Arriba: effects of an educational intervention on prescribing behaviour in prevention of CVD in general practice

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Abstract

Background: Evidence on the effectiveness of educational interventions on prescribing behaviour modification in prevention of cardiovascular disease is still insufficient. We evaluated the effects of a brief educational intervention on prescription of hydroxymethylglutaryl-CoA reductase inhibitors (statins), inhibitors of platelet aggregation (IPA), and antihypertensive agents (AH).

Design: Cluster randomised controlled trial with continuous medical education (CME) groups of general practitioners (GPs).

Methods: Prescription of statins, IPA, and AH were verified prior to study start (BL), immediately after index consultation (IC), and at follow-up after 6 months (FU). Prescription in patients at high risk ($> 15\%$ risk of a cardiovascular event in 10 years, based on the Framingham equation) and no prescription in low-risk patients ($\leq 15\%$) were considered appropriate.

Results: An intervention effect on prescribing could only be found for IPA. Generally, changes in prescription over time were all directed towards higher prescription rates and persisted to FU, independent of risk status and group allocation.

Conclusions: The active implementation of a brief evidence-based educational intervention on global risk in CVD did not lead directly to risk-adjusted changes in prescription. Investigations on an extended time scale would capture whether decision support of this kind would improve prescribing risk-adjusted sustainably.

Keywords

Antihypertensive agents, aspirin, cardiovascular diseases, drug therapy, hydroxymethylglutaryl-CoA reductase inhibitors, primary health care

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Introduction

Guidelines suggest that prevention of cardiovascular disease (CVD) should start with global risk assessment and proceed to suitable treatment of correctable risk factors.^{1,2} Current guidelines recommend aspirin and lipid-lowering therapy for high-risk patients and evidence supports a similar approach for hypertension therapy.^{1–3} Treatment decisions based on single risk factors may more easily result in either over- or under-treatment.³ Various multivariate risk functions form the basis of predictive tools and risk scores.^{4,5} Evidence on the effectiveness of such tools on prescribing behaviour modification in prevention of CVD is still insufficient.⁶ General practitioners (GPs) are also

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expected to involve their patients in decision-making.^{7,8} Up to date, neither individual CVD risk calculation,^{9–11} nor engagement of patients in their own healthcare decisions¹² can be considered as being standard care. However, it still remains unclear which approach is best to implement evidence-based care into daily practice.^{13,14}

We developed Arriba, a simple evidence-based decision aid that combines risk calculation and consultation based on a patient's individual global risk for CVD. For the whole decision aid and materials, please refer to the supplemental appendix of Krones et al.¹⁵

In this article we present our results evaluating the impact on prescription. Changes in prescribing behaviour and appropriateness of prescription were analysed as secondary endpoints.

Methods

Study population

Hessian continuing medical education (CME) groups, coordinated by AQUA, a large German quality management institute, were screened for eligibility. CME groups were excluded if members routinely used cardiovascular risk calculators. Randomization to intervention or control group was done on CME-group level by the Centre for Clinical Trials, University of Marburg. GPs had to recruit up to 15 adult patients in who for any reason cholesterol levels were measured. To ensure that similar patients were included in both study arms, GPs were asked to approach all patients consecutively. Rolling recruitment of patients was spread evenly from May 2005 to March 2006.

The study complies with the Declaration of Helsinki. The research protocol was approved by the local research ethics committee, University of Marburg. Informed consent was obtained from all participating GPs and patients.

Intervention

Intervention GPs had to attend two CME sessions lasting 2.5 hours each. We discussed the epidemiological background of global cardiovascular risk calculation as well as ethics of shared decision-making by putting the emphasis on practical communication strategies and materials to be applied during consultation. Control GPs were invited to seminars on alternative topics that would not interfere with CVD prevention.

Measures

Arriba follows a baseline risk strategy by recommending a discussion with the patient about pharmacological

interventions of hydroxymethylglutaryl-CoA reductase inhibitors (statins), inhibitors of platelet aggregation (IPA), or antihypertensive agents (AH) for anyone who has a risk of 15% or greater of developing CVD within the next 10 years, regardless of single risk factors. We investigated prescription of statins, IPA, and AH as well as any of these (total medication: statins or IPA or AH) as binary variables (yes or no). We compared the pre-study situation (BL), prescription at index consultation (IC), reflecting immediate effects of the intervention, and medication at 6-month follow-up (FU) reflecting long-term effects. Absolute cardiovascular risk was assessed according to the adapted Framingham equation, calibrated for European populations,¹⁶ based on the following risk factors [age, sex, total cholesterol, and high-density lipoprotein (HDL) cholesterol, systolic blood pressure with and without medication, smoking status] and was adjusted for diabetes. Baseline characteristics of GPs and patient data were obtained from study questionnaires. Prescription was considered appropriate in patients with high cardiovascular risk (>15%) and no prescription in patients with low cardiovascular risk (≤15%).

Statistical analysis

For binary/continuous outcome variables, we analysed differences between study groups by using mixed-effects logistic regression (procedure *xtmelogit*) in STATA (version SE 10.0) to allow for clustering by GP and CME-group (GP nested within CME group) with study arm as fixed effect. We added baseline characteristics at GP level (practice size as attendees per 3 months, years in practice, urban/rural, sex of GP) and at patient level (cardiovascular risk, diabetes, CVD (yes/no), subjective risk, age, sex, education) as covariates to the models. We further analysed prescription at IC and FU (total medication, IPA, statins, and AH, respectively) as dependent variables and added study arm and premedication at BL as fixed effects. Pearson chi-squared test was used for the univariate comparison between study groups. Appropriateness of prescription was evaluated using binary variables (yes/no) for each single medication. Results of two-sided tests between study arms were regarded significant at $p < 0.05$. McNemar test was used to test for significant changes in prescribed medication over time within study groups, adjusted for multiple testing by Bonferroni correction ($\alpha = 0.05/24 = 0.002$). GPs failed to measure HDL cholesterol in 108 patients. We imputed missing values for HDL cholesterol, and single missing values for age, total cholesterol, and systolic blood pressure by using a maximum likelihood based algorithm (EM algorithm).

Figure 1. Flow chart: continuous medical education (CME) groups, general practitioners (GPs), and patients.

For analyses at BL and IC we included all patients with valid data at IC. Estimates of prescribing at FU refer only to patients attending after 6 months.

Sample size was calculated using the original primary outcome measure, patient participation and satisfaction by the Patient Participation Scale.¹⁷ Results on main outcome measures have been published recently, including details on power calculations, recruitment rates, and baseline characteristics.¹⁵

Due to the explorative nature of this study, we avoided an additional arbitrary sample size calculation

for this secondary outcome measure, aiming at the minimal clinically important difference as suggested by Man-Son-Hing et al.¹⁸

Results

Study population

Figure 1 gives an overview of the study population. Thirty CME groups consisting of 162 GPs were eligible and agreed to participate. Some small CME groups

Table 1. Baseline characteristics of participating general practitioners (GPs) by intervention and control group ($n = 47$)

Baseline variables	Intervention group ($n = 44$)	Control group ($n = 47$)	Chi-squared p -value
GP age (years)			0.57
≤ 50	26 (59.1)	25 (53.2)	
> 50	18 (40.9)	22 (46.8)	
GP male	27 (61.4)	26 (55.3)	0.56
Geographical location (% urban)	20 (45.5)	20 (42.6)	0.78
Attendees per 3-months (practice size)			0.003
≤ 1000	7 (15.9)	21 (44.7)	
> 1000	37 (84.1)	26 (55.3)	
Years in practice			0.14
0–10	9 (20.4)	18 (38.3)	
11–20	28 (63.7)	21 (34.7)	
21–30	7 (15.9)	8 (17.0)	

Values are n (%), unless otherwise indicated. Data on age, practice size, and years in practice were primarily collected in categories.

were rearranged to make up a size of 9–13 participants in each CME group, resulting in 14 CME groups with 162 GPs. Of those, 127 practitioners attended the first of two educational sessions in either the intervention or the control arm. In the intervention arm, 44 GPs consecutively recruited 550 patients. In the control arm, 47 GPs similarly included 582 patients. At follow-up, 460 intervention patients (83.6%) and 466 control patients (80.1%) were seen. A total of 206 patients were lost to follow-up, 82 of which because their GP was no longer participating.

Baseline characteristics of participating GPs are shown in Table 1. With respect to patient characteristics (Table 2), study arms were well balanced for socio-demographic characteristics, prevalence of individual risk factors, and clinical disease as well as global risk, subjective risk, and subjective health. The number of patients with diabetes and CVD was significantly higher among control patients, which therefore were considered as covariates in the multivariate models. However, effect sizes according to Cohen¹⁹ and Grissom and Kim²⁰ were low.

Prescribing patterns and appropriateness of prescription

Changes in prescription over time are displayed in Table 3. At BL, patients were treated similarly in both study groups, except for statins, where significantly more high-risk patients of the control group were under treatment ($p < 0.001$). Generally, in relation to BL, changes in prescription over time were all directed towards higher prescription rates and persisted to FU, independent of risk status and group allocation. This resulted in an increased appropriateness of medical treatment in high-risk patients and in a decreased

appropriateness in the low-risk patients in both groups. However, there still was an oversupply in the low-risk patients, whereas patients at high risk remained under-supplied. Compared to the control group, prescribing rates of IPA increased significantly ($p < 0.001$) in the intervention arm in both risk groups.

Significant predictors for changes in prescription over time (from BL to FU) could only be found for IPA and are listed in Table 4. In summary, at FU significantly more IPA was prescribed in intervention patients and in patients with lower education. Male GPs increased prescription of IPA up to FU significantly more often than female GPs. Prescription at FU was also highly influenced by premedication at BL. In contrast, no significant predictors were observed for statins and AH.

In summary, it can be stated that we were able to show an intervention effect on prescribing for IPA only.

Discussion

Although we were able to show a slight intervention effect on prescribing for IPA, overall prescribing behaviour did not change when it was risk adjusted.

Our findings demonstrate a gap between recommended and current drug use for primary prevention of CVD, which is reflected by undersupply in the high-risk and the oversupply in the low-risk patients at follow-up.

The importance of a medication is context dependent and influenced by characteristics of the physician and/or patient. In our study, certain complaints of a patient might have been so salient for the physician that his or her attention had been directed towards a specific configuration of medication.

Table 2. Baseline characteristics of patients by intervention and control group

Variable	Intervention group (n = 550)	Control group (n = 582)	Chi-squared <i>p</i> -value
Age (years, mean $\pm$ SD)	59.1 $\pm$ 12.3	58.6 $\pm$ 12.5	0.49 (t-test)
Sex (male)	231 (42.0)	265 (45.5)	0.26
Nationality (German)	528 (96.0)	566 (97.3)	0.46
Education			0.71
No or basic education	284 (51.6)	320 (54.9)	
Medium education	169 (30.7)	168 (28.9)	
Higher education	91 (16.6)	91 (15.6)	
Positive family history of CVD	51 (9.3)	45 (7.8)	0.362
Smokers	102 (18.5)	108 (18.6)	1.0
Diabetes	74 (13.5)	160 (27.5)	<0.001 ^c
Known cardiovascular disease (CVD)	26 (4.7)	61 (10.5)	<0.001 ^c
Subjective risk status			0.021
Low–very low	357 (64.9)	327 (56.2)	
High–very high	95 (17.3)	108 (18.5)	
Don't know	98 (17.8)	147 (25.3)	
Subjective health status			0.27
Good–very good	266 (48.9)	260 (45.0)	
Satisfactory	242 (44.5)	278 (48.1)	
Bad–very bad	36 (6.7)	40 (6.9)	
Cardiovascular risk status ^a			
$\leq 15\%$	289 (54.9)	308 (54.4)	0.86
$> 15\%$	237 (45.1)	258 (45.6)	
Manifest atherosclerosis ^b	110 (21.0)	139 (28.0)	0.12
Risk factors (mean $\pm$ SD)			t-test <i>p</i> -value
Triglycerides (mmol/l)	1.8 $\pm$ 1.2	1.8 $\pm$ 1.1	0.74
Total cholesterol (mmol/l)	6.0 $\pm$ 1.2	5.7 $\pm$ 1.1	<0.001, <i>d</i> = 0.26 ^d
HDL-cholesterol (mmol/l) Syst.	1.6 $\pm$ 0.4	1.5 $\pm$ 0.5	0.15
Blood pressure (mmHG)	132 $\pm$ 17.3	132 $\pm$ 17.4	0.92
HbA _{1c} (%)	6.2 $\pm$ 1.1	6.7 $\pm$ 1.2	<0.001, <i>d</i> = 0.44 ^d
Blood glucose (mmol/l)	5.3 $\pm$ 1.7	5.7 $\pm$ 1.9	<0.001, <i>d</i> = 0.22 ^d

Values are *n* (%), unless otherwise indicated. ^aCardiovascular risk calculation based on Framingham equation (see methods section). ^bManifest atherosclerosis defined according to the International Statistical Classification of Diseases and Related Health Problems (ICD) edited by the WHO (ICD-10, revised version 2006). ^cEffect size calculated from contingency table: diabetes: Cohen's *w* = −0.173 (95% CI −0.23 to −0.12); CVD: Cohen's *w* = −0.11 (95% CI −0.16 to −0.05). ^dEffect size calculated as the difference between two means divided by a SD as Cohen's *d*.¹⁹

Among others, a high-risk patient not on medication might have had side effects in the past, had discontinued medication on his own, had refused to take medication, or preferred to try behavioural changes first, after having discussed the therapeutic options for evidence-based risk reduction, given the potential mixed effects of our decision aid, even if professionals had changed their behaviour. On the other side, a low-risk patient's risk might have been lowered because of treatment and thus prescribed medication might have been appropriate. Apart from that, as patient age increases, age might have been taken into account when reconsidering prescription.

Our findings correspond to studies, which covered treatment recommendations that are inconsistent with contemporary guidelines.^{21,22} Persell et al.²³ reported that providing 10-year coronary risk information improved aspirin-prescribing decisions and lipid management when the short-term risk was moderately high. However, important treatment gaps remained and guidelines still were not followed for eligible patients. This is supported by Vlek et al.²⁴ who evaluated trends in vascular risk factors and medication use in cardiovascular diseases over a 12-year period. In contrast, Hall et al.²⁵ found an increase in prescription of preventive medications to

Table 3. Changes in prescribed medication over time, from prior to study start to index consultation and follow up

Medication	Intervention group		Control group	
	Patients under treatment (%)	McNemar (p-value)	Patients under treatment (%)	McNemar (p-value)
Low risk status ($\leq 15\%$)				
Total medication				
BL-IC	34.3–49.2	<0.001	36.5–47.8	<0.001
BL-FU	34.3–47.7	<0.001	36.5–44.9	<0.001
Statins (hydroxymethylglutaryl-CoA reductase inhibitors)				
BL-IC	5.5–8.7	0.022	8.8–11.7	0.049
BL-FU	5.5–7.5	0.754	8.8–11.9	0.115
Inhibitors of platelet aggregation				
BL-IC	7.3–14.9	<0.001	6.5–6.8	1.0
BL-FU	7.3–14.2	<0.001	6.5–7.8	0.250
Antihypertensive agents				
BL-IC	29.8–34.8	<0.001	32.5–36.2	0.001
BL-FU	29.8–38.7	<0.001	32.5–38.3	0.001
High risk status ($> 15\%$)				
Total medication				
BL-IC	73.1–86.9	<0.001	76.2–84.9	0.022
BL-FU	73.1–85.2	<0.001	76.2–83.7	0.022
Statins (hydroxymethylglutaryl-CoA reductase inhibitors)				
BL-IC	26.1–30.5	0.043	36.7–39.8	0.047
BL-FU	26.1–29.1	0.115	36.7–43.4	0.031
Inhibitors of platelet aggregation				
BL-IC	25.1–46.4	<0.001	27.3–27.7	1.0
BL-FU	25.1–46.2	<0.001	27.3–29.3	1.0
Antihypertensive agents				
BL-IC	63.4–69.1	<0.001	68.8–75.7	<0.001
BL-FU	63.4–73.3	0.001	68.8–79.2	<0.001

p-values adjusted for multiple testing by Bonferroni correction ($\alpha = 0.05/24 = 0.002$), $p < 0.002$. BL, prior to study start; IC, index consultation; FU, follow up.

Table 4. Significant predictors for changes in prescription of inhibitors of platelet aggregation over time: mixed effects logistic regression

Variable	z-values	p-value	Odds ratio (95% CI)
Study arm	−4.69	<0.001	0.056 (0.02–0.19)
Premedication of IPA at BL	9.12	<0.001	4685.52 (762.36–28797.39)
Patient education	−2.35	0.019	0.56 (0.34–0.91)
GP sex	−2.02	0.044	0.49 (0.25–0.98)

BL, prior to study start; CI, confidence interval; GP, general practitioner; IPA, inhibitors of platelet aggregation.

high-risk individuals without a change in the low-risk group. Similarly, Grover et al.²⁶ indicated that discussing coronary risk with the patient was associated with an improvement in the efficacy of lipid therapy.

One contributing factor for inadequate prescription in our study could have been that a patient's risk of CVD was not accurately estimated. This corresponds to van Dis et al.²⁷ who stated that overestimation of CVD mortality risk may result in pharmacological

overtreatment. Sheridan and Crespo³ concluded that physicians' knowledge of global CHD risk scores may translate into modestly increased prescribing of cardiovascular drugs.

Most common barriers to guideline implementation, as reported by Graham et al.,²⁸ are patient compliance, a lack in time, and government health policy. In contrast, financial opportunities and simple guidelines are suggested to improve the implementation.

Implementation requires clear and deliverable evidence-based messages, evidence that current care is sub-optimal, a robust estimate of the cost, impact of alternative methods of behavioural change, and an understanding of the local organization of health care.^{13,29}

Strengths and limitations

Arriba was not conducted as pharmacological intervention study. Therefore, results have to be interpreted with caution. Although we did adjust for multiple testing, our analysis here is based on secondary outcome criteria.

Methodological aspects such as cross-quarterly supply or prescription by specialists could not have been recorded here. That is why 'appropriateness of prescription' as defined in our study should be seen as a measure to compare study arms, but well known that it does not necessarily reflect widespread reality of prescribing behaviour.

GPs in the control arm recruited more diabetic and more high-risk patients, which may reflect a selection bias at patient level.

Furthermore, recruitment of CME groups might implicate a selection bias at physician level, which might result in an overestimation of intervention effects.

Based on our exclusion criterion, not having used any risk calculator ahead of the study, our sample might just as well reflect late adopters, which may then lead to an underestimation of intervention effects.

Since control GPs were treated similarly compared to the intervention GPs and given other constraints regarding prescription (GPs are held accountable for their prescribing costs individually), we regard a Hawthorne effect in the control arm as unlikely.

Given the pragmatic study design, concealment of patient allocation and blinding of GPs were not possible.

Conclusions

Crucial research findings often do not translate automatically into practice. Our results suggest that a brief active implementation of an evidence-based educational intervention does not lead immediately to changes in

prescription. This may be the case because physicians are embedded in an organizational framework which prevents them from changing prescription rapidly. Only investigations on an extended time scale would capture, whether decision support of this kind would improve prescribing risk-adjusted sustainably.

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Clinical trial registration

This trial is registered at www.controlled-trials.com (number ISRCT171348772).

Conflict of interest

None.

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