MY APPROACH to the use of NOACs for stroke prevention in patients with atrial fibrillation
Lip, Gregory

DOI:
10.1016/j.tcm.2014.05.012

License:
Other (please specify with Rights Statement)

Document Version
Peer reviewed version

Citation for published version (Harvard):
Lip, GYH 2014, 'MY APPROACH to the use of NOACs for stroke prevention in patients with atrial fibrillation', Trends in Cardiovascular Medicine, vol. 24, no. 6, pp. 265-266. https://doi.org/10.1016/j.tcm.2014.05.012

Link to publication on Research at Birmingham portal

Publisher Rights Statement:
NOTICE: this is the author’s version of a work that was accepted for publication in Trends in Cardiovascular Medicine. Changes resulting from the publishing process, such as peer review, editing, corrections, structural formatting, and other quality control mechanisms may not be reflected in this document. Changes may have been made to this work since it was submitted for publication. A definitive version was subsequently published in Trends in Cardiovascular Medicine [VOL 24, ISSUE 4, August 2014] DOI: 10.1016/j.tcm.2014.05.012

Eligibility for repository checked October 2014

General rights
Unless a licence is specified above, all rights (including copyright and moral rights) in this document are retained by the authors and/or the copyright holders. The express permission of the copyright holder must be obtained for any use of this material other than for purposes permitted by law.

- Users may freely distribute the URL that is used to identify this publication.
- Users may download and/or print one copy of the publication from the University of Birmingham research portal for the purpose of private study or non-commercial research.
- Users may use extracts from the document in line with the concept of 'fair dealing' under the Copyright, Designs and Patents Act 1988 (?)
- Users may not further distribute the material nor use it for the purposes of commercial gain.

Where a licence is displayed above, please note the terms and conditions of the licence govern your use of this document.

When citing, please reference the published version.

Take down policy
While the University of Birmingham exercises care and attention in making items available there are rare occasions when an item has been uploaded in error or has been deemed to be commercially or otherwise sensitive.

If you believe that this is the case for this document, please contact UBIRA@lists.bham.ac.uk providing details and we will remove access to the work immediately and investigate.

Download date: 21. Mar. 2019
MY APPROACH to the use of NOACs for stroke prevention in Patients with atrial fibrillation

Gregory Y H Lip MD, FRCP, DFM, FACC, FESC

PII: S1050-1738(14)00046-2
DOI: http://dx.doi.org/10.1016/j.tcm.2014.05.012
Reference: TCM6000

To appear in: trends in cardiovascular medicine

Cite this article as: Gregory Y H Lip MD, FRCP, DFM, FACC, FESC, MY APPROACH to the use of NOACs for stroke prevention in Patients with atrial fibrillation, trends in cardiovascular medicine, http://dx.doi.org/10.1016/j.tcm.2014.05.012

This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting galley proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.
MY APPROACH to the Use of NOACs for Stroke Prevention in Patients With Atrial Fibrillation

Gregory Y H Lip MD, FRCP, DFM, FACC, FESC

Professor of Cardiovascular Medicine, University of Birmingham, UK; Visiting Professor of Haemostasis Thrombosis and Vascular Sciences, Aston University, Birmingham, UK; Adjunct Professor in Cardiovascular Sciences, Aalborg University, Denmark; Visiting Professor of Cardiology, University of Belgrade, Serbia

Commentary

Stroke prevention is central to the management of atrial fibrillation (AF). The approach is to initially identify patients at a low risk for stroke (CHA2DS2-VASc score of 0 [men] or 1 [women]), who do not need any antithrombotic therapy. Subsequent to this step, patients with ≥ additional stroke risk factors (thus, CHA2DS2-VASc score of ≥2, as well as men with a score of 1) can be offered effective stroke prevention.

Effective stroke prevention essentially means oral anticoagulation (OAC), whether given as a well-controlled, adjusted-dose vitamin K antagonist (VKA; eg, warfarin), or one of the non-VKA oral anticoagulants (NOACs; previously referred to as new, or novel, OACs1). If VKAs are used, the challenge is to maintain patients within an international normalized ratio (INR) of 2.0 to 3.0. Guidelines recommend good-quality anticoagulation control, as reflected by an average individual time in therapeutic range (TTR) of ≥70%.2,3 The TTR correlates with the efficacy and safety of VKAs whereby a high TTR is associated with low risks for thromboembolism and bleeding, but a low TTR (ie, <60%) is associated with a high risk for thromboembolism or hemorrhage.3,4,5

For newly diagnosed anticoagulation-naïve patients with AF, use of the SAMe-TT2R2 score can help identify those patients (SAMe-TT2R2 score of 0–2) who are likely to do well with a VKA (with TTR ≥70%). On the other hand, patients with a SAMe-TT2R2 score >2 are less likely to do well with a VKA and require more intense review and INR monitoring; therefore, they may be better off with an NOAC.5 We do not practice a "warfarin stress test" whereby patients are initiated with a VKA for 3 to 6 months, and only if the TTR is suboptimal would they be permitted to switch to an NOAC. Such an approach leads to the initial period whereby patients in the inception cohort would have suboptimal TTRs and would lead to a substantial risk for fatal and devastating strokes.6

In an established patient who is already receiving anticoagulation with a VKA, clinical follow-up includes a review of the INRs recorded in the anticoagulation record booklet. If the TTR is suboptimal (ie, <60% despite efforts to improve anticoagulation control by our clinic), the patient would be considered for switching from a VKA to an NOAC. The VKA is stopped, the INR is
allowed to decrease to approximately 2.0, and the NOAC is started.\textsuperscript{7,8} Because all of the NOACs have a fast onset of action, no bridging is necessary.

With the availability of a number of NOACs, prescribers are now spoiled regarding choice, and we can fit the drug to a particular patient profile (and vice versa). For example, in a patient with a high risk for bleeding (HAS-BLED score $\geq 3$), the NOACs that have a safer bleeding profile (eg, dabigatran 110 mg twice daily or apixaban) can be used. When renal function is impaired (eg, creatinine clearance $\approx 30 \text{ mL/L}$), it would be prudent to use an NOAC that is not so dependent on renal excretion, such as apixaban and rivaroxaban. Dabigatran should be avoided, given the high renal excretion of this drug. For maximal potency in reducing ischemic stroke (eg, in a patient with recurrent strokes despite reasonable TTR), dabigatran 150 mg twice daily can be considered.

My patients starting with any OAC are given a counseling or education session, focused on compliance and precautions about use of anticoagulants. Part of the counseling process is drug-specific (eg, advice not to take dabigatran on an empty stomach to avoid any dyspepsia). Also, rivaroxaban needs to be taken with food. Follow-up at intervals requires assessment of any changes in the clinical situation (eg, bleeding problems) and monitoring of renal function.

### Abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>SAMe-TT$_2$R$_2$</td>
<td>Sex female; Age $&lt;60$ years; Medical history (&gt;2 comorbidities); Treatment (interacting medications; eg, amiodarone); Tobacco use (doubled); Race (doubled)</td>
</tr>
<tr>
<td>CHADS$_2$</td>
<td>Congestive heart failure; Hypertension; Age $\geq 75$ years; Diabetes; previous Stroke</td>
</tr>
<tr>
<td>CHA$_2$DS$_2$-VASc</td>
<td>Congestive heart failure, Hypertension, Age $\geq 75$ years, Diabetes, previous Stroke, Vascular disease, Age 65-74 years, Sex category (female)</td>
</tr>
<tr>
<td>HAS-BLED</td>
<td>Hypertension; Abnormal renal/liver function; Stroke; Bleeding history or predisposition; Labile international normalized ratio; Elderly ($&gt;65$ years); Drugs/alcohol concomitantly</td>
</tr>
<tr>
<td>VKA</td>
<td>Vitamin K antagonist</td>
</tr>
<tr>
<td>TTR</td>
<td>Time in therapeutic range</td>
</tr>
<tr>
<td>OAC</td>
<td>Oral anticoagulation</td>
</tr>
</tbody>
</table>
NOAC | Non-VKA oral anticoagulant

References


Corresponding author: g.y.h.lip@bham.ac.uk

First published on PracticeUpdate on May 19, 2014. Republished with permission.