Anticoagulation

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Disclosure

• Speaking fees: Novartis

Objectives

1. Describe the causal mechanisms and pathophysiology of thrombosis formation
2. Explain the appropriate use of anticoagulation for patients with cancer
3. Develop a plan of care for patients receiving anticoagulation in conjunction with complex cancer treatment regimens or requiring invasive procedures
Reasons for Anticoagulation

1. Atrial fibrillation (AF)
2. Artificial heart valve
3. Deep vein thrombosis (DVT)
4. Pulmonary embolism (PE)
5. Prevention of blood clots
6. Stroke (CVA)
7. Heart Attack (MI)

Hemostasis/Thrombosis
A Balance between Bleeding and Clotting
Homeostasis: A Normal Condition – “Equilibrium”

I. Vascular Phase

- Damaging blood vessels leads to vascular spasm of the smooth muscle in the vessel wall.
- Producing vasoconstriction which slows or stops blood flow.
- Lasts up to 30 minutes
- Localized to the damaged area.
II. Platelet Phase

- Damaged endothelial cells lining the blood vessel release von Willebrand's Factor making the surfaces cells "sticky".
- In larger blood vessels, platelets stick to the surfaces of endothelial cells. This effect is called Platelet Adhesion.
- The platelets that adhere to the vessel walls now begin to secrete Adenosine diphosphate (ADP) causing Platelet Aggregation, forming a platelet plug.
- This clumping of platelets serves a number of functions:
  1. It can plug the break in a small blood vessel.
  2. Aggregated platelets release Platelet Thromboplastin (Factor III) which activates the clotting process.
  3. Clumped platelets provide a surface for the clotting process.

(Hudnall, 2012)

III. Coagulation Phase

- This process depends on the presence in the blood of 11 different clotting factors (proteins) & calcium (Factor IV). Ultimately, these factors will generate the production of Prothrombin Activator (Factor X).
- Depending on the initial trigger for the clotting reactions, there are two pathways leading to the formation of the thrombus; the Extrinsic Pathway and the Intrinsic Pathway.

(Hudnall, 2012)

Pathways of the Coagulation Cascade

Extrinsic Pathway

- Initiated with material outside of or "extrinsic" to the blood. This material, Tissue Thromboplastin (Factor III), is released by damaged tissue cells. Factor III permits the clotting process to take a chemical shortcut. As a result, the extrinsic pathway is a very rapid process (12 to 15 seconds). However, the production of Thrombin is low and the resulting clot is small. This pathway is most effective as a "quick patch" process.

Damaged Tissue → Factor III → Factor VII → Factor X

(Schmaier, 2012)
Intrinsic Pathway
Initiated by blood coming in contact with exposed collagen in the vessel wall. This process is considerably slower (5 to 10 minutes) but results in the formation of larger amounts of thrombin. This allows the formation of larger clots.

<table>
<thead>
<tr>
<th>Factor XII</th>
<th>Factor XI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Factor IX</td>
<td>Factor VIII + Ca + Factor III</td>
</tr>
<tr>
<td>Factor X</td>
<td></td>
</tr>
</tbody>
</table>

(Common, 2012)

Common Pathway
1. Factor X (active) engages in a series of reactions with Factor V, Calcium ions and phospholipids derived from platelets. This composite of clotting factors and their reactions is referred to as the Factor V Complex or Prothrombin Activator.
2. Factor V Complex initiates the conversion of Prothrombin to active form of the enzyme Thrombin.
3. Thrombin accelerates the formation of Fibrin threads from Fibrinogen (Factor I).

(Hudnall, 2012)

IV. Fibrinolytic Inhibitor System
Clot Retraction
After 2 or 3 days, the clot begins to contract. Platelets in the clot contain contractile proteins. These proteins pull the edges of the wound together reducing the chance of further hemorrhage. This activity also assists the repair processes.

(Hudnall, 2012)
Fibrinolytic Inhibitor System

Fibrinolysis - Dissolution of the Clot
Breakdown of the clot is due to the production of a powerful proteolytic enzyme - Plasmin.

These reactions demonstrate that materials which induce clot formation (Thrombin and Factor XII) will eventually assist in the breakup of the clot!

(Hudnall, 2012)

Coagulation Factors

<table>
<thead>
<tr>
<th>FACTOR</th>
<th>NAME</th>
<th>PATHWAY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Factor I</td>
<td>Fibrinogen</td>
<td>Common</td>
</tr>
<tr>
<td>Factor II</td>
<td>Prothrombin (enzyme)</td>
<td>Common</td>
</tr>
<tr>
<td>Factor III</td>
<td>Thromboplastin</td>
<td>Extrinsic/Intrinsic</td>
</tr>
<tr>
<td>Factor IV</td>
<td>Calcium ions</td>
<td>Entire Process</td>
</tr>
<tr>
<td>Factor V</td>
<td>Proaccelerin (heat labile cofactor)</td>
<td>Extrinsic/Intrinsic</td>
</tr>
<tr>
<td>Factor VII</td>
<td>Proconvertin (enzyme)</td>
<td>Extrinsic</td>
</tr>
<tr>
<td>Factor VIII</td>
<td>Anti-hemolytic factor (cofactor)</td>
<td>Intrinsic</td>
</tr>
<tr>
<td>Factor IX</td>
<td>Christmas factor (plasma thromboplastin component)</td>
<td>Intrinsic</td>
</tr>
<tr>
<td>Factor X</td>
<td>Stuart Prower factor (enzyme)</td>
<td>Intrinsic/Intrinsic</td>
</tr>
<tr>
<td>Factor XI</td>
<td>Plasma thromboplastin</td>
<td>Intrinsic</td>
</tr>
<tr>
<td>Factor XII</td>
<td>Hageman factor (enzyme)</td>
<td>Intrinsic (activates plasma)</td>
</tr>
<tr>
<td>Factor XIII</td>
<td>Fibrin stabilizing factor</td>
<td>Retards fibrinolysis</td>
</tr>
</tbody>
</table>

Coagulation Cascade
Coagulation Disorders

Loss of this dynamic equilibrium by factor deficiencies or over-activation results in either:

- Significant bleeding - hemorrhage (Hemophilia)
- Abnormal clotting - thrombosis (Thrombophilia)

(Schmaier, 2012)

Risk Factors for Thrombosis

**Extrinsic**
- Advancing age
- Immobilization
- Prolonged air or automobile travel
- Major surgery
- Estrogens
- Obesity
- Smoking

**Intrinsic**
- Prior Thrombosis
- Antiphospholipid Antibody Syndrome
- Myeloproliferative Disorders
- Heparin-induced thrombocytopenia (HIT)

(Schmaier, 2012)

Virchow’s Triad
Deep Vein Thrombosis (DVT)

**Diagnosis** - Duplex Ultrasound of the affected extremity. Often check bilateral

**Symptoms**
1. Pain, tenderness in one extremity
2. Warmth, erythema, edema
3. Presence of dilated veins (collateral circulation) on the chest wall or leg
4. Low grade fever

**Pathophysiology**

(Pothoven et al., 2011)

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Pulmonary Embolism (PE)

**Diagnosis** –
- Spiral CT of Chest with PE Protocol
- VQ Scan (less accurate but may be used if patient allergic to IV Contrast)

**Symptoms** –
1. Dyspnea
2. Tachypnea
3. Pleuritic chest pain
4. Hemoptysis
5. Apprehension
6. Syncope
7. Tachycardia
8. Audible S3 or S4

**Pathophysiology**

(Lichtman et al., 2011)

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Thrombophilia

Increased Tendency for clotting, beyond the normal coagulation cascade
- Inherited or Acquired
- Can lead to DVT or PE, MI or Stroke
- Not everyone with thrombophilia will have a blood clot in their lifetime
- Many patients who do experience thrombotic events (such as DVT) may not have any detectable thrombophilia at all.

(Schmaier, 2012)
**Hereditary Thrombophilic Conditions**

- **Increased Procoagulant Proteins**
  - Factor V Leiden
  - Prothrombin Gene Mutation (G20210A)
  - Increased Levels of Factors VIII, IX, XI

- **Decreased Anticoagulant Proteins**
  - Antithrombin Deficiency
  - Protein C Deficiency
  - Protein S Deficiency

(Schmaier, 2012)

**Clinical Manifestations of Hereditary Thrombophilia**

- Family history of venous thromboembolism
- Thrombosis at a young age (<40 years)
- Unprovoked venous thromboembolism
- Recurrent venous thromboembolism
- Thrombosis in an unusual site
- Unexplained recurrent pregnancy loss (APA Syndrome)

(Schmaier, 2012)

**Factor V Leiden Mutation**

- Autosomal Dominant Mutation of Factor V
- Causes overproduction of Thrombin leading to excess fibrin causing excess clotting
- 5% of Caucasians in North America
- Less common in Hispanics and African-Americans
- Extremely rare in Asians

(Schmaier, 2012)
Prothrombin G20210A Mutation

- Autosomal dominant mutation of Factor II
- A G-to-A substitution in nucleotide position 20210 is responsible for a factor II polymorphism
- The presence of one allele (heterozygosity) is associated with a 3-6 fold increased for all ages and both genders. Homozygosity is extremely rare.
- Approx 2% of Caucasian population in US
- Rare in other populations
- The mutation causes a 30% increase in prothrombin levels.

(Schuessler, 2012)

Antithrombin Deficiency

- Also known as Antithrombin III Deficiency
- Very rare
- Autosomally dominant (rare cases of recessive)
- Inhibits coagulation by irreversibly binding the thrombogenic proteins thrombin (IIa), IXa, Xa, XIa & XIIa
- Antithrombin’s binding reaction is amplified 1000-fold by heparin so higher doses of heparin needed to treat

(Lichtman, et. al., 2011)

Protein C or S Deficiency

Protein C Deficiency
- 1 in 300 population
- Increased incidence of venous thromboembolism (relative risk 8-10)
- Homozygous defect potentially life-threatening
- Cannot be measured on warfarin

Protein S Deficiency
- 1 in 20,000 population
- Vitamin K dependent
- Acts as a non-enzymatic cofactor to activated protein C
- Can also be acquired
- Cannot be measured on warfarin
- Can cause DIC

(Lichtman, et. al., 2011)
### Antiphospholipid Antibody Syndrome

**Diagnosis**

<table>
<thead>
<tr>
<th>Lab Criteria</th>
<th>Clinical Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>IgG or IgM anticardiolipin antibody elevation</td>
<td>Thrombosis: arterial or venous</td>
</tr>
<tr>
<td>Positive results of ACA duplicated at least 3 months after initial elevation.</td>
<td>Pregnancy loss</td>
</tr>
<tr>
<td>Positive Beta2 Glycoprotein</td>
<td>Thrombocytopenia</td>
</tr>
<tr>
<td>Lupus Anticoagulant (DRVVT)</td>
<td>CNS syndromes: stroke</td>
</tr>
<tr>
<td></td>
<td>Cardiac valve disease</td>
</tr>
<tr>
<td></td>
<td>Livedo Reticularis</td>
</tr>
</tbody>
</table>

(Lichtman, et al., 2011)

**Acquired Thrombophilic Conditions**

- Major surgery
- Immobilization
- Prolonged air travel
- Cancer
- Estrogens
- Antiphospholipid antibodies
- Myeloproliferative disorders
- Hyperhomocysteinemia
- Heparin-induced thrombocytopenia

(Schmaier, 2012)

### Heparin-Induced Thrombocytopenia (HITT)

**Type 1: Benign, not true HITT**
- Occurs 1-2 days after starting Heparin
- Mild drop in platelets; transient - normalizes quickly

**Type 2: Life-threatening Prothrombotic Condition**
- Occurs 4-10 days after initiation of Heparin (delayed - can occur up to 3 weeks after stopping heparin)
- Moderate to severe drop in platelets (>50%)
- Results in thrombosis (large vessels) rather than bleeding
- Treatment involves cessation of heparin, treatment with an alternative drug, e.g. argatroban, and switching to warfarin.
- NO MORE HEPARIN!!!
**Treatment Options for VTE**

A. Vascular Intervention

B. Pharmacologic Management
   - **Parenteral Anticoagulation**
     - heparin sodium injection, Pfizer
     - enoxaparin (Lovenox®, Sanofi)
     - fondaparinux (Arixtra®, GlaxoSmithKline)
     - dalteparin sodium injection (Fragmin®, Pfizer)

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**Heparin (unfractionated)**

**Pharmacology**
- Acts at multiple sites in the coagulation process; binds to antithrombin III, catalyzing inactivation of thrombin and other factors
- Excreted in the urine
- Half-life: 1.5 hours

**Dosing**
- Treatment of VTE: start IV @80u/Kg x 1h; alt – 5000u IV x 1, then 1000u IV; 333u/kg SCx1, then 250 u/Kg q12h, adjust to anti-Xa level
- Prophylaxis: 5000u SC q 8 – 12 h

(Hospira, 2014)

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**Lovenox (enoxaparin)**

**Pharmacology**
- Binds to antithrombin III and accelerates activity, inhibiting thrombin and Factor Xa.
- Excreted in urine
- Half-life: 4.5 to 7 hours

**Dosing**
- Dose for VTE: 1mg/kg every 12 hours for > 5 days & bridge w/ warfarin until INR > 2.
- Dose for prophylaxis: 40 mg sq daily
- Also used for DVT prophylaxis for surgery, periods of immobility, pregnancy

(Amphastar Pharmaceuticals, 2012)
Arixtra (fondaparinux)

Pharmacology
- Selectively inhibits antithrombin III, potentiating Factor Xa neutralization & inhibiting thrombin formation
- Excreted in the urine unchanged
- Half-life 17-21 hours

Dosing:
- < 50kg – 5mg sq
- 50 to 100kg – 7.5mg sq
- 100kg – 10mg sq

Prophylaxis: 2.5mg sq daily; duration dep on type of surgery.
- DC if platelets < 100,000
- CrCl 30-50: caution advised
- Contraindicated for CrCl <30

Treat for 5-7 days bridging w warfarin until INR > 2

(Mylan, 2014)

Fragmin (dalteparin)

Pharmacology
- Binds to antithrombin III & accelerates activity, inhibiting thrombin & Factor Xa
- Excreted in urine
- Half-life 3 - 5 h

Dosing
- Treatment of VTE: 200u/kg/d SQ bid; max 18000u/dose; continue for >5d & overlap with warfarin until INR ≥2
- Decrease dose by 250u/d if plt 50K-100K
- DC if plt < 50K
- Prophylaxis: 5000u sq daily

(epocrates, 2016)

Treatment Options for VTE

- Oral Anticoagulation
  - warfarin (Coumadin®, Bristol-Myers Squibb )
  - rivaroxiban (Xarelto®, Janssen)
  - apixaban (Eliquis®, Pfizer)
  - edoxaban (Savaysa®, Daiichi-Sankyo)
  - dabigatran (Pradaxa®, Boehringer-Ingelheim)
Pharmacology

- Inhibits synthesis of coagulation factors II, VII, IX and X
- Factors are synthesized in the liver: CYP 450: 1a2, 2CB, 2C18, 2C9 (primary), 2C19, 3A4 substrate
- Also inhibits synthesis of Protein C and Protein S
- Excreted in urine 92%
- Half-life 20-60h anticoagulant effect; variable based on rate of clotting factor catabolism
- Pregnancy Category X

Dosing

- Loading dose 5 -10 mg for first few days, bridging with parenteral
- Use with extreme caution in:
  - Elderly
  - Malnourished
  - History of congestive heart failure (CHF)
  - Liver disease
  - Had recent major surgery
  - Taking medications known to increase sensitivity to INR
- An observable effect is seen 2-7 days after the initiation of dose
- A maintenance dose of 5mg will usually reach an INR of 2-3 in 4-5 days depending on patient related factors
- Can be reversed by Vitamin K

INDICATION | INR
---|---
Prophylaxis of venous thrombosis | 2.0 - 3.0
Treatment of venous thrombosis | 2.0 - 3.0
Treatment of PE | 2.0 - 3.0
Prevention of systemic embolism in:
  - Tissue heart valves | 2.0 - 3.0
  - Acute myocardial infarctions (AMI) | 2.0 - 3.0
  - Valvular heart disease | 2.0 - 3.0
  - Atrial fibrillation | 2.0 - 3.0
  - Mechanical prosthetic heart valves | 2.5 - 3.5
  - Bileaflet mechanical valve in aortic position | 2.0 - 3.0
**Xarelto (rivaroxaban)**

**Pharmacology**
- Selectively blocks active site of factor Xa, inhibiting blood coagulation
- Excreted in urine 66% (36% unchanged) & feces 28% (7% unchanged)
- Half life: 5-9h & 11-13h in elderly patients

**Dosing**
- Treatment of VTE: Start: 15mg po bid x 21 days then 20mg po daily w/ food
- Prophylaxis: Hip (35) and knee (12) replacement – 10 mg po daily
- Renal dosing – see prescribing info; avoid use of CrCl < 30

(Lammers, 2014)

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**Eliquis (apixaban)**

**Pharmacology**
- Selectively blocks active site of factor Xa, inhibiting blood coagulation
- Excreted in urine 27% & feces
- Half life: 12h

**Dosing**
- Treatment of VTE: Start 10 mg po bid x 7 days, then 5 mg bid
- Decrease dose to 2.5 if 2 of the following: 80y or older, Wt < 60Kg, or Cr > 1.5
- Prophylaxis: 2.5 mg po bid Hip (35) and knee (12) replacement
- Renal dosing & Hepatic dosing – see prescribing info

(Stevens-Bymo, 2015)

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**Savaysa (endoxaban)**

**Pharmacology**
- Selectively blocks active site of factor Xa, inhibiting blood coagulation
- Excreted in urine 50% (primarily unchanged) bile, feces, other
- Half life: 10 -14h

**Dosing**
- Treatment of VTE: <60Kg - 30mg po daily; > 60Kg – 60mg po qd start 4 h after heparin
- Prophylaxis: 60 po qd
- Renal dosing – see prescribing info; avoid use of CrCl < 15
- Hepatic dosing: Child-Pugh class B or C avoid

(Stevens-Bymo, 2015)
**Pradaxa (dabigatran)**

**Pharmacology**
- Directly, reversibly inhibits thrombin
- Pro-drug converted to dabigatran
- Metabolized in the liver CYP450
- Excreted in the urine
- Half-life 12-17h

**Dosing**
- VTE, Non-valvular AFib - 150mg po bid
- Prophylaxis: 220mg po daily x 28 to 35 days post op
- To convert from warfarin: d/c warfarin then start dabigatran when INR <2
- Renal dosing
  - DVT/PE prophylaxis CrCl 15-30 – 75mg po bid; CrCl < 15 hold
  - All other dx – CrCl < 30 hold

(epocrates, 2016)

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**What about Aspirin?**

- Dosing: 75-325mg (81mg most common); higher dosing more toxicity without improved efficacy
- 23% risk reduction in adverse cardiac events as primary treatment for chest pain (ISIS-2, Lancet 2:349, 1988)
- 25% risk reduction in adverse cardiac events as secondary treatment (Lancet 360:3, 2002)
- 23% reduction in total cardiac heart disease
- 24% reduction in non-fatal MI
- No effect on stroke or overall mortality
- 40% risk reduction in recurrent VTE when added after stopping oral or parenteral anticoagulants (NEJM 366:1959, 2012)

(Slide compliments of Alvin Schmaier MD, 2015)

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**Anticoagulation in the Cancer Patient**

**Thrombosis – Frequent complication of Cancer**
- Venous or Arterial (Most often VTE/PE, but also CVA/MI)
- Second leading cause of death in cancer patients
- May be seen within the first few months prior to DX
- Diagnosis often incidental (splenic, portal, mesenteric)
- Associated with recurrent VTE as well as bleeding, both at significantly higher rates than seen in non-cancer patients
- Associated with a threefold increase in hospitalizations and higher total health care costs

(Khorana, et al., 2016)
**Predictive Model for VTE**

<table>
<thead>
<tr>
<th>PATIENT CHARACTERISTICS</th>
<th>RISK SCORE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Site of cancer</td>
<td></td>
</tr>
<tr>
<td>Very high risk (stomach, pancreas)</td>
<td>2</td>
</tr>
<tr>
<td>High risk (lung, lymphoma, GYN, bladder, testicular, myeloma on imid)</td>
<td>1</td>
</tr>
<tr>
<td>Pre-chemotherapy platelet count &gt;350,000/mm³</td>
<td>1</td>
</tr>
<tr>
<td>Hemoglobin level less than 10 g/dl or use of red cell growth factors</td>
<td>1</td>
</tr>
<tr>
<td>Body mass index &gt;35 kg/m² or more</td>
<td>1</td>
</tr>
</tbody>
</table>

High-Risk Score ≥ 3 - Consider prophylaxis with LMWH
LMWH or ASA for Myeloma patient on imid-based tx
Intermediate-Risk Score 1-2 – Inpatient prophylaxis w LMWH or Heparin for acute medical illness or undergoing major surgery
Low-Risk Score 0 – Treat as a non cancer patient

(Khorana, et al., 2016)

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**Which Drug to Choose?**

**Evidence for CA patients with VTE:**

- 12% annual risk of bleeding complications & 21% risk of recurrent VTE on warfarin
- Epidemiologic studies suggest that cancer related VTE may be resistant to warfarin
- Two studies CLOT and CATCH trials confirmed that LMWH was superior to warfarin for management of VTE in patients with Cancer
- Efficacy and safety of oral anti-Xa inhibitors has yet to be determined

(Khorana et, al. 2016)

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**Recommendations**

1. Anticoagulation with LMWH should be prescribed for a minimum of 6 months after dx of cancer associated VTE and continued beyond 6 months if patient has active malignancy or if ongoing anti-cancer Tx.
2. Cancer patients with symptomatic or recurrent VTE despite therapeutic anticoagulation with other AC agent should be transitioned to LMWH, assuming no contraindication.
3. IVC filters are NOT recommended except in the presence of absolute contraindication for AC (i.e. active bleeding or surgery). If necessary, retrievable filters should be used and retrieved when appropriate.

(Khorana et, al. 2016)
Cancer Patients on Chemotherapy

Practical Considerations
- Financial Issues
- Allergies and complications (HITT)
- Co-morbidities
- Thrombocytopenia
  - ITP
  - Chemo-induced
- Organ failure 2/2 to disease (Kidney, Liver)

Management of Patients for Surgical Procedures

Guidelines for CA patients & non-cancer patients:
1. Specific period to hold AC agent dependent upon half life and how invasive the procedure
2. For cancer patients on Tx - try to plan procedure around NADIR
3. Benefit vs risk always considered
4. Bridging with lovenox at therapeutic dose is recommended when holding longer acting AC
5. Lovenox is held for approx 24 hours prior to procedure
6. Use of IVC filter when anticoagulation is contraindicated and removed as soon as appropriate
7. Resume anticoagulation as soon as hemostasis is achieved
8. Heme consult is often recommended for management

(Dacelt Guidelines, 2016)

Case Study

A 20 year old female presents for consultation regarding contraception. She states that her mother has a history of Heterozygous Factor V Leiden mutation and suffered a lower extremity thrombosis during pregnancy. She also has a maternal aunt who is on lifelong anticoagulation for recurrent thrombosis.

The most appropriate intervention is:

a. Prescribe a high progesterone, low estrogen oral contraceptive & advise the patient to call if she has symptoms of DVT/PE
b. Send the patient for a Heme consult for a prothrombotic work-up prior to prescribing oral contraception
c. Recommend an IUD and advise the patient not to use oral contraceptives
d. Prescribe a high progesterone, low estrogen oral contraceptive and recommend the patient take an 81mg aspirin daily for thrombosis prophylaxis
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d. Prescribe a high progesterone, low estrogen oral contraceptive and recommend the patient take an 81mg aspirin daily for thrombosis prophylaxis

Case Study

A 64 yo male presents with a previous history of hypertension. He has a screening colonoscopy showed a right cecal mass requiring surgery. The patient is diagnosed with a T3, N4, M0 lesion. 24 h post-op, the patient has a PE and he was anti-coagulated with enoxaparin. Unfortunately, management was complicated with heparin-induced thrombocytopenia and thrombosis in his left leg. Finally, after a long hospitalization you are arranging discharge planning. What anticoagulant should you consider for his management at this time?

Your choice for an anticoagulant is which of the following?

a. fondaparinux
b. warfarin

c. rivaroxaban

d. heparin

Case Study

Your choice for an anticoagulant is which of the following?

a. fondaparinux
b. Warfarin – not recommended for cancer patients with thrombosis

c. Rivaroxaban - not recommended for cancer patients with thrombosis

d. Heparin – contraindicated for HITT
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Division of Hematology/Oncology
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