Managing Anticoagulation in the Hospitalized Patient

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Financial Disclosures—NONE
Objectives

- Know when to restart anticoagulation after a bleed
- Appreciate limitations in data for DOACs at extremes of weight
- Understand place of reduced dosing of DOACs in AFIB
- Develop approach to patient bleeding on DOAC
- Review evidence on dual therapy with DOAC in PCI

THESE SHOULD BE AT YOUR FINGERTIPS

**EXPERT CONSENSUS DECISION PATHWAY**

2017 ACC Expert Consensus Decision Pathway for Periprocedural Management of Patients with Atrial Fibrillation

Updated European Heart Rhythm Association Practical Guide on the use of non-vitamin K antagonist anticoagulants in patients with non-valvular atrial fibrillation

What To Do After the Bleed

76 y/o man with CAD (NSTEMI 2006), AFIB CHADS-Vasc=4 on warfarin and ASA is admitted with UGIB. INR is 3.0. He requires 3u PRBCs, vit K and FFP. EGD shows peptic ulcer disease. He is started on high dose PPI therapy, bx for H Pylori done. When should his anticoagulation be restarted?

a) Never
b) In two weeks
c) In three months
d) Let the primary provider deal with this one
What To Do After the Bleed

Witt Hematology 2016
What To Do After the Bleed

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“Two weeks may provide the best balance among GI bleed recurrence, thromboembolism and mortality”
AC FORUM Clinical Guidance
Antithrombotic Therapy for VTE

“In the event of GI bleed we suggest waiting at least 7 days without evidence of active bleeding and after endoscopic tx before reinitiating AC”

GIBs: DOACs vs Warfarin

Table 2: GIBs in studies on patients with AF. DOAC vs warfarin.

<table>
<thead>
<tr>
<th>DOAC</th>
<th>GIBs (events)</th>
<th>Life-threatening GIB events (events)</th>
<th>Total pts (n)</th>
<th>Life-threatening GIB events (n)</th>
<th>Total pts (n)</th>
<th>DOAC/W</th>
<th>RR (95% CI) GIB (years-pts)</th>
<th>DOAC/W</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rivaroxaban 20 mg twice daily arm</td>
<td>132</td>
<td>67</td>
<td>60/5</td>
<td>120</td>
<td>57</td>
<td>60/2</td>
<td>1.12 (1.02-1.23)</td>
<td>p = 0.001</td>
</tr>
<tr>
<td>Rivaroxaban 10 mg twice daily arm</td>
<td>192</td>
<td>94</td>
<td>60/5</td>
<td>120</td>
<td>57</td>
<td>60/2</td>
<td>1.15 (1.05-1.26)</td>
<td>p = 0.001</td>
</tr>
<tr>
<td>Edoxaban 30 mg once daily arm</td>
<td>224</td>
<td>52 (1 fatal event)</td>
<td>71/1</td>
<td>154</td>
<td>47 (3 fatal events)</td>
<td>71/2</td>
<td>2.00 (1.04-3.84)</td>
<td>p = 0.039</td>
</tr>
<tr>
<td>Apixaban 5 mg twice daily arm</td>
<td>115</td>
<td>ND</td>
<td>50/0</td>
<td>150</td>
<td>ND</td>
<td>50/2</td>
<td>1.00 (0.00-1.00)</td>
<td>p = 0.999</td>
</tr>
<tr>
<td>Edoxaban 60 mg once daily arm</td>
<td>232</td>
<td>ND</td>
<td>70/0</td>
<td>190</td>
<td>ND</td>
<td>70/2</td>
<td>1.23 (1.00-1.50)</td>
<td>p = 0.043</td>
</tr>
</tbody>
</table>

ND: not determined.

Table 2: GIBs in studies on patients with AF: DOACs vs warfarin.


<table>
<thead>
<tr>
<th>DOAC</th>
<th>Warfarin</th>
<th>DOAC/W</th>
<th>RR (95% CI) GIB DOAC/W</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nadroglutirax 100 mg twice daily arm</td>
<td>133</td>
<td>67</td>
<td>60/5</td>
</tr>
<tr>
<td>Nadroglutirax 150 mg twice daily arm</td>
<td>182</td>
<td>94</td>
<td>60/5</td>
</tr>
<tr>
<td>Apixaban 5 mg twice daily arm</td>
<td>105</td>
<td>ND</td>
<td>50/8</td>
</tr>
<tr>
<td>Edoxaban 30 mg once daily arm</td>
<td>150</td>
<td>ND</td>
<td>70/12</td>
</tr>
<tr>
<td>Edoxaban 60 mg once daily arm</td>
<td>252</td>
<td>ND</td>
<td>70/12</td>
</tr>
</tbody>
</table>

ND: not determined.

**Resumption of DOACs**

Anticoagulation FULLY therapeutic within 1-2 hours

Only dabigatran has a reversal agent

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**Considerations After GIB on AC**

- Reassess risk benefit of anticoagulation secondary prevention of VTE therapy, low CHADS-vasc
- Assess risk of rebleeding from source identifiable source, treatable lesion?
- Take steps to decrease risk of bleeding related to AC regimen
- Reconsider need for antiplatelet therapy if warfarin-was INR in range, is control good? spurious elevation in INR or poor TTR → DOAC increase INR monitoring→home POC INR?
- If ongoing strong indication for AC determine best time to restart therapy in multidisciplinary meeting with proceduralist - Remember DOAC immediately active
What To Do After the Bleed

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DOACs in Extremes of Weight

A 56 year old obese man, BMI 42, weight 155 kg presents with bilateral lower extremity swelling and SOB. D-Dimer is elevated CTa shows multiple bilateral segmental PE. What anticoagulant regimen do you recommend?

a) Rivaroxaban 15 mg BID x21 days then 20 mg daily
b) Apixaban 10 mg BID x 7 days then 5 mg BID
c) Enoxaparin bridging to warfarin
d) Admission for IV heparin bridging to warfarin
DOACs in Extremes of Weight

- Systematic review of 6 trials of DOACS vs warfarin in VTE
- Proportion of patients classified as high body weight (15-28%)
- Variability may be related to definition (e.g., > 90 kg vs > 100 kg)
- Very little information on extreme body weight (e.g., < 40 kg, > 150 kg)


DOACs in Obesity

**RECOMMENDATIONS AND GUIDELINES:**

*Use of the direct oral anticoagulants in obese patients: guidance from the SSC of the ISTH*

K. Martin,*, J. Beyer-Westendorf,† B. L. Davidson,‡ M. V. Huisman,§ P. M. Sandset*, and S. Moll*

*Department of Medicine, Division of Hematology-Oncology, University of North Carolina, Chapel Hill, NC, USA; †Thrombosis Research Unit, Center for Vascular Biology, University Hospital, Technische Universität Dresden, Dresden, Germany; ‡Division of Pulmonary and Critical Care Medicine, University of Washington School of Medicine, Seattle, WA, USA; and §Department of Thrombosis and Haemostasis, Leiden University Medical Center, Leiden, the Netherlands.*

**Indications:**

- **Reduced exposure, lower peaks, shorter t 1/2**

**ISTH RECOMMENDATIONS:**

- **Recommend standard dosing if BMI< 40 and weight < 120 kg.**
- **Suggest DOACS not be used if BMI> 40 or weight > 120 kg**
- **If DOACs used in BMI > 40 or weight> 120 kg suggest drug specific peak and trough level. If level within expected range continue DOAC; if below suggest warfarin**

Martin et al. Journal of Thrombosis and Haemostasis, 2016 14: 1308–1313

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DOAC Dose Reduction

78 year old man with HTN CHF CKD is found to be in AFIB. His creatinine is 1.6. He weighs 61 kgs. He has a remote history of GI bleeding. What dose of apixaban do you recommend?

1. Apixaban 5 mg BID
2. Apixaban 2.5 mg BID
3. How about we start anticoagulation tomorrow when it is no longer my shift?
U.S. FDA Labeling Guidelines for DOAC Dose Reduction

<table>
<thead>
<tr>
<th>AF</th>
<th>VTE Treatment/Recurrent VTE Prevention</th>
<th>Orthopedic Surgery VTE Prophylaxis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Usual dose</td>
<td>Dose reductions</td>
</tr>
<tr>
<td>Dabigatran</td>
<td>150 mg BID</td>
<td>1. 75 mg BID if CrCl 15-30 mL/min</td>
</tr>
<tr>
<td></td>
<td>2. 75 mg BID if CrCl 30-50 mL/min</td>
<td>220 mg once daily</td>
</tr>
<tr>
<td>Rivaroxaban</td>
<td>20 mg once daily</td>
<td>15 mg once daily if CrCl 15-50 mL/min</td>
</tr>
<tr>
<td>Apixaban</td>
<td>5 mg BID</td>
<td>1. 2.5 mg BID if at least two of the following: A. Age ≥ 80 years B. Weight ≤ 60 kg C. SCr ≥ 1.5</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2. 50% dose ↓ if taking strong dual p-gp/CYP3A4 inhibitors</td>
</tr>
<tr>
<td>Edoxaban</td>
<td>60 mg once daily</td>
<td>30 mg once daily if CrCl 15-50 mL/min</td>
</tr>
</tbody>
</table>


DOAC Dose Reduction

- A DOAC dose reduction contrary to FDA labeling was made in > 50% patients-apixaban had highest rate of discrepancy
- History of prior bleeding may have influenced reduction-25% had prior bleed
- Rate of TE was similar to no therapy (10% apix, 5% dabi 3.6% riva)
- Despite lower doses of DOAC there was a high rate of bleeding-20%, ? Due to concomitant antiplatelet therapy (80% on those with bleeds on antiplatelet)


DOAC Dose Reduction

Off-Label Dosing of Non-Vitamin K Antagonist Oral Anticoagulants and Adverse Outcomes
The ORBIT-AF II Registry

Steinberg BA et al. JACC 2016;68:2597-2604
Steinberg BA et al. JACC 2016;68:2597-2604

DOAC Dose Reduction

Apixaban 5 mg Twice Daily and Clinical Outcomes in Patients With Atrial Fibrillation and Advanced Age, Low Body Weight, or High Creatinine

Patients with AFIB & isolated advanced age, low body weight, or renal dysfunction have higher risk of stroke/SE & major bleeding but show consistent benefits with 5 mg twice daily apixaban vs warfarin compared with patients without these characteristics
78 year old man with HTN CHF CKD is found to be in AFIB. His creatinine is 1.6. He weighs 61 kgs. He has a remote history of GI bleeding. What dose of apixaban do you recommend?

1. Apixaban 5 mg BID
2. Apixaban 2.5 mg BID
3. How about we start anticoagulation tomorrow when it is no longer my shift?

Reducing the Hospital Burden of HIT

- Not uncommon
  - HIT occurs in 5% of those exposed to UFH
  - Risk reduced 10 fold with LMWH
- High morbidity and mortality
  - Thromboembolic complications 20-50%
- Expensive
Avoid Heparin Protocol

- Systematic replacement of most IV and SQ UFH with SQ LMWH in prophylactic or therapeutic doses (remaining uses of UFH were for hemodialysis, intraoperative use for cardiovascular surgery, ACS)
- Replacement of heparinized saline in arterial and central venous lines with saline flushes
- Modification of order sets to exclude UFH options
- Removal of UFH stores from most nursing units.
Avoid Heparin Protocol

~40% reduction in suspected HIT
~80% reduction in HIT
~80% reduction in HIT related costs

Bleeding on DOAC

A 65 year old man with AFIB CHADS-Vasc=5 on rivaroxaban for stroke prevention presents with melena, BP 120/80, HR 99, HCT 30 (baseline 40). PT 18. INR 2.5. How do you manage his anticoagulation?

1. hold rivaroxaban and transfuse PRBCs & IV fluids as needed
2. hold rivaroxaban and administer PCC
3. hold rivaroxaban and transfuse FFP and vitamin K
4. Where is that protocol? I know we have one somewhere.
Bleeding on DOAC

- Is drug still present?
  - When was last dose of drug?
  - What is patient’s renal function?
  - Will laboratory data help?
- If present should drug be reversed?

Timing of Last Dose

<table>
<thead>
<tr>
<th>Property</th>
<th>Dabigatran&lt;sup&gt;1&lt;/sup&gt;</th>
<th>Rivaroxaban&lt;sup&gt;2&lt;/sup&gt;</th>
<th>Apixaban&lt;sup&gt;3&lt;/sup&gt;</th>
<th>Edoxaban&lt;sup&gt;4&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Est t ½ hrs</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CrCl &gt; 80</td>
<td>14</td>
<td>8</td>
<td>7-8</td>
<td>8-9</td>
</tr>
<tr>
<td>CrCl 30-79</td>
<td>17-19</td>
<td>9</td>
<td>17-18</td>
<td>9-10</td>
</tr>
<tr>
<td>CrCl 15-30</td>
<td>28</td>
<td>10</td>
<td></td>
<td>17</td>
</tr>
<tr>
<td>Renal clearance of absorbed dose</td>
<td>80%</td>
<td>35%</td>
<td>27%</td>
<td>50%</td>
</tr>
<tr>
<td>Approximate anticoagulation resolution&lt;sup&gt;a&lt;/sup&gt; (nml renal fxn)</td>
<td>Day 2.5–3.5 after last dose</td>
<td>Day 1.5–3.5 after last dose</td>
<td>Day 1–2 after last dose</td>
<td>Day 1.3–2 after last dose</td>
</tr>
</tbody>
</table>

<sup>a</sup>Estimated as the time it takes for 5 half-lives to elapse since the last dose
**Interpreting Lab Tests on DOACS**

<table>
<thead>
<tr>
<th>Drug</th>
<th>High aPTT (clinically significant)</th>
<th>Normal PTT (rules out supratherapeutic drug levels, does not rule out clinically significant residual AC effect)</th>
<th>Normal dTT (rules out residual dabigatran)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dabigatran</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rivaroxaban</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Apixaban</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

- Elevated PT (clinically significant levels (rivaroxaban only))
- Normal PT (rules out supratherapeutic drug levels, does not rule out clinically significant residual AC effect)
- In a pinch can use UFH or LMWH level. If negative it is a reasonable surrogate for excluding above trough drug levels


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**Managing DOAC Bleeding: EHRA 2015 Recommendations**

- **MILD BLEEDING**
  - Delay/discontinue next dose
  - Reconsider concomitant medication, especially antiplatelets

- **MODERATE TO SEVERE BLEEDING**
  - **Supportive measures**
    - Mechanical compression
    - Endoscopic hemostasis if GI bleed
    - Surgical hemostasis
    - Fluid replacement (colloids if needed)
    - RBC substitution if needed
    - FFP (as plasma expander)
    - Platelet substitution if platelet count ≤ 60 x 10⁹/L

- **LIFE-THREATENING BLEEDING**
  - **Consider**
    - PCC (eg, CoFact®) 50 U/kg; +25 U/kg if indicated
    - aPCC (Feiba®) 50 U/kg; max 200 U/kg/d
    - rFVIIa (NovoSeven®) 90 µg/kg (no data about additional benefit)
    - For dabigatran, administer idarucizumab 5 g IV

Heidbuchel H et al. Europace. 2015. 40
A 65 year old man with AFIB CHADS-Vasc=5 on rivaroxaban for stroke prevention presents with melena, BP 120/80, HR 99, HCT 30 (baseline 40). PT 18. INR 1.5. How do you manage his anticoagulation?

1. hold rivaroxaban and transfuse PRBCs & IV fluids as needed
2. hold rivaroxaban and administer PCC
3. hold rivaroxaban and transfuse FFP and vitamin K
4. I knew we should have had a protocol for this. Didn’t she mention at last year’s conference?
DUAL Therapy with DOAC

A 68 yo man with AFIB, DM, HTN on dabigatran, ASA and metoprolol presents with NSTEMI. Cardiac cath shows 90% occlusion in left circumflex, he has PCI with drug eluting stent. You are preparing him for discharge. What antithrombotic regimen should he be discharged on?

1) Triple therapy with dabigatran, clopidigrel and ASA
2) Dual therapy with dabigatran and clopidigrel
3) Dual therapy with clopidigrel and ASA-restart dabigatran after dropping one of the antiplatelet agents
4) Whatever cardiology tells me to do

DUAL THERAPY WITH DOAC-PCI

AFIB and PCI for ACS or CAD
110 dab + PGY12 inhibitor; 150 mg dab + PGY 12 inhibitor or triple therapy with warfarin ASA plus PGY 12 inhibitor
1° endpoint-major and CRNM bleeding
2° endpoint composite of TE, death or revasculation
DUAL THERAPY WITH DOAC-PCI

Dual therapy with dabigatran and PGY12 inhibitor resulted in lower risk of bleeding with non inferior Rates of TE when compared to triple therapy.

DUAL Therapy with DOAC

A 68 yo man with AFIB, DM, HTN on dabigatran, ASA and metoprolol presents with NSTEMI. Cardiac cath shows 90% occlusion in left circumflex, he has PCI with drug eluting stent. You are preparing him for discharge. What antithrombotic regimen should be discharged on?

1) Triple therapy with dabigatran, Plavix and ASA
2) Dual therapy with dabigatran and Plavix
3) Dual therapy with Plavix and ASA-restart dabigatran after dropping one of the antiplatelet agents
4) Whatever cardiology tells me to do
Thrombophilia Testing

A 54 year old man presents with sudden onset chest pain and shortness of breath. D-dimer is elevated and CT chest shows bilateral PE. He has no past medical history and denies recent trauma, travel or surgery. Should he have a thrombophilia work up for this unprovoked VTE event?

- Yes
- No
- More coffee, please?
Clinical Characteristics Suggestive of Inherited Thrombophilia in Patients with Venous Thromboembolism (VTE).

Thrombosis at a young age (<50 yr), especially in association with weak provoking factors (minor surgery, combination oral contraceptives, or immobility) or unprovoked VTE

Strong family history of VTE (first-degree family members affected at a young age)

Recurrent VTE events, especially at a young age*

VTE in unusual sites such as splanchnic or cerebral veins†

* The antiphospholipid syndrome must also be considered, but it is not inherited.
† Patients with splanchnic-vein VTE should be assessed for myeloproliferative neoplasms and paroxysmal nocturnal hemoglobinuria.

Table 1. Summary of Recommendations Regarding Testing for Thrombophilia.*

<table>
<thead>
<tr>
<th>Recommendation</th>
<th>Explanation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Do not test if VTE is provoked by strong risk factors</td>
<td>Strong risk factors are major trauma, major surgery, immobility, major illness</td>
</tr>
<tr>
<td>Do not test while patient is receiving anticoagulant therapy</td>
<td>Test at completion of anticoagulant therapy for provoked VTE, or unprovoked VTE, or after treatment for acute event if cessation of anticoagulant therapy is contemplated, and test results might change management strategy</td>
</tr>
<tr>
<td>Consider testing</td>
<td>Test when VKA has been stepped for at least 2 wks, DOAC has been stepped for at least 2 days (preferably longer), and UFH or LMWH for anti-factor Xa levels has been stepped for more than 24 hr</td>
</tr>
<tr>
<td>Identify goals of testing</td>
<td>Identify goals in order to aid decision making regarding future VTE prophylaxis, to guide testing of family members (especially regarding risk associated with COC or pregnancy in female family members), and to determine cause (especially for severe VTE, fatal VTE in family members, or VTE in an unusual location); test results alone should not be used for decision making regarding duration of anticoagulant therapy</td>
</tr>
</tbody>
</table>

*COC denotes combination oral contraceptives, DOAC direct oral anticoagulant, LMWH low-molecular-weight heparin, UFH unfractionated heparin, and VKA vitamin K antagonist.
Table 1. Thrombophilia Tests and Prevalence of Risk Factors.

<table>
<thead>
<tr>
<th>Thrombophilia Type</th>
<th>Assay</th>
<th>Prevalence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inherited</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Factor V Leiden</td>
<td>APCR, PCT</td>
<td>White: 5.0%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hispanic: 3.2%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Black: 1.2%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Native American: 0.3%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Asian: 0.6%</td>
</tr>
<tr>
<td>Prothrombin gene mutation</td>
<td>PCT</td>
<td>White: 3%</td>
</tr>
<tr>
<td>Decreased anticoagulant activity</td>
<td>Activity assay</td>
<td>Protein C: &lt;0.3%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Protein S: &lt;0.3%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Antithrombin: &lt;0.3%</td>
</tr>
<tr>
<td>Acquired</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lupus anticoagulant</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Information on prevalence for Factor V Leiden is from Roffe et al. for prothrombin gene mutation is from Roffe et al., for protein C, protein S, and antithrombin is from Middeldorp et al., and for lupus anticoagulant is from van der Poll et al. APCR, activated protein C resistance; PCT, protein C activity; PCC, protein C concentration; PTT-LA, partial thromboplastin time-lupus anticoagulant; and SD, standard deviation.

Up to 35% of healthy people have positive antiphospholipid tests with no apparent clinical significance. Tests are positive in 10 to 12% of patients with VTE and in up to roughly 15% of patients with SLE who do not have VTE (up to 80% in some studies).
A 54 year old man presents with sudden onset chest pain and shortness of breath. D-dimer is elevated and CT chest shows bilateral PE. He has no past medical history and denies recent trauma, travel or surgery. Should he have a thrombophilia work up for this unprovoked VTE event?

- Yes
- No
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**Take Home Points**

- After GIB on AC-reconsider risk benefit of therapy; multidisciplinary approach to determining when to restart AC after bleeding event
- Caution using DOACs in morbidly obese pending additional data
- Adhering to FDA labeling for DOACs may provide better outcomes in AFIB patients
- Consider renal function and timing of last dose to determine residual anticoagulant effect in patient bleeding on DOAC. No evidence-based reversal agents with exception of idarucizumab for dabigatran
- Have institutional protocol for reversal of all anticoagulants
- Avoid thrombophilia work up in hospitalized patients
WORKSHOP

- IVC filters
- Incidental PE
- Does this patient need to be bridged?
- Thrombophilia work up
- Management of patient with recurrent VTE despite therapeutic anticoagulation
- Calf vein thrombosis, superficial vein thrombosis, PICC line thrombosis and more

THE END