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Jointly provided by the Annenberg Center for Health Sciences at Eisenhower and Gastroenterology and Hepatology Advanced Practice Providers.
Thromboembolism in Liver Disease – To Treat or Not to Treat?

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Disclosures

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Speakers Bureau: Salix Pharmaceuticals, Clinical Area – Hepatic Encephalopathy
Learning Objectives

• To describe the **potential risks of portal vein thrombosis**

• Identify parameters that are useful in determining bleeding risk

• To explore the **recommended treatments and be aware of contraindications** for portal vein thrombosis
Case Study 1

- JD is a 50-year-old male with decompensated liver disease from alcohol was admitted for esophageal variceal bleeding
- Patient’s liver disease has been complicated by PVT, was on Eliquis and this was discontinued. He underwent EGD and had 10 EV banding patient
- Next day: Underwent TIPS
- Patient improved 2 weeks later. US doppler of the liver showed partial PVT
Overview: End Stage Liver Disease

• Liver disease moves all aspects of “clotting” including hemostasis, coagulation, fibrinolysis

• Reduced clotting factor levels are accompanied by decreased anticoagulant proteins: inappropriate balance between bleeding and clotting

• Standard coagulation tests DO NOT accurately predict the risk of bleeding, particularly in decompensated cirrhosis and ACLF (acute on chronic liver failure)

• Thrombocytopenia in cirrhosis is challenging related to risk of bleeding particularly during procedures
Mechanisms of **Dys-coagulation** in ESLD

- Portal hypertension – slow flow
- Imbalance of clotting factors
- Accelerated Intravascular coagulation and fibrinolysis – weak clots
Simplified View of Normal Hemostasis

Coagulation Cascade

A. Vasoconstriction

B. Primary Hemostasis

C. Secondary Hemostasis

D. Thrombus and Antithrombotic Events
Coagulation Cascade

Contact activation (intrinsic) pathway

Damaged surface

XII → XIIa

XI → Xa

IX → IXa

VIII → VIIIa

Prothrombin (II) → X

Active Protein C

Protein S

Protein C + Thrombomodulin

Tissue factor (extrinsic) pathway

Trauma

VIIa → VII

VIIa → VII

Tissue factor

Antithrombin

I

Common pathway

Fibrinogen (I) → Fibrin (Ia)

XIIIa → XIII

Cross-linked fibrin clot

Coagulation Cascade

Virchow’s Triad Risk for Thrombosis

ENDOTHELIAL INJURY → THROMBOSIS

→ ABNORMAL BLOOD FLOW

→ HYPERCOAGULABILITY
Portal Hypertension
Virchow’s Triad Risk for Thrombosis

- Endothelial Injury
- Thrombosis
- Abnormal Blood Flow
- Hypercoagulability
# Platelet Function

## Hypo-coagulable Factors
- Thrombopoietin
- Splenic Sequestration
- Platelet dysfunction

## Hyper-coagulable Factors
- Endothelial factor – von Willebrand Factor
Cirrhosis: Effect on Fibrinolysis

- All profibrinolytic and antifibrinolytic proteins are synthesized by the liver EXCEPT tissue plasminogen activator (tPA) and PAI-1 (endothelial cells)

- Plasminogen, alpha 2 antiplasmin, thrombin-activatable fibrinolysis inhibitor (TAFI) and FXIII levels are reduced

- tPA levels elevated

The fibrinolytic process is controlled by completing players:

- Thrombin- activatable fibrinolysis inhibitor (TAFI),
- activated by thrombin and plasminogen – activator inhibitor (PAI) both inhibit plasmin
- Ultimately, plasmin cleaves the fibrin cross-links to initiate clot breakdown

**Hemostasis in Patients With ESLD**

**Antihemostatic (promote bleeding) Drivers**
- Thrombocytopenia
- Abnormal platelet function
- Decreased production of thrombopoietin
- Increased production of nitric oxide and prostacyclin

- Low levels of factors II, V, VII, IX, X, and XI
- Vitamin K deficiency
- Dysfibrinogenemia

- Low levels of α2-antiplasmin factor XIII, and TAFI
- Elevated level of t-PA

**Prohemostatic (promote clotting) Drivers**
- Elevated level of von Willebrand factor
- Low level of ADAMTS 13

- Elevated level of factor VII
- Low levels of protein C, protein S, antithrombin, and heparin cofactor II
- Inherited thrombophilia

**Equilibrium**
- Normal hemostasis
- Primary hemostasis

**Coagulation**

**Fibrinolysis**
- Low level of plasminogen

ADAMTS 13 = VW cleaving protease
Coagulation Cascade

Anti-coagulant (Increased up to 200%)
- Low levels of pro-coagulants Factor V, VII, X

Pro-coagulant (decreased by 25-70%)
- Low Protein C
- Elevated Factor VIII
- Venous Stasis
- Endothelial dysfunction

Hemostatic Dysbalance: Dysfibrinogenemia; Platelet Dysfunction; RES dysfunction
Cirrhosis and Its Effect on Coagulation

- Diminished synthesis of procoagulants Factors II, V, VII, IX, X and XI
- Fibrinogen levels are normal or increased, however 50-78% of patients have dysfibrinogenemia (abnormal fibrinogen that does not polymerize)
- High Factor VIII levels (cytokine release, decreased hepatic clearance)
- Decreased anticoagulant levels (Antithrombin, Protein C and S)
- Tissue factor pathway inhibitor (TFPI) levels increased (synthesized by endothelial cells). These levels are normal or elevated in cirrhosis, but impaired due to decreased protein S levels
A week later, JD’s bleeding was stable. Noted to have pleural effusion and symptomatic with SOB.

Radiology just called and the INR is 2.1 and the platelet count is 40.

Can the patient safely get the planned thoracentesis?
Thrombocytopenia

Thrombocytopenia

• Half-life of a transfused platelet is **2.5-4.5 days** and they don’t work as well
  – Giving platelets on Friday for Monday’s tooth extraction might not work

• Thrombopoietin agonists
  – Increase platelets without increasing portal hypertension
  – For a planned procedure might be nice
  – Eltrombopag can cause portal vein thrombosis
  – Avatrombopag and lusutrombopag?
Thrombopoietin Agonists

**Avatrombopag**
- Platelets 40-49: 40 mg po qd
- <40: 60 mg po qd

**Lusuthombopag**
- 3 mg po qd

**Procedure**
- Mean Δ 33–36 x 10⁹/L
- Mean Δ 25–28 x 10⁹/L
- Max median Δ 45 x 10⁹/L
Should I Give Platelets?

- Platelets promote thrombin generation
- 50,000-60,000
- Where’s the evidence?
- Platelets are activated due to endothelial dysfunction
  - Elevated vWF
- Potential transplant patient has antibodies...

Where’s the evidence?
INR

• Measures pro-coagulants: I, II, V, VII, X
• Does not measure deficits of liver derived factors such as protein C
• Technical factors – the reagents used affect ability to compare between labs
What Can I Use to Determine Risk

- **Fibrinogen**
  - Synthesized normally in the liver
  - Cryoprecipitate can be used which is lower volume than FFP
Should I Correct the INR?

- FFP does not change Thrombin (Factor II) production
- Volume expansion increases portal hypertension
TEG

**Alpha Angle**: Angle of tangent line from 2mm-20mm amplitude.
- Hypofibrinogenemia
- Cryoprecipitate

**Reaction Time**: Time from start to 2mm amplitude.
- Clotting factor deficiencies, anti-coagulants
- Hypercoagulable states
- Fresh frozen plasma

**Clot Formation Time**: Time from 2mm-20mm amplitude.
- Hypofibrinogenemia
- Cryoprecipitate

**Maximum Amplitude**: Amplitude measured at peak clot strength.
- Thrombocytopenia, platelet dysfunction
- Platelets, DDAVP

**Lysis Index**: Percent loss of amplitude at 30min after MA
- Enzymatic or mechanical fibrinolysis
- Tranexamic acid
TEG

- Normal
- Anticoagulants/hemophilia
- Platelet blockers
- Fibrinolysis
- Hypercoagulability
<table>
<thead>
<tr>
<th>Apten</th>
<th>Fibten</th>
<th>Intern</th>
<th>Extern</th>
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<tr>
<td>Normal findings</td>
<td>Decreased fibrinogen concentration</td>
<td>Decreased platelet count</td>
<td>Hyperfibrinolysis</td>
</tr>
</tbody>
</table>
Should I Replace Factors

• FFP
  – 250mL/U
  – Dosed 10mL/kg
  – 70 kg man → 70kg x 10ml/kg = 700 ml ≈ 4 units of FFP

• Vitamin K
  – Maybe especially in malnutrition, antibiotic exposure, malabsorption
  – 10mg IV (anaphylaxis) or Oral x 1. If no response within 12 hours, unlikely beneficial

• Antifibrinolytics – used in emergencies
  – Aminocaproic Acid
  – Tranexamic Acid

• Desmopressin
  – May have a role in ESRD
Hemoglobin

- Hematocrit $\geq 25$ may improve margination of platelets
- 1 unit PRBCs raises HCT by 3% at a cost of 250mL/unit
What About Esophageal Varices?
Risk of Progression of Varices 10% Per Year

Fig. 4

- Stage 1: NO VARICES NO ASCITES
  - 7% progress to VARICES NO ASCITES
  - 1%

- Stage 2: VARICES NO ASCITES
  - 4.4% progress to ASCITES ± VARICES
  - 3.4%

- Stage 3: ASCITES ± VARICES
  - 6.6% progress to BLEEDING ± ASCITES
  - 7.6%

- Stage 4: BLEEDING ± ASCITES
  - 20% progress to DEATH
  - 57%

- DEATH
Variceal Screening

Cirrhosis Suspected

Platelet Count (Plt), Imaging, Elastography (LS)

LS > 20kPa & Plt <150
Screening EGD

Variceal Screening and Surveillance Intervals

- **3 years**
  - Compensated No Varices Quiescent Disease

- **2 years**
  - Compensated No Varices Ongoing Injury
  - Small Varices

- **1 year**
  - Small Varices Ongoing Injury
  - Treated Varices Not on Beta-blockade

- **Now**
  - Decompensation

How Do I Prevent Bleeding in Cirrhosis?
How Do I Prevent Bleeding in Cirrhosis?

- Assessment of lab parameters
- Risk stratification for portal hypertensive bleeding
- **Should I ever anticoagulate a liver patient?**
Confused?
Hypercoagulability Contributes to Morbidity in Patients With Cirrhosis

- PVT: Incidence up to 25%

- Venous thromboembolism:
  - Prevalence: 2% of hospitalized cirrhotics vs. 0.9% controls
  - Incidence: 2-fold higher in cirrhotics than controls

- Renal replacement circuit life decreased in liver failure vs. controls

- Ischemic stroke incidence adjusted for risk factors 30% higher in cirrhotics compared to controls (RR 1.3 [1.2-1.5])
Contributions to Abnormal Coagulation From the Complications of Cirrhosis

- Hct >25% to promote plt adherence to vascular wall
- Uremia
  - Increased Infection
  - Decreased serotonin
- Anti-coagulants
- Antibiotics
- SSRIs
- Portal pressure
- Inflammatory
- Impair plt and clot formation BUT decreases fibrinolysis net even

Proposed Model for Occurrence of Microvascular and Macrovascular Thrombosis in Cirrhosis

Cirrhosis → Portal hypertension

↓

Slow portal flow (stasis) Increased intestinal permeability

Bacterial translocation
Endotoxemia
Inflammation

Endothelial perturbation → Increased vonWillebrand factor release

↓

Pro-coagulant activation Low protein C/High Factor VIII

Increased platelet adhesion

Microvascular thrombosis → Macrovascular thrombosis

↓

Parenchymal extinction
Portal vein thrombosis

Liver disease progression

Natural History of Nontumorous Portal Vein Thrombosis in Cirrhosis and Possible Therapeutic Approaches

Incidence of PVT:
- 4.6%–12.8% at 1 year
- 10.2%–20% at 5 years
- ~38.7% at 8–10 years

Spontaneous resolution: 31%–70%
Regression: 47%
Stable disease: 45%
Progression: 7.2%

Asymptomatic: 43%–62%
Symptomatic: 38%–57%

Stable disease: Follow-up
Progressive disease: Inherited thrombophilia
- With or without large spontaneous shunts

Management options:
1. Anticoagulation
2. TIPS
3. Transplant
4. Treat varices—NSBB ± EVL
5. Treat collateral—BRTO/shunt closure

Chronic

Acute
PVT Does Not Affect Outcome

- French outpt study using US for HCC surveillance
  - 1,243 pts: Child A: 863 Child B: 380
- Cumulative incidence of PVT 1-, 3-, and 5-year
  - 4.6%, 8.2%, and 10.7%
  - Nonocclusive in 101/118 patients
- Nonocclusive PVT resolved in 70% (70/101) then reappeared in 19% (19/101)
- PVT not independently associated with progression of liver disease or death
- Development of PVT is a marker, but not a direct cause of the progression of liver disease

<table>
<thead>
<tr>
<th>Models</th>
<th>Univariate Models Unadjusted Estimates</th>
<th>Multivariate Models Adjusted for the Baseline Prognostic Variables</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>HR</td>
<td>95% CI</td>
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<tr>
<td>Liver disease progression</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Partial PVT</td>
<td>1.58</td>
<td>1.02 – 2.45</td>
</tr>
<tr>
<td>- Partial or Complete PVT</td>
<td>1.48</td>
<td>0.97 – 2.26</td>
</tr>
<tr>
<td>Decompenensation</td>
<td></td>
<td></td>
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<tr>
<td>- Partial PVT</td>
<td>1.77</td>
<td>1.07 – 2.92</td>
</tr>
<tr>
<td>- Partial or Complete PVT</td>
<td>1.61</td>
<td>0.98 – 2.62</td>
</tr>
</tbody>
</table>

Early Stage Disease, low rate of complete thrombosis

Decompensation Inpatient Cirrhotics +/- PVT

- 7,296,968 total unweighted admissions in the 2012 NIS
  - 113,766 (1.6%) inpatient admissions for cirrhosis
- Prevalence of PVT among all inpatient admissions was 0.07% (n = 5046) and 1.8% (n = 2046) in patients with cirrhosis (P < .001)

Anticoagulation Improves Outcomes in Cirrhotic Patients With PVT

Complete recanalization of PVT

Progression of PVT

Variceal Bleeding

<table>
<thead>
<tr>
<th>Rx anticoagulants vs no Rx</th>
</tr>
</thead>
<tbody>
<tr>
<td>PVT recanalization: 71% vs 42% (P &lt; .0001)</td>
</tr>
<tr>
<td>Complete recanalization: 53% vs 33% (P = .002)</td>
</tr>
<tr>
<td>PVT progressed: 9% vs 33% (P &lt; .0001)</td>
</tr>
<tr>
<td>No difference major or minor bleeding: 11% vs 11%</td>
</tr>
<tr>
<td>Variceal bleeding 2% vs 12% (P = .04). ?</td>
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</table>

Anticoagulation of PVT Improves Survival

- All with cirrhosis and PVT ≥ 3 months FU after the first PVT detection.
  - 81 anticoagulants (56 LMWH, 15 fondaparinux, 10 VKA) vs 101 none
- Extension thrombosis decreased by > 50%: 56.8% vs 25.7%
- 36% recurrent thrombosis after stopping anticoagulation
- Higher survival rate in the treated group (p = 0.010)
- Bleeding complications 19.7% vs 21.8%

182 pts (1/2008 to 3/2016)
PVT Prior to LT

• More technically complex surgery
• More complications with increase in
  – Early hepatic artery thrombosis
  – Acute post-transplant PVT
  – Need for PRBC’s during their operation
• Lower graft and patient survival
  – Increased risk only in the first year
  – Associated with complete main PV thrombosis

PVT

• May be associated with increased complications and decreased survival in cirrhotic pts
  – More pronounced with more advanced liver dz
  – Complete occlusion and more extensive thrombosis likely worse outcome

• Pre LT PVT may decrease survival and increase complications in first year post LT

• Anticoagulation may decrease complications and improve survival (controversial)
  – No increase bleeding risk in selected pts
Anticoagulation of PVT

WHO?

• Acute or recent PVT in potential or listed LT candidates
  – Symptomatic acute occlusive PVT (e.g., worsening portal hypertension)
  – Progression of PVT on imaging (particularly when proximal veins, i.e., SMV involved)
  – If concern for risk of mesenteric ischemia
  – Hypercoagulable risk factors and PVT

WHEN?

• No spontaneous resolution or recanalization after reimaging (1–3 mo)
  – No medical contraindications to anticoagulation after detailed clinical history (e.g., bleeding risk assessment, fall assessment)
  – After EGD assessment of portal HTN or other mucosal lesions and subsequent prophylaxis (band ligation or non-selective b-blocker for high-risk varices)

Anticoagulation of PVT

WHY?

- In LT candidates
  - Recanalization to allow physiologic anastomosis decreasing difficulty of LT, complications, mortality
  - In non-LT candidates
  - *Recanalization to decrease complications of Portal HTN
  - *Improve survival
    - *But data are less clear as to overall benefit
  - Individualized approach is essential
  - Consider Low MELD with occlusive or progressive thrombus^.

WITH WHAT?

- Selection of anticoagulant should be individualized.
  - Review limitations and benefits of each medication with patient
  - Often a multidisciplinary approach with hematology is advised

^RCD additions.
Anticoagulation of PVT

**HOW LONG?**

- Expert consensus recommendations: **MINIMUM OF 6 MONTHS** duration though no definitive data
  - Consider high risk of recurrence unless underlying risks change and clinical implications of recurrence

- May consider indefinite anticoagulation if underlying hypercoagulability or when the patient is listed for transplantation

- Interval imaging to assess response to anticoagulation every 3 months on therapy is advised

- If anticoagulation is stopped, imaging at 3–6 months intervals to assess for recurrence

Sites of Action for Commonly Used Anticoagulants

- Direct:
  - Heparin
  - LMWH
  - Rivaroxaban
  - Apixaban
  - Edoxaban
  - Betrixaban

- Indirect:
  - Fondaparinux
  - Arixtra
  - Xarelto
  - Eliquis
  - Bevyxxa
  - Savaya

# DOAC Hepatic Metabolism, Pharmacokinetics, INR Effects

<table>
<thead>
<tr>
<th>Mechanism Action</th>
<th>Rivaroxaban (Xarelto)</th>
<th>Apixaban (Eliquis)</th>
<th>Fondaparinux (Atrixtra)</th>
<th>Dabigatran (Pradaxa)</th>
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<tbody>
<tr>
<td>Hepatic Metabolism</td>
<td>+ +</td>
<td>+ + + +</td>
<td>+</td>
<td>Thrombin</td>
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<td>Peak drug levels</td>
<td>↑ ↑</td>
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<tr>
<td>CTP-A</td>
<td>↑ ↑</td>
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<td>CTP-B</td>
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<td>Drug exposure (AUC)</td>
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<tr>
<td>CTP-A</td>
<td>↑ +</td>
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<td>CTP-B</td>
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<tr>
<td>Effect on INR</td>
<td>↑</td>
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<tr>
<td>CTP-A</td>
<td>↑ ↑</td>
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<tr>
<td>CTP-B</td>
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<tr>
<td>Renal clearance</td>
<td>33%</td>
<td>25%</td>
<td>100%</td>
<td>80%</td>
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<tr>
<td>Reversal (FDA appr)</td>
<td>Andexanet</td>
<td>Andexanet</td>
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<td>Idarucizumab</td>
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Comparison Different Anticoagulants for PVT in Cirrhosis

<table>
<thead>
<tr>
<th></th>
<th>DOACs</th>
<th>LMWH</th>
<th>Warfarin</th>
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<tr>
<td><strong>Safety</strong></td>
<td>Appear as safe as warfarin and LMWH</td>
<td>Safety risks well-documented</td>
<td>Safety risks well-documented</td>
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<tr>
<td><strong>Clinical Experience</strong></td>
<td>Short track record: Approved in 2010 (dabigatran)</td>
<td>Long track record: Approved in 1993</td>
<td>Longest track record: Approved in 1954</td>
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<tr>
<td><strong>Efficacy</strong></td>
<td>May be more effective than warfarin for PVT resolution</td>
<td>May be more effective than warfarin for PVT resolution</td>
<td>May be more effective than warfarin for PVT resolution</td>
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<td><strong>Monitoring</strong></td>
<td>Marketed as not necessary, but more information is needed</td>
<td>Not necessary</td>
<td>Lifelong</td>
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<td><strong>Wash out period</strong></td>
<td>~2 d</td>
<td>~2–3 d</td>
<td>~5–7 d</td>
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<td><strong>FDA–approved for indication</strong></td>
<td>No</td>
<td>No</td>
<td>No</td>
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<td><strong>Antidote</strong></td>
<td>Expensive and not widely available</td>
<td>Expensive and not widely available</td>
<td>Cheap antidote and widely available</td>
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<td><strong>Route of administration</strong></td>
<td>Oral</td>
<td>Injection</td>
<td>Oral</td>
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<tr>
<td><strong>Pharmacokinetic studies in cirrhosis published</strong></td>
<td>Yes</td>
<td>No</td>
<td>No</td>
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<tr>
<td><strong>Hepatotoxicity</strong></td>
<td>Rates vary with DOAC – highest with rivaroxaban</td>
<td>Extremely rare</td>
<td>Rare</td>
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<tr>
<td><strong>Antifibrotic effects and evidence</strong></td>
<td>Possible, animal studies</td>
<td>Possible, clinical trial</td>
<td>Possible, animal studies</td>
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</table>
Not Candidates for Full Anticoagulation

- More advanced liver disease
  - Childs C, High MELD (? >18)
  - Not enough data to determine safety
- Plts <50,000
  - Increased risk bleeding (not enough plt mass for thrombin burst?)
- Risk of falls
  - Unsteady, history of falls, episodes of PSE
- Patients high risk varices, GAVE, gastropathy
  - Eligible can be treated
- HCC out of Milan
  - Risk benefit ratio is poor
- Creatinine Clearance <30 ml/min
Acute Kidney Injury in Decompensated Cirrhosis

**Decompensated Cirrhosis** = Fragile Hemostatic Rebalance

**Decompensated Cirrhosis With Aki** = Unstable Hemostatic Balance (May Easily Shift Towards Either Hypo- or Hypercoagulability)

Case Study

- JD is a 50-year-old male with decompensated liver disease from alcohol was admitted for esophageal variceal bleeding. Patient’s liver disease has been complicated by PVT, was on Eliquis and this was discontinued. He underwent EGD and had 10 EV banding patient

- Next day: Underwent TIPS

- Patient developed AKI, was on CRRT; HD

- Patient improved 2 weeks later; off HD. US doppler of the liver showed partial PVT
Case Study

- 3 months later, patient’s MELD 16. Creatinine 1.6; GFR <30 ml/min
- Started on Eliquis, renally-dosed 2.5 mg twice a day
- Currently awaiting LT; US Doppler of liver in 3-6 months to reassess PVT
Q&A


