors**
 - Antithrombotics (aspirin, Plavix, etc.) interfere with **platelet** function
- Liver disease: Causes decreased production of clotting factors
- Autoimmune inhibitors to clotting factors, e.g., lupus anticoagulant

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Laboratory Findings: PT/INR Hemorrhagic Disorders

Prothrombin is a clotting factor made by the liver.

- PT (prothrombin time)* test measures how long it takes for a clot to form in a blood sample.
- INR (international normalized ratio) is a calculation based on the PT.

PT/INR determines if the blood is clotting normally and measures the effectiveness of **Coumadin** for patients with atrial fib, thromboembolism, stroke, etc.

- High/Prolonged INR = Therapeutic or Bleeding

*Also known as protime, PT protime, PT/INR.



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| INR | Indicates |
|--------------|---|
| < 1.1 | Average level for a healthy person not currently on anticoagulation drugs |
| 1.1 to < 2.0 | Elevated; below the therapeutic range for patients on Coumadin therapy; may indicate liver disease |
| 2.0 to ≤ 3.0 | Therapeutic range for patients on Coumadin therapy |
| > 3.0 | Risk of excessive bleeding. Therapeutic level up to 3.5 for some patients with high risk for thrombosis or who have an older generation artificial heart valve. |

Other reasons for high INR: Inherited/acquired clotting factor deficiency (I, II, V, VII, X), DIC, Vitamin K deficiency, liver disease.

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Laboratory Findings: PTT/aPTT Hemorrhagic Disorders

PTT (Partial Thromboplastin Time): Time in seconds it takes plasma to clot and measures a different set of clotting factors than the PT.

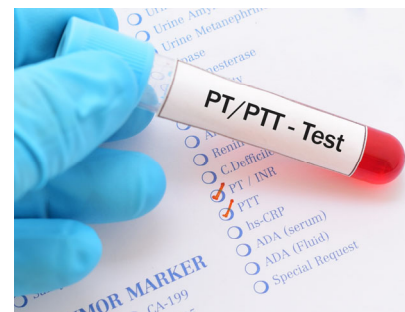
- Normal = 60-70 sec

aPTT (activated Partial Thromboplastin Time).

Same test as PTT, but an activator is added that speeds up the clotting time and test results.

- Normal = 30-40 sec.

PTT/aPTT is primarily used to measure the effectiveness of Heparin therapy. Patients on Heparin should have a PTT or aPTT 2-2.5 times the normal range.



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Laboratory Findings: INR, PTT Hemorrhagic Disorders

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| INR | PTT/aPTT | Acquired Conditions | Hereditary Conditions |
|----------|------------------------------|---|---|
| Elevated | Normal | Coumadin therapeutic range* or adverse effect or poisoning Liver disease Vitamin K deficiency | Factor VII deficiency |
| Normal | Prolonged | Heparin therapeutic range | Hemophilia A, B; Factor XI, XII deficiency Von Willebrand disease Lupus anticoagulant |
| Elevated | Prolonged | Severe liver disease Acute DIC | Factor I, II, V, X deficiency or abnormality |
| Normal | Normal or slightly prolonged | Usually normal blood clotting | Mild deficiency in coagulation factors Mild von Willebrand disease |

***Pts on Coumadin therapy:**
Expected therapeutic range for INR is 2.0 to 3.0. For some patients who have a high risk for thrombosis, the INR needs to be higher: 2.5 to 3.5.

Presurgical: Coumadin is held and INR/PTT should be ≤ 1.5 (some exceptions must be normal) to reduce the risk of bleeding. Pts not on Coumadin: INR should be normal (<1.1) or cause investigated.

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Hyper-coagulopathy: Thrombosis

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Hereditary (Primary)

- Elevated clotting Factor VIII
- Deficiencies in antithrombin, Protein C, Protein S
- Gene mutations: Factor V Leiden, prothrombin, etc.

Acquired (Secondary)

- **Autoimmune disease:** Antiphospholipid syndrome and lupus anticoagulant (D68.61, D68.62)
- **Immobilization** (due to hospitalization, travel, obesity, stroke, etc.): Local venous stasis by accumulation of clotting factors and fibrin resulting in blood clot formation.
- **Adverse effect of drugs:** steroids, oral contraceptives, anticoagulants, heparin
- **Clinical conditions:** Major trauma, pregnancy, malignancy, diabetes, myeloproliferative disorders

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Secondary Hypercoagulable State

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Coding Clinic Second Quarter 2021, p. 8

Question: A 79-year-old patient is diagnosed with **secondary hypercoagulable state** and has a history of paroxysmal atrial fibrillation (AF) on anticoagulant maintenance. Does the provider need to link the secondary hypercoagulable state with the atrial fibrillation? What is the appropriate code assignment for secondary hypercoagulable state in this scenario?

Answer: Assign code D68.69, **Other thrombophilia**, for secondary hypercoagulable state. Secondary hypercoagulable state is specifically indexed to this code and includes secondary hypercoagulable state NOS.

Secondary hypercoagulable states are acquired disorders of thrombosis due to complex and multifactorial mechanisms. Patients with AF on chronic anticoagulant therapy may have an increased incidence of acquired hypercoagulable state. However, unless specifically documented by the provider, coding professionals should not assume the presence of a secondary (acquired) hypercoagulable state, in patients with AF.

Anticoagulant therapy for conditions such as atrial fibrillation is to prevent thrombosis, i.e., “hyper-coagulopathy,” and therefore would be rare in this scenario.

Coding Clinic provides coding advice for diagnoses documented by the provider.

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Secondary Hypercoagulable State

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Primary hypercoagulable states are inherited abnormalities of coagulation in which an anticoagulant mechanism is defective, e.g., antithrombin III deficiency, protein C and protein S deficiency, and abnormalities of the fibrinolytic system. Lab tests: Lupus anticoagulant (LA) panels, activated protein C resistance, protein C and protein S activity, antithrombin activity, and genetic tests.

Secondary (acquired) hypercoagulable states include those due to autoimmune disease (lupus anticoagulant, antiphospholipid) and those due to precipitating factors such as:

- Immobilization: due to hospitalization, travel, obesity, stroke, etc.
- Adverse effect of drugs: steroids, oral contraceptives, anticoagulants, heparin
- Major trauma, pregnancy, malignancy, diabetes, myeloproliferative disorders

Primary and secondary hypercoagulable states are rare.

An acquired hypercoagulable state due to high-risk circumstances cannot be identified until a thrombosis occurs. If a thrombosis occurs due to immobilization, adverse effect of drugs, trauma, pregnancy, etc. and the precipitating factor is no longer present, the patient is no longer in a hypercoagulable state.

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Disseminated Intravascular Coagulation (DIC) Hypercoagulopathy and Hypocoagulopathy

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Overactive clotting (hypercoagulopathy) causing depletion of clotting factors and bleeding (hypocoagulopathy)

Common causes:

- Sepsis
- Malignancy
- Trauma
- Obstetrical complications
- Intravascular hemolysis, e.g., acute ABO hemolytic transfusion reaction

Findings:

- Vascular thrombosis and bleeding
- Thrombocytopenia
- Prolonged PT/INR and aPTT
- Low plasma fibrinogen
- Elevated fibrin split products (FSP)
- Elevated D-dimer
- Reduced clotting factors and inhibitors
- Multisystem organ failure

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Thrombocytopenia

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Thrombocytopenia is a platelet deficiency that can cause bleeding into the tissues, bruising, and can affect blood clotting post injury.

Common causes:

- Bone marrow diseases: aplastic anemia, leukemia, certain lymphomas and myelodysplastic syndromes.
- Chemotherapy and radiation therapy.
- Immune thrombocytopenia (ITP)
- Alcohol abuse and dependence
- Viral infections, i.e., hepatitis C or HIV

Thrombocytopenia is a separate diagnosis from coagulation disorders of the clotting factors.

150—450K = Normal reference range

< 50K = Risk of bleeding

PT/INR/aPTT are typically unaffected

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ICD-10 Classification

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| | |
|--------|---|
| D68.32 | Hemorrhagic disorder due to extrinsic circulating anticoagulants Drug induced hemorrhagic disorder Hemorrhagic disorder due to increase in anti-IIa Hemorrhagic disorder due to increase in anti-Xa Hyperheparinemia |
| D68.4 | Acquired coagulation factor deficiency Deficiency of coagulation factor due to liver disease Deficiency of coagulation factor due to Vitamin K deficiency |
| D68.9 | Coagulation defect, unspecified Deficiency, coagulation Coagulopathy |
| R79.1 | Abnormal coagulation profile |
| R79.01 | Long term use (current) of anticoagulants |

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Principal Diagnosis Sequencing

Bleeding due to Anticoagulant/Antithrombotic

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Which is the Principal Diagnosis?

Hemorrhagic disorder due to extrinsic circulating anticoagulants (D68.32)

or

Bleeding site/type

Depends on the circumstances of admission
Was the “focus” of the admission the bleeding itself
(e.g., evaluation, procedures, transfusion, monitoring, H/H)
or the correction of the bleeding disorder?

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Case #1

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GI Bleeding due to Antithrombotic

Patient admitted with GI bleeding while properly taking Plavix.

Treatment included discontinuation of Plavix, blood transfusion, EGD and colonoscopy, which identified a bleeding gastric ulcer.

Principal Diagnosis:

- K25.4, Gastric ulcer with bleeding

Secondary Diagnosis:

- D68.32, Hemorrhagic disorder due to extrinsic circulating anticoagulants [if documented]
- T45.525A, Adverse effect of antithrombotic

The ulcer with bleeding was the focus of the admission and an adverse effect of Plavix (antithrombotic).

Case #2

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GI Bleeding due to Anticoagulant

Patient with epistaxis and taking Coumadin as prescribed; nosebleed treated with nasal packing.

Admitted with INR 6.8, Coumadin discontinued, treated with fresh frozen plasma to reverse elevated INR, serial hemoglobin and INR, and subsequent cautious resumption of Coumadin based on INR.

Principal Diagnosis:

- D68.32, Hemorrhagic disorder due to extrinsic circulating anticoagulants [if documented]

Secondary Diagnosis:

- T45.515A, Adverse effect of anticoagulants
- R04.0, Epistaxis

The hemorrhagic disorder was the focus of the admission and an adverse effect of Coumadin.

If Coumadin was taken improperly, i.e., poisoning, the poisoning code (T45.511- or T45.521) would be sequenced first.

Case #3

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GI Bleeding due to Anticoagulant

83-year-old female with PMHx of HTN, CHF, CKD, Afib on Eliquis. Presented to ED with weakness, SOB. Hgb 4.6. Chronic black stools due to iron. Admit PT/INR 17.8/1.5.

H&P DX: Acute on chronic iron deficiency anemia 2nd to GI blood loss. GI consult: Severe anemia and melena in the setting of anticoagulation. Transfused 3 units PRBC.

Pt had a scheduled colonoscopy in one month so scope done while inpatient. Findings: Two small nonbleeding gastric ulcers. GI note post scope: "Anemia 2nd to PUD in setting of anticoagulation."

Queried and documentation clarification: "GI bleed due to coagulopathy from Eliquis as evidenced by improvement after stopping Eliquis."

Principal Diagnosis:

- D50.0, Iron deficiency anemia secondary to blood loss (chronic)

Secondary Diagnosis:

- K25.4, Chronic or unspecified gastric ulcer with hemorrhage
- D68.32, Hemorrhagic disorder due to extrinsic circulating anticoagulants

The **anemia** was the primary reason for admission and an adverse effect of Coumadin.

Case #4

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Denial: D68.4 Acquired Coagulation Factor Deficiency

Denial Rationale:

"This 61-year-old patient presented to the ED with complaints of not speaking and is uncooperative. The patient had a history of hepatitis C, liver cirrhosis, pancreatic mass and acute kidney injury. The lab results showed protime 21.8, INR 2.12, PTT 50.0.

The impression was hepatic encephalopathy, hyper-ammonemia, and coagulopathy due to liver failure. Although the submitted documentation showed the patient had acquired coagulation deficiency, the documentation did not show the patient had signs or symptoms such as nosebleed, bleeding gums, hematuria or GI bleeding, and the documentation did not show the patient required treatment or increased monitoring after initial lab work.

Acquired coagulation factor deficiency is not clinically supported as a complication for this episode of care based on the documented clinical findings in the medical record."

Case #4, continued

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Appeal: D68.4 Acquired Coagulation Factor Deficiency (Overturned)

- *The auditor states: The documentation did not show the patient required treatment or increased monitoring after initial blood work. This statement is invalid. This patient was being treated with **Phytonadione**, which is a specific treatment for coagulopathy due to liver failure.*
- *The documentation did not show the patient had signs or symptoms such as nosebleed, bleeding gums, hematuria, or gastrointestinal bleeding. Although signs and symptoms of a chronic condition that is being treated is unnecessary to code as a secondary diagnosis, the patient did experience a **nosebleed** while in the hospital, which often occurs with patients with a coagulation disorder.*
- *The finding is based on inadequate physician documentation in the medical record. The physician documentation in the medical record is hardly "inadequate": The diagnosis of "coagulopathy due to liver disease" was documented within the **History & Physical, Discharge Summary, and all progress notes.***
- *The auditor states there was no documentation to support that this diagnosis meets the Official Coding Guidelines for reporting "other diagnoses." This patient **clearly meets the definition of a secondary diagnosis** according to Official Coding Guidelines based on the above and below:*
 - Clinical evaluation: Labs (INR/PTT)
 - Therapeutic treatment: Phytonadione
 - Increased nursing care: Attention to nosebleed.

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Case #5

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"Coagulopathy"

Patient admitted with sepsis and known history of chronic anticoagulation for atrial fibrillation. During the stay, PT and INR are elevated from use of the anticoagulant warfarin. The patient is not identified as having a "bleed". PT 18.5, INR 2.5. Physician documented: "Coagulopathy due to chronic anticoagulation."

Is it appropriate to use the D68.9, Coagulation defect, along with Z79.01, abnormal coagulation profile?

What if the patient were on Apixaban (Eliquis)? How would one clinically validate "coagulopathy" when you don't have a lab test available?

What do we use to clinically validate "coagulopathy" in these two scenarios?

In the 1st scenario, the patient does not have a coagulopathy, i.e., coagulation defect, since there was no bleeding or other adverse effect. The patient is on anticoagulants and has an elevated INR.

Abnormal PT/INR or PTT due to an anticoagulant that does not cause bleeding is assigned **R79.1, Abnormal coagulation profile.**

In the 2nd scenario, if there is no evidence of abnormal bleeding due to anticoagulants, the appropriate code assignment would be **Z79.01, Long term (current) use of anticoagulants.**

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Cases #6 to 8

Surgical patient

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52-year-old female, morbidly obese, admitted for exploratory lap, abdominal hysterectomy/BSO for uterine fibroids on 10/26.

Pt noted to have considerable post-operative incisional bleeding due to subcutaneous hematoma, return to OR for wound exploration and closure.

Pt on Coumadin held for surgery and Lovenox given. Labs 10/26: PT 18.3/INR 1.61.

Clinically indicates:
D68.32, Hemorrhagic disorder due to extrinsic circulating anticoagulants.

52-year-old female, morbidly obese, admitted for exploratory lap, abdominal hysterectomy/BSO for uterine fibroids on 10/26 without complication.

Pt on Coumadin held for surgery and Lovenox given. Labs 10/26: PT 18.3/INR 1.61. PN: Elevated PT/INR.

Abnormal PT/INR or PTT due to an anticoagulant that does not cause bleeding is assigned code **R79.1, Abnormal coagulation profile.**

52-year-old female, morbidly obese, admitted for exploratory lap, abdominal hysterectomy/BSO for uterine fibroids on 10/26 without complication.

Pt on Coumadin held for surgery and Lovenox given; INR 1.1.

PT/INR in therapeutic range. Assign code **Z79.01, Long term (current) use of anticoagulants.**

In Summary...

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1. Hypocoagulopathies and hypercoagulopathies may be inherited or acquired.
2. Hypocoagulopathy is manifest by bleeding and hypercoagulopathy by thrombosis (primarily DVT).
3. Acquired hypocoagulopathies are most commonly due to therapeutic or adverse reaction to medication.
4. Primary and secondary hypercoagulable states are rare.
5. If a thrombosis occurs due to immobilization, adverse effect of drugs, trauma, pregnancy, etc. and the precipitating factor is no longer present, the patient is no longer in a hypercoagulable state.
6. Hemorrhagic disorder due to extrinsic circulating anticoagulants, code D68.32, is assigned for bleeding associated with anticoagulant/antithrombotic therapy with an additional code for the site of bleeding. Sequencing of the hemorrhagic disorder vs. bleeding site/type depends on the circumstances of admission.
7. Abnormal PT/INR or PTT that does not cause bleeding is assigned R79.1, Abnormal coagulation profile. If patient is treated with anticoagulants and the PT/INR is in therapeutic range, assign R79.01, Long term (current) use of anticoagulants.