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CLINICAL INVESTIGATIONS

Treatment in a preventive cardiology clinic utilizing advanced practice providers effectively closes atherosclerotic cardiovascular disease risk-management gaps among a primary-prevention population compared with a propensity-matched primary-care cohort: A team-based care model and its impact on lipid and blood pressure management

Emilio Fentanes[1] | Anthony G. Vande Hei[1] | R. Scott Holuby[1] | Norma Suarez[1] | Yousif Slim[1] | Jennifer N. Slim[2] | Ahmad M. Slim[1] | Dustin Thomas[1]

[1]Cardiology Department, San Antonio Military Medical Center, San Antonio, Texas

[2]Department of Medicine, San Antonio Military Medical Center, San Antonio, Texas

Correspondence
Dustin Thomas, MD, FACC, San Antonio Military Medical Center, 3551 Roger Brooke Drive, San Antonio, TX 78219
Email: dustin.m.thomas4.mil@mail.mil

Background: Advanced practice providers (APPs) can fill care gaps created by physician shortages and improve adherence/compliance with preventive ASCVD interventions.

Hypothesis: APPs utilizing guideline-based algorithms will more frequently escalate ASCVD risk factor therapies.

Methods: We retrospectively reviewed data on 595 patients enrolled in a preventive cardiology clinic (PCC) utilizing APPs compared with a propensity-matched cohort (PMC) of 595 patients enrolled in primary-care clinics alone. PCC patients were risk-stratified using Framingham Risk Score (FRS) and coronary artery calcium scoring (CACS).

Results: Baseline demographics were balanced between the groups. CACS was more commonly obtained in PCC patients ($P < 0.001$), resulting in reclassification of 30.6% patients to a higher risk category, including statin therapy in 26.6% of low-FRS PCC patients with CACS ≥75th MESA percentile. Aspirin initiation was higher for high and intermediate FRS patients in the PCC ($P < 0.001$). Post-intervention mean LDL-C, non–HDL-C, and triglycerides (all $P < 0.05$) were lower in the PCC group. Compliance with appropriate lipid treatment was higher in intermediate to high FRS patients ($P = 0.004$) in the PCC group. Aggressive LDL-C and non–HDL-C treatment goals (<70 mg/dL, $P = 0.005$ and < 130 mg/dL, $P < 0.001$, respectively), were more commonly achieved in high-FRS PCC patients. Median post-intervention SBP was lower among intermediate and low FRS patients ($P = 0.001$ and $P < 0.001$, respectively). Cumulatively, this resulted in a reduction in median post-intervention PCC FRS across all initial FRS risk categories ($P < 0.001$ for all).

Conclusions: APPs within a PCC effectively risk-stratify and aggressively manage ASCVD risk factors, resulting in a reduction in post-intervention FRS.

KEYWORDS

Atherosclerosis, Blood Pressure Control and Regulation, Computed Tomography, General Clinical Cardiology/Adult, Imaging, Preventive Cardiology

1 | INTRODUCTION

Atherosclerotic cardiovascular disease (ASCVD) remains the leading cause of mortality in the United States.[1,2] This high prevalence of disease, morbidity, and mortality continues to be observed despite significant advancements in ASCVD risk assessment and management. In the face of this high disease prevalence, data from the National Cardiovascular Data Registry Proactive Innovation and Clinical Excellence

(NCDR PINNACLE) observed that 43.9% of cholesterol treatment–eligible primary-prevention patients were receiving a statin medication and up to 35.9% were not receiving any lipid-lowering therapy.[3]

Downstream costs associated with the care of patients presenting with ASCVD events are tremendous. However, more robust implementation of primary-prevention therapies is complicated by the fact that the United States is in the midst of a shortage of primary-care physicians and cardiologists.[4] Advanced practice providers (APPs) may provide an opportunity to fill this vital gap in the healthcare delivery team to both expand access and relieve some burden from primary-care managers.[5–7] The appropriate utilization of APPs in a primary-prevention, subspecialty clinic population has the possibility to positively impact adherence to guideline-directed therapy, as it has been shown to do in secondary-prevention, diabetes mellitus (DM), and heart failure populations previously.[4,8,9]

We sought to analyze the effectiveness of risk stratification, initiation of recommended medical therapies, and resultant changes in global ASCVD risk by APPs with indirect oversight by a cardiologist utilizing locally developed treatment algorithms based on published guidelines.

2 | METHODS

2.1 | Study population

A population of 595 patients without known ASCVD referred to a preventive cardiology clinic (PCC) at a single-center military treatment facility from January 1, 2009, to December 31, 2013, was included in the study population. Baseline demographic data, initial and follow-up laboratory and imaging data, and cardiovascular risk factors were abstracted. An age and risk-factor propensity-matched cohort (PMC) was derived in a 1:1 fashion from an initial population of 20 604 patients enrolled in internal medicine and family medicine clinics in the same healthcare system over the same time period.

2.2 | The PCC

The PCC is embedded within the cardiology division and utilizes a clinical pharmacist, physician assistants, and a nurse practitioner supervised by a board-certified cardiologist. The PCC accepts primary-prevention adult patients from primary-care clinics and other specialty-care clinics within the local healthcare system. The APPs manage primary-prevention medications and pursue smoking cessation working with a guideline-based, locally developed algorithm utilizing Framingham Risk Score (FRS) and coronary artery calcium scoring (CACS). Treatment and follow-up testing decisions are made by APPs independently.

2.3 | Initial evaluation

Baseline evaluation obtained at the initial visit included a fasting lipid profile, fasting serum glucose, blood pressure (BP) measurements, and height/weight measurements. Cardiovascular risk factors as defined in PCC algorithms were as follows: smoking (active or prior >10 pack-years), hypertension (HTN; previous diagnosis, active treatment with an antihypertensive medication, or a systolic blood pressure [SBP] ≥140 mm Hg), DM (previous diagnosis, active treatment with an oral antihyperglycemic or insulin, glycated hemoglobin [HbA1c] level ≥ 6.5%, or fasting serum glucose ≥126 mg/dL), and hyperlipidemia (previous diagnosis or active treatment with lipid-lowering medication). Based on these risk-factor definitions and initial laboratory testing, APPs perform risk-factor counseling utilizing the National Cholesterol Education Program (NCEP) Adult Treatment Panel (ATP) III panel recommendations[10–12].

2.4 | Lipid-lowering therapy

Initiation, escalation, or discontinuation of lipid-lowering medication(s) was based on low-density lipoprotein cholesterol (LDL-C) targets as defined by the ATP III recommendations. All patients referred to the PCC were counseled on heart-health dietary interventions. Lipid therapy escalation was defined as an increase in statin therapy intensity, initiation of statin therapy in untreated patients, or addition of a secondary lipid-lowering medication. Lipid therapy de-escalation was defined as a decrease in statin intensity or statin dose, or discontinuation of statin therapy.

2.5 | CACS

The CACS studies were obtained using an electrocardiogram gated 128-slice dual-source computed tomography (CT) scanner (SOMATOM Definition Flash CT; Siemens, Erlangen, Germany). Foci of CAC were identified using semiautomatic commercial software (Vitrea 6.3 software; Vital Images, Minnetonka, MN). A total calcium score was derived using the Agatston scoring method, as was the Multi-Ethnic Study of Atherosclerosis (MESA) percentile.[13]

2.6 | Management of HTN

Patients were screened and treated for HTN in accordance with Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7) recommendations. Lifestyle-modification counseling was performed on every patient. Antihypertensive therapy was initiated at the discretion of the treating APP per a locally developed protocol based on JNC 7 treatment recommendations; the goal BP was <140/90 mm Hg in all patients, except for patients with DM or chronic kidney disease (goal BP <130/80 mm Hg in these patients).

2.7 | Management of DM

Patients with initial HbA1c levels >7.0% were considered for initiation of antihyperglycemic therapies. Recommended annual screening for microalbuminuria, peripheral neuropathy, and diabetic retinopathy was also performed. Patients achieving target HbA1c levels were monitored in 3- to 6-month intervals, whereas patients with persistently elevated HbA1c levels were referred to a specialized DM care clinic within the endocrinology division for evaluation of insulin or other more advanced therapies.

2.8 | Tobacco-cessation counseling

All patients referred to the PCC were screened for tobacco use. Patients willing to quit were referred to a weekly tobacco-cessation information class, as well as assessed for individualized intervention intensity level. Interventions ranged from a single counseling session without pharmacotherapy intervention to ≥6 counseling sessions, pharmacotherapeutic initiation, short-interval clinic follow-up, and referral to a weekly support group.

2.9 | Follow-up

Follow-up FRS in all patients was performed based on data obtained 12 months (±6 months) from the initial clinic encounter. Changes in BP and lipids were calculated as percent changes from baseline laboratory and BP data, with a positive percentage representing a favorable change and a negative percentage indicating a negative change. Initial and follow-up laboratory data within 6 months prior to the initial primary-care visit were labeled as baseline data, and follow-up laboratory data ≥3 months after the initial visit was abstracted and used to assess follow-up therapy and laboratory changes.

2.10 | Statistical analysis

Discrete variables were reported as proportions. Normally distributed continuous variables were reported as a mean ±SD, and non-normal continuous data were reported as median (interquartile range). Statistical significance was defined at the <0.05 level for all analyses (2-tailed). The PMC was derived utilizing Mahalanobis metrics matching. Between-group comparisons of continuous variables was obtained using 1- and 2-way ANOVA testing or Wilcoxon rank-sum tests, as appropriate. Categorical variables were compared using the Pearson χ^2 test. All data variables were analyzed using SPSS version 22 (IBM Corp., Armonk, NY).

3 | RESULTS

3.1 | Clinical characteristics

The baseline clinical characteristics for the PCC and PMC patient groups are shown in Table 1. The PCC group was more likely to be treated initially for HTN ($P < 0.001$). Otherwise, baseline demographics and cardiac risk factors were well balanced between the groups. The median FRS at initial evaluation was higher in the PCC cohort (15.9%) compared with the PMC patients (11.5%; $P < 0.001$). This was driven by more high-FRS patients ($P < 0.001$) and fewer low-FRS patients ($P < 0.001$) in the PCC cohort (Table 1).

3.2 | CACS

Testing for CACS was obtained in 82.9% of PCC patients, compared with only 10.9% of patients in the PMC cohort ($P < 0.001$). PCC patients had a mean CACS of 131.56 ±305.16 AU, with a CACS of 0 AU seen among 39.0%, and 12.4% having a CACS >300 AU. Among high-FRS patients, 38 (20.8%) had a CACS of 0 AU; among low-FRS patients, 62 (41.3%) had a detectable CACS, of which 20 (13.3%) had a CACS >100 AU. In the low- and intermediate-FRS groups, a total of 96 (30.6%) patients had a CACS that placed them into the 75th percentile for their age and sex, thus reclassifying them to a higher risk category (see Supporting Information, Figure 1, in the online version of this article).

3.3 | Utilization of aspirin

Overall, aspirin utilization among intermediate- and high-FRS patients was higher than national trends in both cohorts (65.2% in PCC vs 33.5% in PMC). Aspirin prescription post-intervention was higher for high- and intermediate-FRS patients in the PCC ($P < 0.001$; Table 1).

3.4 | Lipid management

Patients in the PCC had lower baseline LDL-C ($P = 0.036$) and were more commonly on a high-intensity statin ($P = 0.013$) and nonstatin lipid therapy ($P < 0.001$) when compared with the PMC (see Supporting Information, Figure 2, in the online version of this article). The remaining baseline lipid-panel values were not different between the groups (Table 1). Initiation of lipid-lowering therapy in treatment-naïve patients was pursued in 64.6% of PCC patients, compared with 49.3% of PMC patients ($P = 0.001$; see Supporting Information, Figure 2, in the online version of this article). This difference was driven both by higher rates of appropriate treatment among intermediate- to high-FRS patients (77.3% vs 60.6%; $P = 0.004$) and treatment of 26.6% of low-FRS PCC patients with CACS >75th percentile per MESA database (see Supporting Information, Figure 2, in the online version of this article).

Post-intervention, median LDL-C values were reduced in the PCC cohort compared with PMC patients ($P < 0.001$; Figure 1A). More high-FRS PCC patients (Table 2) achieved an LDL-C < 70 mg/dL ($P = 0.005$) and a non–high-density lipoprotein cholesterol (non–HDL-C) of <130 mg/dL ($P < 0.001$). Additionally, reduction in median LDL-C ($P = 0.030$), non–HDL-C ($P = 0.001$), and triglycerides ($P = 0.009$) was observed in the PCC cohort (Table 2).

CACS-stratified changes in median LDL-C (Figure 2B) were also significant in that patients with a CACS >100 AU had a more dramatic reduction in LDL-C post-intervention than did those with CACS of <100 AU ($P < 0.001$). The algorithm-driven risk-factor management among PCC patients resulted in significant reductions in median FRS across all risk categories when compared with PMC patients ($P < 0.001$ for all groups; Figure 2C).

3.5 | Management of BP

Baseline BP readings (Table 1) were well controlled (defined as SBP ≤120 mm Hg) in more than three-quarters of the patients in both groups ($P = 1.000$). There was no difference in mean SBP between the 2 cohorts post-intervention (126 ±13 mm Hg vs 129 ±14 mm Hg; $P = 0.189$) or the rate of well-controlled SBP ($P = 0.134$). There was a substantial 4% to 5% reduction in SBP in the high-FRS patients (Table 2) in both groups ($P = 0.237$ for between-group difference).

TABLE 1 Baseline characteristics

	PCC, N = 595	PMC, N = 595	*P* Value
Age, y	58.3 ± 10.0	57.9 ±10.9	0.480
Male sex	430 (72.3)	430 (72.3)	0.526
HTN	419 (70.4)	428 (71.9)	0.304
Hyperlipidemia	595 (100)	595 (100)	1.00
DM	152 (25.5)	151 (25.4)	0.500
Smoker	94 (15.8)	96 (16.1)	0.468
AF	6 (1.0)	4 (<1.0)	0.376
Treatment for HTN	392 (65.9)	302 (50.8)	<0.001
BP controlled	458 (77)	459 (77)	0.500
Initial SBP, mm Hg	128 (120–140)	128 (119–138)	0.772
BMI, kg/m^2	29 (26–33)	29.4 (26.3–32.8)	0.653
CV risk estimates			
Initial FRS	15.86 (9.11–26.58)	11.53 (6.73–18.89)	<0.001
High	238 (40)	135 (22.7)	<0.001
Intermediate	184 (31)	208 (35)	0.078
Low	173 (29.1)	252 (42.4)	<0.001
CACS obtained	493 (82.9)	65 (10.9)	<0.001
CACS, AU	131.56 ± 305.16	147.631 ± 420.35	0.704
Initial lipid values, mg/dL			
TC	195 (164–221)	194 (166–223)	0.833
LDL-C	109 (83–137)	115 (91–143)	0.036
TG	124 (81–191)	117 (85–169)	0.211
HDL-C	48 (39–63)	47 (40–57)	0.575
Non–HDL-C	140 (113–169)	144 (117–170)	0.309
ASA prescription following initial evaluation			
High FRS	169	51	<0.001
Intermediate FRS	106	64	<0.001
Low FRS	62	32	<0.001
Lipid medications			
Low-intensity statin therapy	51 (8.6)	40 (6.7)	0.275
Moderate-intensity statin therapy	176 (29.6)	169 (28.4)	0.708
High-intensity statin therapy	85 (14.3)	59 (9.9)	0.013
Nonstatin lipid therapy only	85 (14.3)	33 (5.5)	<0.001
No lipid therapy	198 (33.2)	294 (49.4)	<0.001
Combination lipid therapy[a]	94 (15.8)	24 (4.0)	<0.001
Initial BP values/treatment			
Treatment for HTN	392 (65.9)	302 (50.8)	<0.001
SBP, mm Hg	130 ± 16	129 ± 14	0.060
Initial SBP well controlled	458 (77.0)	459 (77.1)	1.000

Abbreviations: AF, atrial fibrillation; ASA, acetylsalicylic acid (aspirin); AU, Agatston units; BMI, body mass index; BP, blood pressure; CACS, coronary artery calcium score; CV, cardiovascular; DM, diabetes mellitus; FRS, Framingham Risk Score; HDL-C, high-density lipoprotein cholesterol; HTN, hypertension; IQR, interquartile range; LDL-C, low-density lipoprotein cholesterol; PCC, preventive cardiology clinic; PMC, propensity-matched cohort; SBP, systolic blood pressure; SD, standard deviation; TC, total cholesterol; TG, triglycerides. Data are presented as n (%), mean ± SD, or median (IQR).

[a] Statin and nonstatin medication use.

However, among the intermediate- and low-FRS patients post-intervention, significantly lower mean SBP was observed in the PCC cohort when compared with the PMC patients ($P = 0.001$ and $P < 0.001$, respectively), driven primarily by maintenance of stable BP readings from baseline compared with a higher proportion of patients with worsening in SBP readings post-intervention in the PMC group (Table 2).

4 | DISCUSSION

The use of APPs in a PCC utilizing guideline-based local risk factor–modification algorithms, combined with routine utilization of CACS, resulted in higher rates of lipid-lowering therapy initiation in treatment-naïve patients, more frequent appropriate escalation of lipid-lowering therapy, and more frequent use of combination lipid-

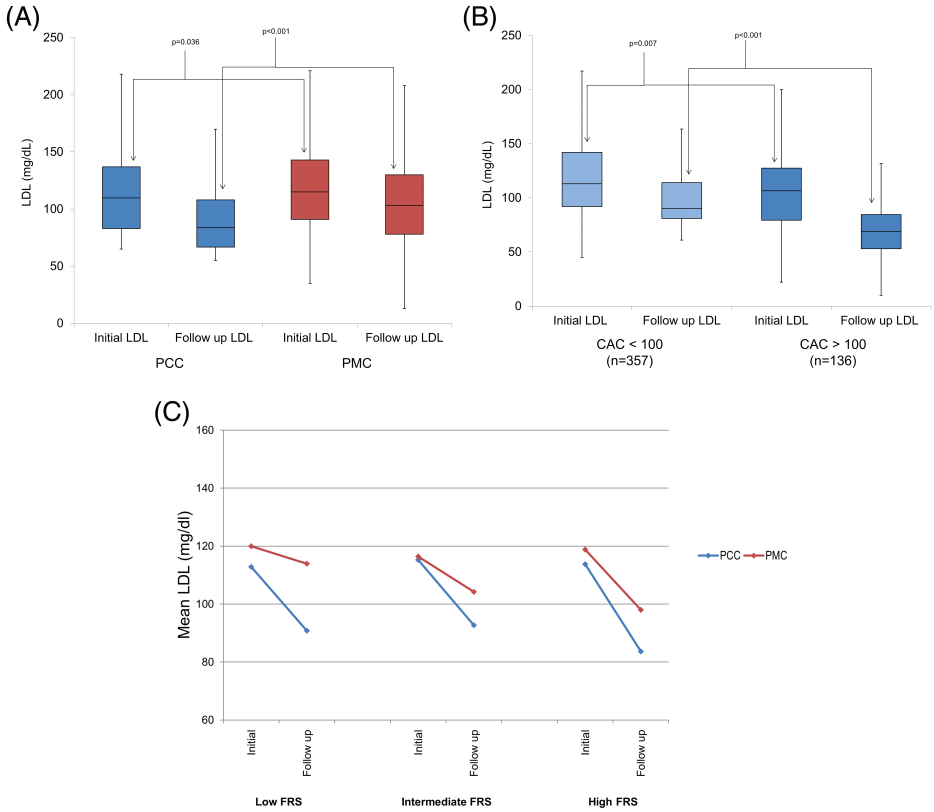

FIGURE 1 Observed LDL changes. (A) Median (IQR) initial and post-intervention LDL-C values in the PCC and PMC cohorts; (B) median (IQR) initial and post-intervention LDL-C values PCC patients who underwent CACS stratified by CACS < or > 100 arbitrary units; (C) mean LDL-C changes post-intervention in the PCC and PMC groups (P < 0.05 for follow-up LDL-C between all FRS groups). Abbreviations: CACS, coronary artery calcium scoring; FRS, Framingham Risk Score; IQR, interquartile range; LDL-C, low-density lipoprotein cholesterol; PCC, preventive cardiology clinic; PMC, propensity-matched cohort

lowering therapy when compared with age- and risk factor–matched patients treated by primary-care managers. Additionally, intermediate- and high-risk PCC patients were more commonly prescribed aspirin therapy, and mean SBP was lower following PCC intervention in intermediate- and low-FRS PCC patients. This resulted in a significant global risk reduction, regardless of initial FRS category, in the PCC cohort compared with PMC patients. Additionally, frequent use of CACS as an individualized risk-stratification approach identified a significant cohort of low-FRS patients with CACS exceeding the 75th MESA percentile.

In the United States, team-based care models comprising various combinations of cardiologists and APPs have been pioneered for the management of chronic cardiovascular conditions, ranging from chronic heart failure management to coronary artery disease and lipid-management clinics.[6,9,14–17] Additionally, data from a primary-care outreach network in Oregon demonstrated improved lipid monitoring

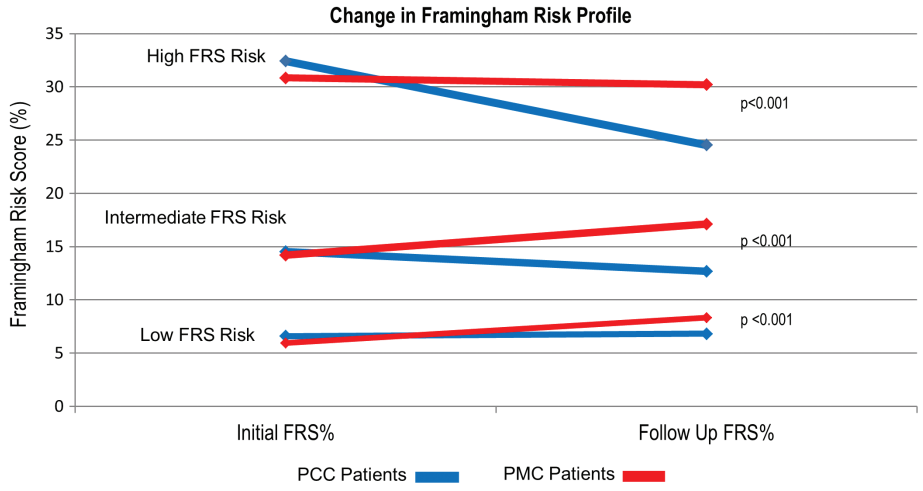

FIGURE 2 Changes in median FRS predicted 10-year ASCVD risk at initial and post-intervention in PCC and PMC groups. Abbreviations: ASCVD, atherosclerotic cardiovascular disease; FRS, Framingham Risk Score; PCC, preventive cardiology clinic; PMC, propensity-matched cohort

TABLE 2 Changes in clinical risk factors, medical treatment, and laboratory values

	High FRS			Intermediate FRS			Low FRS		
	PCC, n = 238	PMC, n = 135	*P* Value	PCC, n = 184	PMC, n = 208	*P* Value	PCC, n = 173	PMC, n = 252	*P* Value
Changes in lipid profile									
LDL <70 mg/dL	88 (36.9)	32 (23.7)	0.005	34 (18.5)	52 (25)	0.053	31 (17.9)	71 (28.2)	0.013
TG <150 mg/dL	161 (67.6)	82 (60.7)	0.112	48 (26.1)	51 (24.5)	0.480	120 (69.4)	155 (61.5)	0.024
Non–HDL-C <130 mg/dL	181 (76.1)	73 (54.0)	<0.001	50 (27.2)	39 (18.8)	0.055	162 (93.6)	220 (87.3)	<0.001
TC reduction, %	15 (3.03, 31.63)	9.94 (−8.1, 22.8)	0.030	130 (70.7)	121 (58.2)	0.019	8.7 (−3.6, 21.0)	1.0 (−13.5, 14.3)	<0.001
LDL-C reduction, %	21 (1.25, 45.12)	17 (−7.87, 35.63)	0.030	159 (86.4)	152 (73.1)	0.004	15.5 (−4.0, 35.8)	1.2 (−15.9, 22.6)	<0.001
HDL-C increase, %	0.04 (−0.08, 0.16)	0.04 (−0.06, 0.19)	0.403	8.8 (−3.6, 26.3)	2.0 (−9.9, 16.1)	0.001	0.0 (−0.1, 0.1)	0.0 (−0.1, 0.1)	0.593
Non–HDL-C reduction, %	21.5 (3.77, 42.5)	14.7 (−7.3, 30.1)	0.001	13.2 (−4.2, 35.2)	9.1 (−11.8, 27.7)	0.033	11.9 (−4.8, 32.7)	1.2 (−19.2, 18.3)	<0.001
TG reduction, %	17.1 (−9.76, 38.4)	5.98 (−24.0, 26.0)	0.009	0.0 (−0.1, 0.1)	0.0 (−0.1, 0.1)	0.101	2.1 (−32.3, 27.4)	−15.3 (−57.5, 13.1)	<0.001
Changes in BP									
SBP at follow-up, mm Hg	128 (122, 138)	132 (123, 141)	0.102	124 (116, 132)	129 (119, 138.2)	0.001	121 (111, 131)	127 (118, 135)	<0.001
SBP reduction, %	5.5 (−1.47, 12.0)	4.1 (−4.4, 11.95)	0.237	0.8 (−2.2, 7.1)	0.0 (−9.9, 7.8)	0.035	0 (−6.2, 5.8)	−2.3 (−11, 4.8)	0.022
Changes in lipid medications									
No therapy	20 (8.4)	30 (22.2)	<0.001	21 (11.4)	46 (22.1)	0.005	43 (24.9)	109 (43.3)	<0.001
Combination therapy[a] at follow-up	81 (34)	5 (3.70)	<0.001	38 (31.1)	9 (5.3)	<0.001	40 (23.1)	10 (3.96)	<0.001

Abbreviations: BP, blood pressure; FRS, Framingham Risk Score; HDL-C, high-density lipoprotein cholesterol; IQR, interquartile range; LDL-C, low-density lipoprotein cholesterol; PCC, preventive cardiology clinic; PMC, propensity-matched cohort; SBP, systolic blood pressure; TC, total cholesterol; TG, triglycerides. Data are presented as n (%) or median (IQR); IQR presented as "n, n" to avoid confusion between dashes and minus signs in values <0.

[a] Statin therapy with nonstatin therapy.

and higher rates of lipid-lowering prescriptions in patients with DM managed remotely by a clinical pharmacist-physician team.[18] Clinical pharmacist-led care teams effectively manage HTN across multiple health systems within a primary-care setting.[19] The PCC model presented in this analysis differs from published data in several ways. Through the creation of algorithms of care, there was less ambiguity for APPs regarding escalation of medical care. Additionally, the breadth of ASCVD risk factors addressed within a single clinic is novel. Finally, the frequent, up-front utilization of CACS to individualize ASCVD risk stratification allowed for a more patient-centered approach to primary prevention and may have improved compliance with recommended therapies.

ASCVD events continue to be the primary cause of morbidity and mortality in the first world. Data suggest that both physicians and APPs practicing within cardiology clinics do not routinely prescribe recommended medical therapies for various ASCVD events in patients who meet guideline criteria to be offered therapy. Physicians and APPs were compliant with ASCVD medication interventions in approximately 12% of patients in a large PINNACLE NCDR registry.[4] In our analysis, treatment in a PCC cohort resulted in 85.8% of patients eligible for lipid-lowering therapy actually being on lipid-lowering therapy, which is tremendously higher than reported compliance rates. More striking is the fact that compliance with lipid therapies was high at baseline in both cohorts (approximately 50%), thus demonstrating that this model is effective even in high-compliance healthcare systems.

Costs associated with care in our 2 cohorts could not be calculated due to a lack of patient-level billing data; however, the potential cost implications of improved ASCVD event prevention, in addition to direct patient care administered by APPs, has been demonstrated.[20] Medicare reimburses for care administered by APPs at up to 85% of a physician's rate.[21] Costs associated with long-term management of patients following a ASCVD event are higher, as shown in DM populations, among others.[22] Thus, higher rates of medication compliance observed in our PCC cohort would be assumed to lead to lower downstream event rates and reduction in direct costs due to APPs delivering the care and fewer hospitalizations/revascularization procedures. Additionally, indirect costs may decrease as a result of increased patient productivity, decreased days off work, and increased quality-adjusted life-years.

Utilization of CACS as part of the initial risk-stratification strategy was very high within the PCC cohort, at nearly 83%. Although this degree of CACS exceeds the volume utilized in most clinical practices nationwide, there is abundant data that CACS incrementally improves risk assessment alone and as an adjunctive test to global risk scores.[23–25] Among a cohort of the MESA population deemed statin ineligible, CACS reclassified 6.8% of patients upward, with a calculated number needed to screen of 14.7 to prevent a ASCVD event.[26]

Conversely, among statin candidates, CACS identified 44% of individuals with a CACS of 0 AU in whom statins were recommended but had an observed event rate of <0.5% per year.[27] There are robust data supporting treatment based on CACS compared with a risk-assessment strategy involving no imaging. The Early Identification of Subclinical Atherosclerosis by Noninvasive Imaging Research (EISNER) trial showed that use of CACS resulted in a lower FRS at 4 years of follow-up when compared with no scanning.[28] Additionally, a CACS-based strategy was also associated with favorable changes in SBP (P = 0.02), LDL-C (P = 0.04), and waist circumference (P = 0.01), without increased downstream medical testing.[28] A recently published meta-analysis found that, compared with risk-assessment strategies not involving CACS, individuals found to have CAC were more likely to be started on aspirin, statin therapy, and antihypertensive medications or to have intensification of baseline medical therapy.[29]

4.1 | Study limitations

Lipid-lowering treatment algorithms reported on in this analysis are based on ATP III treatment guidelines utilizing FRS for global risk estimation; thus, applicability to current clinical practice may be lessened.[10,30,31] Despite acceptable propensity matching for individual ASCVD risk factors between the groups, there was an observed baseline difference between median FRS between the groups. PMC patients were evaluated and treated by primary-care managers with numerous other clinical metrics to address, in addition to ASCVD prevention. Thus, a singularly focused PCC model would be expected to perform well in comparison. Finally, complications resulting from more aggressive risk-factor treatment, such as statin-induced myalgias or bleeding relating to aspirin, were not tracked in this population. Therefore, no comment or conclusions can be made about the potential negative ramifications of more aggressive treatment in the PCC population.

5 | CONCLUSION

A PCC staffed with APPs practicing under guideline-based treatment algorithms can effectively risk-stratify and aggressively treat patients with ASCVD risk with observed improvement in serum lipid panels and estimated global cardiovascular risk over an intermediate follow-up period.

Conflicts of interest

The views expressed herein are those of the authors and do not reflect the official policy or position of Brooke Army Medical Center, the US Army Medical Department, the US Army Office of the Surgeon General, the Department of the Army, the Department of the Air Force, the Department of Defense or the U.S. government. The authors declare no potential conflicts of interest.

ORCID

Dustin Thomas https://orcid.org/0000-0001-9445-9100

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

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