Two-year outcomes of UK patients newly diagnosed with atrial fibrillation: findings from the prospective observational cohort study GARFIELD-AF

INTRODUCTION
Atrial fibrillation (AF) increases the risk of ischaemic stroke fivefold and the risk of death twofold.1 AF-related strokes are more severe than strokes in people without AF and are more likely to be fatal, lead to long-term disability, extended hospital stays, and increased healthcare costs.2 Anticoagulation therapy reduces the risk of AF-related stroke (and systemic embolism) and death, with a 68% relative risk reduction for ischaemic stroke and a 25% reduction in the relative mortality.3 Anticoagulation, however, increases the risk of bleeding with the most serious complication being intracranial haemorrhage, which can be fatal.4

Anticoagulant drugs recommended by AF guidelines have included vitamin K antagonists (VKAs), usually warfarin, and direct-acting oral anticoagulants (DOACs), namely dabigatran, rivaroxaban, apixaban, and edoxaban.5 Anticoagulant use overall and good uptake of DOACs is limited.8–12 However, evidence on outcomes for UK patients newly diagnosed with AF at risk of stroke following the introduction of DOACs is limited.

This study investigates the 2-year event rates for non-haemorrhagic stroke/systemic embolism, all-cause mortality, and major bleeding in UK patients enrolled in the Global Anticoagulant in the FIELD — Atrial Fibrillation registry (GARFIELD-AF).

METHOD
Study design
GARFIELD-AF is a prospective, observational, international registry of adults aged 18 years newly diagnosed with AF.13 GARFIELD-AF was conducted in 35 countries worldwide, including the Americas, Europe, Africa, Asia-Pacific and the Middle East between 2010 and 2016. Inclusion criteria comprised five prospective sequential cohorts between 2011 and 2016. Inclusion criteria comprised

Since the introduction of DOACs in clinical practice several UK studies have reported the clinical management of AF and changes in prescribing patterns, indicating increases in anticoagulant use overall and good uptake of DOACs.13–15 However, evidence on outcomes for UK patients newly diagnosed with AF at risk of stroke following the introduction of DOACs is limited.

This study investigates the 2-year event rates for non-haemorrhagic stroke/systemic embolism, all-cause mortality, and major bleeding in UK patients enrolled in the Global Anticoagulant in the FIELD — Atrial Fibrillation registry (GARFIELD-AF).
males and females aged ≥18 years with a
ew diagnosis of non-valvular AF of up to
6 weeks before entry into the registry and an
investigator-determined risk factor for stroke,
meaning the risk factors for stroke were not
pre-specified in the protocol and left to the
clinical judgement of the site investigator.

Eligible patients were recruited
consecutively at participating sites to prevent
selection bias. All participants provided
informed consent. Patients were followed up
for a minimum of 2 years (study end) or the
occurrence of the event of interest, or loss
to follow-up, whichever came first. Patients
with transient AF, secondary to a reversible
cause, and patients for whom follow-up was
not possible were excluded.

Setting
The UK specific study recruited from primary
care, with 185 sites (GP practices) across the
country. Participants were enrolled between
June 2011 and August 2016. Data were
collected from participants’ primary care
records at baseline and at 4-month intervals
up to 24 months post-diagnosis using an
electronic case report file by trained local
site staff.

Variables
Data collected at baseline included patient
characteristics, medical history, and
antithrombotic therapy initiated at diagnosis.
The main outcomes were non-haemorrhagic
stroke and systemic embolism, major
bleeding, and death. Major bleeding was
defined as clinically overt bleeding associated
with:
• a fall in haemoglobin of ≥2 g/dL; or
• a transfusion of ≥2 units of packed red
blood cells or whole blood; or
• a critical site (intracranial, intraspinal,
intraocular, pericardial, intra-articular,
intramuscular with compartment
syndrome, and retroperitoneal); or
• a fatal outcome.

The full classification of bleeding events
can be found in Supplementary Box S1. Anticoagulant use was measured as
anticoagulation prescribed at diagnosis.
Anticoagulant use includes patients receiving
anticoagulants with an antiplatelet (that is
with or without an antiplatelet).

Statistical analysis
Continuous variables are expressed as mean
[standard deviation [SD]] and categorical
variables as frequency and percentage.
Use of antithrombotic therapy at baseline
was analysed by CHA2DS2-VASc and HAS-
BLED scores; HAS-BLED was modified
to exclude fluctuations in the international
normalised ratio as these data were not
available. Females with no other risk factors
were assigned a CHA2DS2-VASc score of 0.
The occurrence of the major outcomes non-
haemorrhagic stroke/systemic embolism,
major bleeding, and mortality are presented
using number of events and person–time
event rate per 100 person–years and
95% confidence intervals (CIs). Only the first
occurrence of each event was taken into
account.

A propensity score was applied using
an overlap weighting scheme to reduce
biased estimates of the treatment effect.
Weights were applied to Cox proportional
hazards models to estimate the effects of
the anticoagulant versus no anticoagulant
comparison on the occurrence of death,
non-haemorrhagic stroke/systemic embolism,
and major bleeding within
2 years of enrolment. This newly developed
method of overlap propensity weighting
avoids excluding patients (as with matching)
and gives the most weight to propensities
where there is equipoise. This applied
method overlaps weights and optimises
the efficiency of comparisons by defining
the population with the most overlap in the
covariates between treatment groups. This
scheme eliminates the potential for outlier
weights by avoiding a weight based on a
ratio calculation using values bounded by
0 and 1. Thus, when using overlap weights,
many of the concerns regarding the
assessment and the trimming of the weights
are eliminated.\(^15\) Covariates evaluated in the
weighting scheme included demographic
characteristics [sex, age, and ethnicity],
lifestyle factors [current smoking and alcohol
consumption], clinical measurements at

How this fits in
Atrial fibrillation (AF) increases the risk of
stroke and death; anticoagulation reduces
these risks at the cost of an increased risk
of bleeding. There has been an increase in
the proportion of patients with AF receiving
anticoagulants, with patients receiving
either vitamin K antagonists or direct-
acting oral anticoagulants. Evidence on
outcomes following the increase in the use
of anticoagulant therapy in the UK is limited.
In this study, the benefit of anticoagulation
in this real-world cohort of patients with
AF affirms recommendations in AF
management guidelines. Addressing gaps in
anticoagulation treatment for patients with
AF may reduce AF-related stroke and death.
diagnosis (body mass index, heart rate, and blood pressure), medical history (congestive heart failure, acute coronary syndromes, vascular disease, carotid occlusive disease, prior stroke/transient ischaemic attack/systemic embolism, prior bleeding, venous thromboembolism, hypertension, hypercholesterolemia, diabetes, cirrhosis, moderate-to-severe chronic kidney disease, dementia, hyperthyroidism, and hypothyroidism), and baseline antiplatelet use.

Treatment was defined as the first treatment received at the time of enrolment, approximating ‘intention to treat’. Patients with missing values were not removed from the study; multiple imputation combining estimates from five imputed datasets was applied for the comparative effectiveness analysis.

Data analysis was performed centrally by study statisticians using SAS (version 9.4).

RESULTS

Participants

In total, 3574 patients were prospectively enrolled to the UK study, comprising 6.9% (n = 3574/52 057) of the global cohort. Of these, 2.5% (n = 89/3574) were lost to follow-up and had incomplete 2-year data. At baseline, the mean age was 74.5 (SD 9.5) years (data not shown), 42.6% (n = 1522/3574) of participants were female (Table 1), and 98.8% (n = 3441/3483) were White.

The median CHA2DS2-VASc and HAS-BLED scores were 3.0 (quartile 1 [Q1] 2.0; quartile 3 [Q3] 4.0) and 2.0 (Q1 1.0; Q3 2.0), respectively (Table 1). In total, 89.3% (n = 3150/3528) had a CHA2DS2-VASc score ≥ 2 and 18.9% (n = 479/2530) had a HAS-BLED score ≥ 3.

Antithrombotic treatment

Of the participants, 65.8% (n = 2344/3564) received anticoagulant therapy at diagnosis; of these 70.6% (n = 1656/2344) received VKA and 29.4% (n = 688/2344) received a DOAC. In total, 12.5% (n = 447/3564) received anticoagulant therapy and an antiplatelet, 20.8% (n = 742/3564) received an antiplatelet only, and 13.4% (n = 478/3564) received neither anticoagulant nor antiplatelet therapy (data not shown).

Anticoagulant therapy was prescribed in 67.1% (n = 2108/3142) of patients with CHA2DS2-VASc score of ≥ 2, 56.1% (n = 161/287) of patients with CHA2DS2-VASc score of 1 (that is males with a score of 1) and 47.2% (n = 428/89) of patients with CHA2DS2-VASc score of 0 (that is females with a score of 1 and males with a score of 0). Of the participants, 51.6% (n = 247/479) with a HAS-BLED score ≥ 3 received anticoagulation (data not shown).

The proportion of patients receiving anticoagulant therapy increased progressively with CHA2DS2-VASc score (Figure 1a). The proportion of patients receiving anticoagulant therapy peaked in patients with a HAS-BLED score ≥ 3, then decreased with increasing

| Variable | Value | % |
|----------|-------|---|
| Age, years, median [Q1; Q3] | 75.0 [69.0; 81.0] | — |
| Age, years, n/N | | |
| <65 | 471/3574 | 13.2 |
| 65–74 | 1178/3574 | 33.0 |
| ≥75 | 1925/3574 | 53.9 |
| Sex, female, n/N | 1522/3574 | 42.6 |
| Ethnicity, White, n/N | 3441/3563 | 98.8 |
| Body mass index, median [Q1; Q3] | 28.1 [25.0; 32.3] | — |
| Medical history, n/N | | |
| Congestive heart failure | 274/3573 | 7.7 |
| Coronary artery disease | 678/3573 | 19.0 |
| Acute coronary syndromes | 363/3561 | 10.2 |
| Carotid occlusive disease | 52/3515 | 1.5 |
| Prior stroke/TIA/systemic embolism | 450/3551 | 12.7 |
| Vascular disease* | 760/3551 | 21.4 |
| History of bleeding | 109/3560 | 3.1 |
| Hypertension | 2483/3564 | 69.7 |
| Hypercholesterolemia | 1318/3492 | 37.7 |
| Diabetes mellitus | 629/3573 | 17.6 |
| Chronic kidney disease (grade ≥3) | 89/3499 | 25.6 |
| Cirrhosis | 11/3573 | 0.3 |
| CHA2DS2-VASc, median [Q1; Q3] | 3.0 [2.0; 4.0] | — |
| CHA2DS2-VASc score categories, n/N | | |
| 0 | 89/3528 | 2.5 |
| 1 | 289/3528 | 8.2 |
| 2 | 659/3528 | 18.7 |
| 3 | 972/3528 | 27.4 |
| 4 | 848/3528 | 24.0 |
| 5 | 398/3528 | 11.3 |
| ≥6 | 273/3528 | 7.7 |
| HAS-BLED score, median [Q1; Q3] | 2.0 [1.0; 2.0] | — |
| HAS-BLED score categories, n/N | | |
| 0 | 160/2530 | 6.3 |
| 1 | 941/2530 | 37.2 |
| 2 | 950/2530 | 37.5 |
| 3 | 391/2530 | 15.5 |
| 4 | 88/2530 | 3.5 |
| Care setting at diagnosis, n/N | | |
| Cardiology | 544/3573 | 15.2 |
| Geriatrics | 63/3573 | 1.8 |
| Internal medicine | 779/3573 | 21.8 |
| Neurology | 42/3573 | 0.1 |
| Primary care/general practice | 2183/3573 | 61.1 |

*Defined as peripheral artery disease and/or coronary artery disease. The risk factor ‘Labile INRs’ is not included in the HAS-BLED score as these data are not collected at baseline. As a result, the maximum HAS-BLED score at baseline is eight points (not nine). INR = international normalisation ratio. Q1 = quartile 1. Q3 = quartile 3. TIA = transient ischaemic attack.
The proportion of patients receiving antiplatelet treatment only according to their CHA2DS2-VASc score ranged from 16.9% to 22.5% (Figure 1a). The proportion of patients receiving antiplatelet treatment only increased progressively with their HAS-BLED score from 0 in patients with a HAS-BLED score of 0 to 47.7% in patients with a HAS-BLED score from 4 to 6 (Figure 1b).

Most baseline characteristics were similar in patients who received anticoagulant therapy and patients who did not receive anticoagulant therapy; however, a higher proportion of patients receiving anticoagulation had hypertension, diabetes, prior stroke, and venous thromboembolism, whereas a higher proportion of patients with heavy alcohol consumption and history of bleeding did not receive anticoagulants (Table 2). This is of course due to hypertension, diabetes, and prior stroke being risk factors for AF-related stroke. Heavy alcohol consumption and history of bleeding are risk factors for bleeding.

**Clinical outcomes**

At 2-year follow-up, the incidence rates of all-cause mortality, non-haemorrhagic stroke/systemic embolism, and major bleeding were 4.15 (95% CI = 3.69 to 4.65), 1.45 (95% CI = 1.19 to 1.77), and 1.21 (95% CI = 0.97 to 1.50) per 100 person-years, respectively (data not shown).

The rates of all-cause mortality, non-haemorrhagic stroke/systemic embolism, and major bleeding increased with...
Table 2. Baseline characteristics by anticoagulant treatment versus no anticoagulant treatment

| Baseline characteristics                        | No OAC (N = 1219) | OAC (N = 2342) | P-value* |
|------------------------------------------------|-------------------|----------------|----------|
| **Sex, n(%)**                                   |                   |                |          |
| Male                                           | 719 (59.0)        | 1324 (56.5)    | 0.161    |
| Female                                         | 500 (41.0)        | 1018 (43.5)    |          |
| **Age, years, median (Q1; Q3)**                | 75.0 (69.0; 82.0) | 75.0 (69.0; 81.0) | 0.353   |
| **Ethnicity, n(%)**                            |                   |                |          |
| White                                          | 1172              | 2298           |          |
| Hispanic/Latino                                | 1160 (99.0)       | 2268 (98.7)    | 0.659    |
| Asian                                          | 2 (0.2)           | 8 (0.3)        |          |
| African Caribbean/mixed/other                  | 7 (0.6)           | 12 (0.5)       |          |
| **Body mass index, kg/m², median (Q1; Q3)**     | 27.5 (24.6; 31.3) | 28.4 (25.1; 32.7) | 0.002   |
| **Systolic blood pressure, mmHg, median (Q1; Q3)** | 134.0 (121.0; 143.0) | 132.0 (120.0; 140.0) | 0.021   |
| **Diastolic blood pressure, mmHg, median (Q1; Q3)** | 79.0 (70.0; 84.0) | 78.0 (70.0; 83.0) | 0.127   |
| **Pulse, BPM, median (Q1; Q3)**                | 82.0 (70.0; 102.0) | 80.0 (70.0; 100.0) | 0.033   |
| **Type of atrial fibrillation, n(%)**           |                   |                |          |
| Permanent                                      | 379 (31.1)        | 852 (36.4)     | <0.001   |
| Persistent                                     | 65 (5.3)          | 209 (8.9)      |          |
| Paroxysmal                                     | 257 (21.1)        | 392 (16.7)     |          |
| New onset (unclassified)                       | 517 (42.4)        | 889 (38.0)     |          |
| **Medical history, n(%)**                      |                   |                |          |
| Heart failure                                  | 70 (5.7)          | 201 (8.6)      | 0.003    |
| Acute coronary syndromes                       | 114 (9.4)         | 249 (10.7)     | 0.224    |
| Vascular disease                               | 251 (20.7)        | 504 (21.7)     | 0.521    |
| Carotid occlusive disease                      | 14 (1.2)          | 37 (1.6)       | 0.294    |
| Venous thromboembolism                         | 40 (3.3)          | 124 (5.3)      | 0.007    |
| Prior stroke/TIA/systemic embolism             | 122 (10.1)        | 328 (14.1)     | <0.001   |
| Prior bleeding                                 | 69 (5.7)          | 40 (1.7)       | <0.001   |
| Hypertension                                   | 810 (66.6)        | 1664 (71.3)    | 0.004    |
| Hypercholesterolaemia                          | 429 (36.1)        | 883 (38.5)     | 0.172    |
| Diabetes                                       | 161 (13.2)        | 464 (19.8)     | <0.001   |
| Cirrhosis                                      | 6 (0.5)           | 5 (0.2)        | 0.155    |
| Moderate-to-severe CKD                         | 308 (25.9)        | 585 (25.9)     | 0.774    |
| Dementia                                       | 8 (0.7)           | 20 (0.9)       | 0.529    |
| Heavy alcohol consumption, n(%)                | 68 (5.6)          | 58 (2.8)       | <0.001   |
| Current smoker, n(%)                           | 87 (7.3)          | 155 (6.7)      | 0.554    |
| Antiplatelet treatment, n(%)                   | 742 (60.9)        | 447 (19.1)     | <0.001   |
| CHA2DS2-VASc score, median (Q1; Q3)            | 3.0 (2.0; 4.0)    | 3.0 (2.0; 4.0) | <0.001   |
| HAS-BLED score, median (Q1; Q3)                | 2.0 (1.0; 3.0)    | 2.0 (1.0; 2.0) | <0.001   |

*P-values calculated using t-test or Wilcoxon-Mann-Whitney test for categorical variables and χ² test or Fisher exact test for categorical variables, as appropriate. Some patients have unavailable baseline characteristics information. Percentages are calculated among those with available information.

Effectiveness of anticoagulant use

After adjustment for demographic and lifestyle factors [see Supplementary Figure S1], clinical measures at diagnosis, and medical history, anticoagulant use was associated with significantly lower all-cause mortality (adjusted hazard ratio [aHR] 0.70, 95% CI = 0.53 to 0.93, $P = 0.013$), non-haemorrhagic stroke/systemic embolism (aHR 0.39, 95% CI = 0.24 to 0.62, $P = 0.001$), and major bleeding in patients who received antithrombotic only were 4.35 (95% CI = 3.37 to 5.60), 2.61 (95% CI = 1.87 to 3.63), and 1.17 (95% CI = 0.72 to 1.91), respectively [Table 3].

Treatment changes are described in Supplementary Box S2.

DISCUSSION

Summary

In this recent cohort of UK patients newly diagnosed with AF, death was the most frequent clinical outcome at 2 years occurring at 2.9 times the rate of non-haemorrhagic stroke/systemic embolism and 3.4 times the rate of major bleeding. Death remained the most frequent outcome regardless of whether patients were receiving anticoagulation or not. Anticoagulation treatment compared with no anticoagulation treatment was associated with significantly lower all-cause mortality, significantly lower risk of non-haemorrhagic stroke/systemic embolism, and a non-significant higher risk of major bleeding.
Figure 2. a) Event rates according to CHA₂DS₂-VASc score. Includes only patients with available CHA₂DS₂-VASc scores (n = 3528). b) Event rates according to HAS-BLED scores. Includes only patients with available HAS-BLED scores (n = 2530). SE = systemic embolism.

Table 3. Two-year event rates per 100 person–years in the GARFIELD-AF UK population by treatment at baseline

| Treatment at baseline | All-cause mortality | Non-haemorrhagic stroke/systemic embolism | Major bleeding |
|-----------------------|---------------------|------------------------------------------|---------------|
|                       | Events | Rate (95% CI) | Events | Rate (95% CI) | Events | Rate (95% CI) |
| OAC                   | 172    | 3.89 [3.35 to 4.52] | 46     | 1.05 [0.79 to 1.40] | 55     | 1.26 [0.97 to 1.64] |
| No OAC                | 106    | 4.68 [3.87 to 5.66] | 49     | 2.21 [1.67 to 2.92] | 25     | 1.11 [0.75 to 1.65] |
| DOAC                  | 52     | 3.98 [3.03 to 5.23] | 13     | 1.00 [0.58 to 1.73] | 10     | 0.77 [0.41 to 1.43] |
| VKA                   | 120    | 3.85 [3.22 to 4.61] | 33     | 1.07 [0.76 to 1.51] | 45     | 1.46 [1.09 to 1.96] |
| OAC + antiplatelet    | 37     | 4.43 [3.21 to 6.11] | 6      | 0.72 [0.32 to 1.61] | 15     | 1.82 [1.10 to 3.02] |
| OAC only              | 135    | 3.77 [3.18 to 4.46] | 40     | 1.13 [0.83 to 1.54] | 40     | 1.13 [0.83 to 1.54] |
| Antiplatelet only     | 60     | 4.35 [3.37 to 5.60] | 35     | 2.61 [1.87 to 3.63] | 16     | 1.17 [0.72 to 1.91] |
| No OAC nor antiplatelet| 46     | 5.19 [3.89 to 6.93] | 14     | 1.60 [0.95 to 2.70] | 9      | 1.02 [0.53 to 1.97] |

DOAC = direct-acting oral anticoagulant. GARFIELD-AF = Global Anticoagulant in the FIELD — Atrial Fibrillation registry. OAC = oral anticoagulant. VKA = vitamin K antagonist.
Figure 3. Unadjusted and adjusted hazard ratios of OAC versus no OAC (reference) and corresponding 95% confidence intervals for selected outcomes at 2 years of follow-up in UK patients. Adjusted hazard ratios were obtained using an overlap-weighted Cox model. Variables included in the weighting scheme are: cohort enrolment, sex, age, ethnicity, type of AF, care setting, specialty and location, congestive heart failure, acute coronary syndromes, vascular disease, carotid occlusive disease, prior stroke/transient ischaemic attack/SE, prior bleeding, venous thromboembolism, hypertension, hypercholesterolaemia, diabetes, cirrhosis, moderate-to-severe chronic kidney disease, dementia, hyperthyroidism, hypothyroidism, current smoking, heavy alcohol consumption, body mass index, heart rate, systolic and diastolic blood pressure at diagnosis, and baseline antiplatelet use. AF = atrial fibrillation. OAC = oral anticoagulant. SE = systemic embolism.

Strengths and limitations
GARFIELD-AF was conducted to high-quality standards and data for 20% of the UK cohort were monitored against source documentation. Enrolling patients within 6 weeks of diagnosis ensured the sample included patients who may not survive long after an AF diagnosis by capturing disease burden early on.

The main limitation of the study is that the analysis is intention to treat, based on therapy initiated at diagnosis, and does not account for treatment changes during the 2-year follow-up. Also, the study did not collect data on deprivation and therefore it was not possible to adjust for deprivation in the analysis. Despite having applied appropriate propensity score methodology to balance confounding factors across treatment groups, the authors cannot exclude the presence of unobserved confounding.

Comparison with existing literature
The findings of the present study regarding the benefit of anticoagulation fits with previous meta-analyses of randomised controlled trials in the VKA-only era as well as the DOAC trials.14,17,20 The estimated lower risk of 30% and 61% for all-cause mortality and non-haemorrhagic stroke/systemic embolism in UK patients on anticoagulants in this study is similar to the results from these studies. The estimated 31% higher risk of major bleeding did not reach statistical significance, which might be because of the relatively small number of these events in this cohort of patients.

As in the UK cohort, death was the most frequent outcome in the global cohort, occurring at over three times the rate of non-haemorrhagic stroke/systemic embolism and almost five times the rate of major bleeding. Nevertheless, the 2-year event rates per 100 person-years were numerically higher in the UK cohort compared with the global cohort excluding the UK: all-cause mortality 4.15 (95% CI = 3.69 to 4.65) versus 3.80 (95% CI = 3.68 to 3.93), non-haemorrhagic stroke/systemic embolism 1.45 (95% CI = 1.19 to 1.77) versus 0.97 (95% CI = 0.91 to 1.04), and major bleeding 1.21 (95% CI = 0.97 to 1.50) versus 0.96 (95% CI = 0.90 to 1.03). Overall, a similar proportion received anticoagulation at baseline (65.8% versus 66.9%); the median CHA2DS2-VASc score was similar for the UK and global cohort (median CHA2DS2-VASc 3.0 [Q1 2.0; Q3 4.0] versus 3.0 [Q1 2.0; Q3 4.0]) but the median HAS-BLED scores were higher in the UK cohort (median HAS-BLED 2.0 [Q1 1.0, Q3 2.0] versus 1.0 [Q1 1.0 to Q3 2.0]).

The reduction in the risk of all-cause mortality and non-haemorrhagic stroke/systemic embolism was more marked in the UK cohort compared with the global cohort (30% versus 18% and 61% versus 29%, respectively), and the increment in the risk of bleeding was not statistically significant in the UK but statistically significant in the global cohort (aHR 1.31 [95% CI = 0.77 to 2.24] versus 1.46 [95% CI = 1.1 to 1.86], respectively).22 Overall though, the findings on the relative effects of anticoagulation in the global study are reproduced in this UK-only cohort. Marginal differences might be because of the wider uncertainty around the obtained estimates in the UK data. In addition, the global analysis on effectiveness of anticoagulants was based on a different group of patients comprising patients enrolled to cohort 3 to 5 and patients with a CHA2DS2-VASc score of ≥2.

Implications for practice
Findings regarding the benefit of anticoagulation for reduction of all-cause mortality and non-haemorrhagic stroke/systemic embolism without a significant increase in the risk of bleeding suggests that for most patients the benefits of anticoagulation outweigh the risks, and affirms recommendations relating to anticoagulation in AF management guidelines.

The findings regarding a non-significant increase in the risk of bleeding are reassuring, particularly as 47.0% of patients with a HAS-BLED score of 4–6 received anticoagulation. Nevertheless, this finding should be interpreted with caution, and it must be emphasised that despite the benefit, anticoagulation is recommended for patients at increased risk of stroke with a CHA2DS2-
VASc score of ≥2 and should be considered for those patients with a CHA2DS2-VASc score of 1.

There is scope for improvement in the management of patients newly diagnosed with AF to align with NICE guidelines. Overall, 67.1% of participants at high risk of stroke (CHA2DS2-VASc score ≥2) received anticoagulation at diagnosis. The authors have previously reported a progressive increase in the proportion of patients in the UK cohort receiving anticoagulation, with 75.6% of the final cohort of patients (diagnosed June 2015 to July 2016) receiving anticoagulation. Further increment in the proportion of patients at high risk of stroke receiving anticoagulation will optimise anticoagulation in patients with AF and improve outcomes. On the other hand, 47.2% of patients defined as very low risk in the NICE guidelines (CHA2DS2-VASc score of 0 for males or 1 for females) received anticoagulation, which is contrary to the guidelines.

In addition, the practice of prescribing antiplatelet treatment alone (20.8%) is contrary to AF guidelines; the guidelines indicate patients at risk of stroke must receive anticoagulants or no antithrombotic therapy. The practice of prescribing anticoagulant with antiplatelet treatment (12.5%) is also not recommended in the guidelines.

The older profile of the patient population and the prevalence of comorbidities are likely to be contributory factors to the all-cause mortality. The prominence of all-cause mortality as an outcome indicates more attention should be given to mortality risk in the management of patients newly diagnosed with AF.
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