What is new in the 2016 European Society of Cardiology atrial fibrillation guidelines?

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INTRODUCTION

The present review seeks to highlight important changes in the recently published atrial fibrillation (AF) guidelines of the European Society of Cardiology (ESC) in comparison to previous recommendations, which are summarized as follows: (1)

Key words: atrial fibrillation, anticoagulation, cardioversion, multidisciplinary heart team

RATE AND RHYTHM CONTROL

Acute Rate Control

Acute rate control is essential for patients with depressed left ventricular ejection fraction (LVEF <40%) or signs of congestive heart failure (HF) by beta-blockers in the smallest dose necessary to achieve rate control below 110 BPM and/or digoxin or digitoxin. In contrast to previous guidelines, amiodarone is recommended as the last option in patients with severely reduced LV function and/or hemodynamic instability, while verapamil and diltiazem should be avoided due to their negative inotropic effects in patients with pre-existing HF. (1, 2)

Long-term Rate Control

LVEF has been similarly implemented as criterion for the selection of agents for long-term rate control: Beta-blockers and/or digoxin (digitoxin) are recommended in AF patients with a LVEF <40%, while in patients with a LVEF ≥ 40%, beta-blockers, digoxin, verapamil or diltiazem are equally recommended. A heart rate of <110 BPM is the true goal for therapy, since a more difficult to achieve BPM goal of <80 had no positive impact on cardiovascular events. (1)

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ORAL ANTICOAGULATION

Based on the CHA2DS2-VASc score, oral anticoagulation (OAC) is recommended in male AF patients with a CHA2DS2-VASc score of 2 or more, and female AF patients with a CHA2DS2-VASc score of 3 more, and should be considered in the presence of a single additional stroke risk factor, i.e. a score of 1 in men and 2 in women. Patients with mechanical heart valves should receive a vitamin K antagonist (VKA), regardless of estimated stroke risk. (1)

Choice of the Anticoagulant

Due to the beneficial efficacy/safety profile, Non-Vitamin K antagonist oral anticoagulants (NOACs) are now recommended for stroke prevention and preferred over VKA. (1, 5) In principle all four clinically available NOACs (dabigatran, rivaroxaban, apixaban, edoxaban) can be used interchangeably, although certain patient-related characteristics might support the use of a specific NOAC (e.g. dabigatran in patients with high bleeding risk and recurrent falls, as this agent has a specific antidote, see later).

BAN OF ANTIPLATELET THERAPY

Antiplatelet monotherapy is now contraindicated for stroke prevention in patients with non-valvular AF, irrespective of the CHA2DS2-VASc Score, as is platelet inhibition on top of OAC for secondary prevention after stroke or transient ischemic attack (TIA). (1)

TRIPLE OR DUAL ANTITHROMBOTIC THERAPY IN SECONDARY PREVENTION

The strategy for antithrombotic combination therapy (OACs plus one = dual therapy; or plus two antiplatelet agents = triple therapy) in AF patients with acute coronary syndromes (ACS) or after elective coronary stent-implantation is mostly based on expert opinion. Duration of triple therapy consisting of an OAC, acetylsalicylic acid (ASA) and clopidogrel (the P2Y12-inhibitors prasugrel an ticagrelor are currently contraindicated together with OAC) should be performed as short as possible (1 month in stable/elective patients, 1-3-6 months depending on the individual bleeding- and atherothrombotic risk in ACS patients), followed by dual therapy consisting of one antiplatelet agent in combination with either a VKA (INR goal 2.0-2.5) or with a NOAC in the lower tested dose (dabigatran 110mg BID, rivaroxaban 15 mg OD, apixaban 2.5 mg BID, edoxaban 30 mg OD), respectively. Six- to 12 months after PCI (or ACS without PCI), OAC monotherapy seems to sufficiently prevent (recurrent) coronary and thromboembolic events. The use of second generation modern drug eluting stents with a very low risk for stent thrombosis has to be preferred over bare metal stents and supports a short antithrombotic triple combination strategy for 1 month only. (4, 5)

RE-INITIATION OF OAC AFTER ISCHEMIC STROKE OR TIA

After TIA, OAC can be initiated 1 day after the event, if an intracerebral bleeding has been excluded by computer tomography or magnetic resonance imaging. For patients with ischemic stroke, the 3-6-12-day-rule might be applied („Diener’s law“). (1)

CARDIOVERSION UNDER NOACS

Two prospective randomized controlled trials (X-VeRT and ENSURE-AF) and post-hoc analyses of randomized controlled trials investigated the use of NOACs before, during and after cardioversion, as compared to warfarin. All studies confirmed the safety of NOACs in this indication and, although underpowered, suggested similar outcomes in terms of efficacy. (6, 7)

The initiation of either unfractionated heparin or a NOAC should be considered for acute cardioversion in AF or atrial flutter of >48 hours or unknown duration, while NOACs have not been tested for acute cardiodversion in patients with recent onset AF < 48 hours. In case of delayed cardioversion, effective OAC for minimum of 3 weeks is recommended, irrespective of the agent used. A transeosophageal echocardiography to exclude left trial thrombi is useful in certain indications e.g. if a patient has not received or fully taken the recommended anticoagulation prior to cardioversion. (1)

HOW TO MANAGE SEVERE ACTIVE BLEEDING UNDER ORAL ANTICOAGULANTS

Besides red blood cell support and local mechanical measures (if applicable), the combination of fresh frozen plasma (FFP) and prothrombin complex concentrates (PCC) has been shown to be helpful in intracerebral bleedings on a VKA. Both activated (e.g. FEIBA®) and non-activated (e.g. Beriplex®) PCCs reverse the anticoagulant effect of NOACs sufficiently and are therefore indicated if specific antidotes are not available. Recently, the humanized antibody idarucizumab, an effective antidote against dabigatran, has been introduced for clinical use in many countries. (8) Moreover, an antidot against FXa-inhibitors and heparins, andexanet alpha, is in phase-3 clinical testing. (9)

REFERENCES


