Aficamten for Symptomatic Nonobstructive Hypertrophic Cardiomyopathy

Primary Results of the ACACIA-HCM Trial

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on behalf of the ACACIA-HCM investigators

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Disclosures

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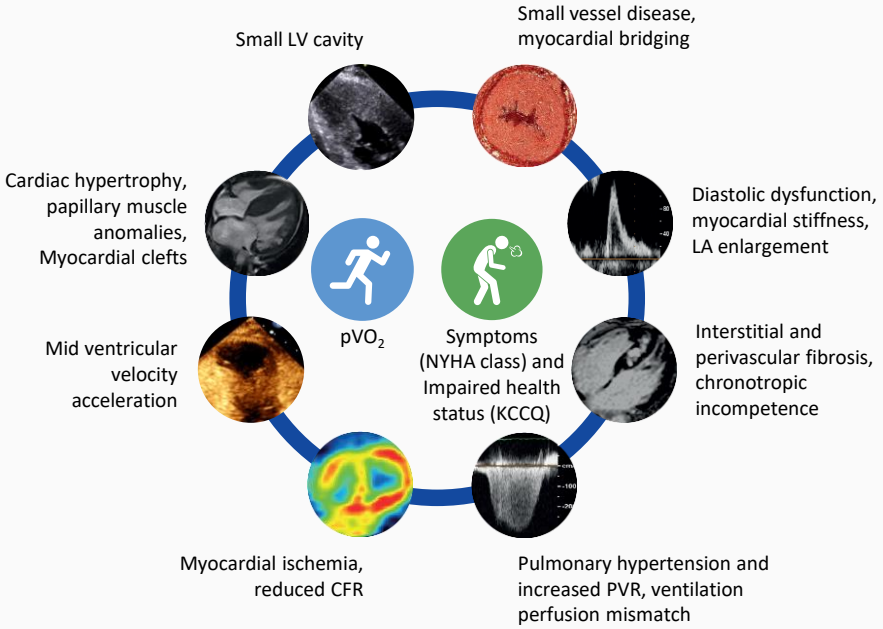
Background

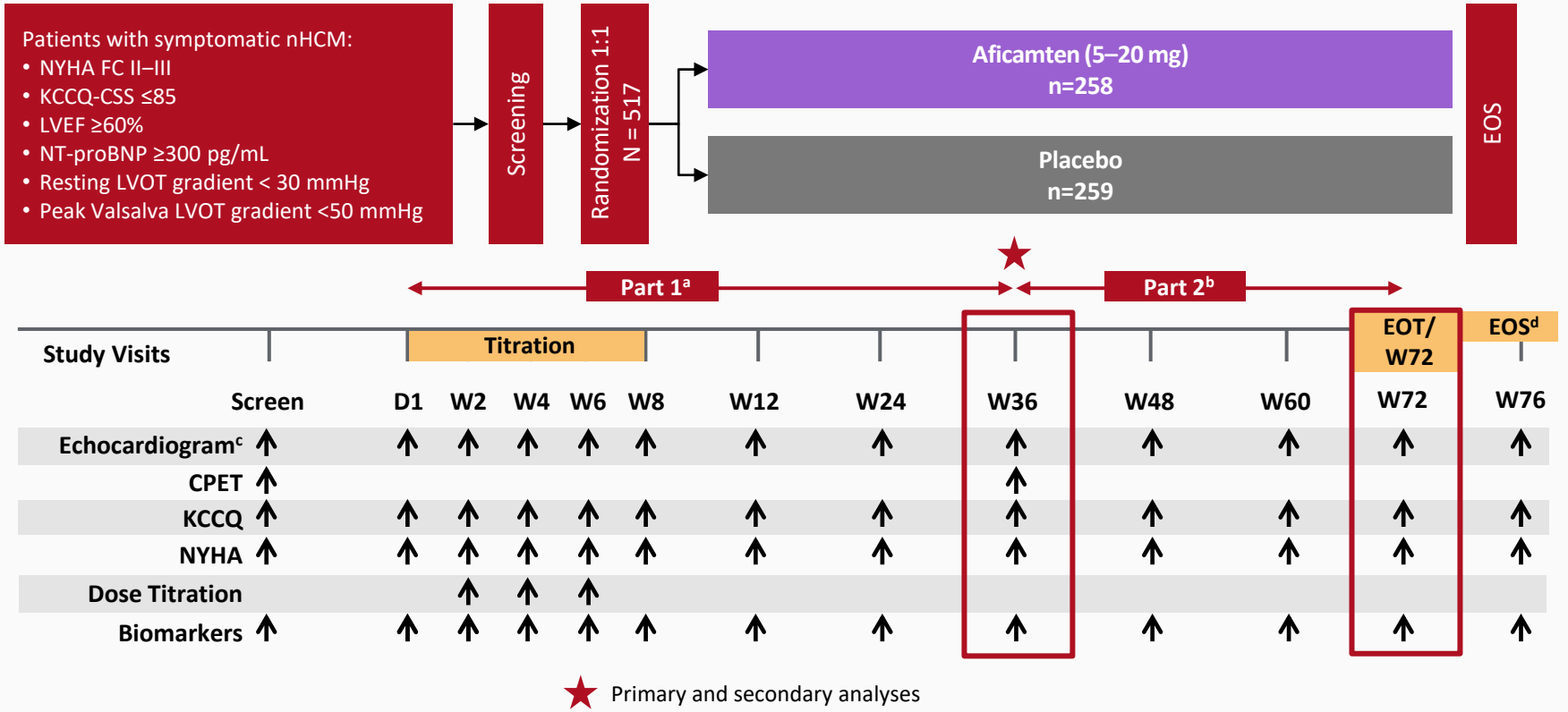
- Nonobstructive hypertrophic cardiomyopathy (nHCM) is a common heart disease associated with significant disease burden and no effective therapies.[1]
- Patients with nHCM experience debilitating symptoms with exertion that limit their exercise capacity.[2]
- Aficamten is an allosteric, reversible cardiac myosin inhibitor approved in obstructive HCM.[3] It has:
  - Favorable effects on diastolic function, a predominant pathophysiologic mechanism driving symptoms and decreased exercise capacity in nHCM.[4-7]
  - Favorable pharmacologic properties that allow for rapid titration, dose adjustment, and good tolerability.[8]



CFR, coronary flow reserve; HCM, hypertrophic cardiomyopathy; HR, heart rate; KCCQ, Kansas City Cardiomyopathy Questionnaire; LA, left atrial; MV, mitral valve; nHCM, nonobstructive hypertrophic cardiomyopathy; NYHA, New York Heart Association; pVO_2, peak oxygen uptake; PVR, pulmonary vascular resistance; SAM, systolic anterior motion; SV, stroke volume.

1. Ommen SR, et al. *Circulation.* 2024;149:e1239-311. **2.** Butzner M, et al. *JACC Adv* 2026;5: 102552. **3.** Myqorzo (aficamten) prescribing information. https://cytokinetics.com/wp-content/uploads/2025/12/MYQORZO_US_Prescribing_Information_and_Med_Guide.pdf. Accessed June 10, 2026. **4.** Hegde SM, et al. *J Am Coll Cardiol* 2024;84:1789-802. **5.** Masri A, et al. *J Card Fail* 2024;30:1439-448. **6.** Hartman JJ, et al. *Nat Cardiovasc Res* 2024;3:1003-16. **7.** Kawana M, et al. *Front Physiol* 2022;13:975076. **8.** Chuang C, et al. *J Med Chem* 2021:64:14142-152.
Adapted from Coats CJ, et al. *JACC Heart Fail* 2024;12(1):199-215.

Methods: Study Design



[a]Part 1: All patients followed until W36; [b]Part 2: When ≥200 patients reached 52 weeks of treatment, all remaining active study patients who completed W36 were eligible for an EOT visit followed by an EOS visit; [c]Site-read focused echocardiogram for titration visit (sole criterion); [d]Occurred ≥4 weeks after the last dose of investigational product.
CPET, cardiopulmonary exercise test; D, day; EOS, end of study; EOT, end of treatment; KCCQ-CSS, Kansas City Cardiomyopathy Questionnaire Clinical Summary Score; LVEF, left ventricular ejection fraction; LVOT, left ventricular outflow tract; NT-proBNP, N-terminal pro-B-type natriuretic peptide; nHCM, nonobstructive hypertrophic cardiomyopathy; NYHA, New York Heart Association; W, week.

Methods: Endpoints

- **Dual Primary Endpoints**
  - Change from baseline to Week 36 **in KCCQ-CSS.**
  - Change from baseline to Week 36 in **pVO_2 on CPET.**

Graphical multiplicity testing with equal alpha-split (0.025) for each

- **Secondary Efficacy Endpoints (hierarchical)**
  - Changes from baseline to Week 36 in:
    - Proportion of participants with improvement of ≥1 NYHA functional class.
    - Composite Z-score of pVO_2 and ventilatory efficiency slope.
    - Proportional change in NT-proBNP concentration.
    - Left atrial volume index.[a]
    - Time to first composite cardiovascular event.[b]
- **Safety Outcomes**
  - Safety endpoints included incidences of adverse events, LVEF <50% in association with clinical heart failure, and LVEF <40% leading to treatment interruption.

[a] In patients without atrial fibrillation or flutter on baseline electrocardiogram.
[b] Includes cardiovascular death, heart transplantation or left ventricular assist device, aborted sudden cardiac death, non-fatal stroke, heart failure hospitalization, or cardiac arrhythmia (atrial fibrillation or ventricular tachyarrhythmia requiring treatment or hospitalization).
CPET, cardiopulmonary exercise test; KCCQ-CSS, Kansas City Cardiomyopathy Questionnaire Clinical Summary Score; LVEF, left ventricular ejection fraction; NT-proBNP, N-terminal pro-B-type natriuretic peptide; NYHA, New York Heart Association; pVO_2, peak oxygen uptake.

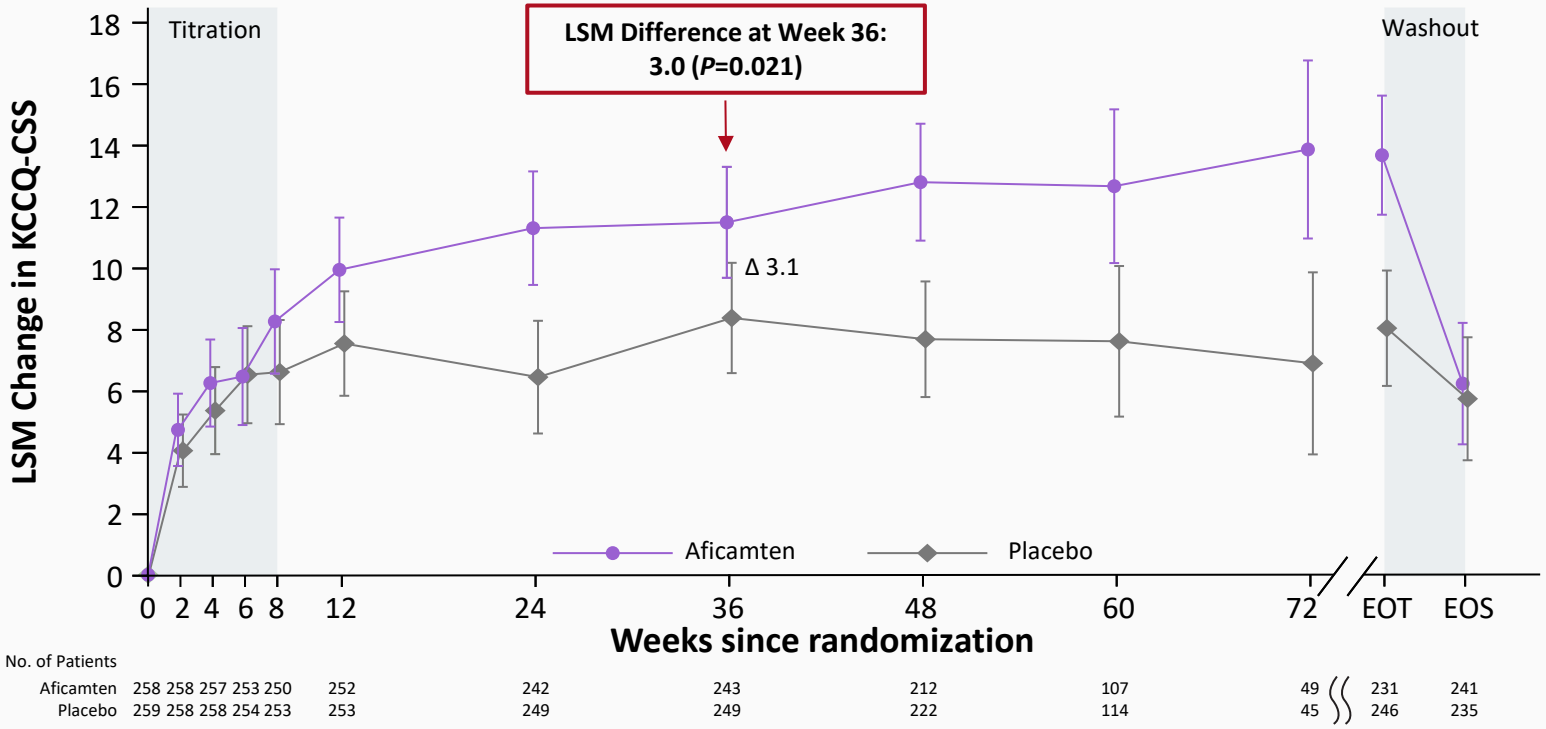
Results: Baseline Characteristics

Characteristic	Aficamten (n=258)	Placebo (n=259)
Age, y	55.7 ± 16.2	54.5 ± 16.0
Female sex	140 (54.3)	137 (52.9)
Race or ethnic group[a]		
White	200 (77.5)	188 (72.6)
Asian	30 (11.6)	39 (15.1)
Black	17 (6.6)	16 (6.2)
Medical history		
Hypertension	101 (39.1)	101 (39.0)
Familial HCM[b]	173 (67.1%)	168 (64.9%)
Paroxysmal AF/AFL	78 (30.2)	68 (26.3)
Persistent or permanent AF	20 (7.8)	19 (7.3)
Intracavitary obstruction	22 (8.5)	26 (10.0)
Background HCM therapy		
Beta-blocker	203 (78.7)	197 (76.1)
Nondihydropyridine CCB	31 (12.0)	39 (15.1)
None	34 (13.2)	42 (16.2)

Characteristic	Aficamten (n=258)	Placebo (n=259)
Implantable cardioverter-defibrillator	107 (41.5)	105 (40.5)
KCCQ-CSS	66.3 ± 16.2	65.0 ± 15.4
NYHA class III	84 (32.6)	74 (28.6)
NT-proBNP, pg/mL	906 [508–1529]	893 [492–1609]
hs-cTnI, ng/L	30 [15–77]	28 [14–113]
CPET		
pVO_2, mL/kg/min	18.0 ± 5.3	18.0 ± 5.1
Predicted pVO_2, %	55.3 ± 13.1	54.9 ± 12.7
Peak RER	1.2 ± 0.1	1.2 ± 0.1
VE/VCO_2	35.10 (7.9)	34.29 (6.7)
Echocardiographic parameters		
Valsalva LVOT-G, mmHg	11.8 ± 8.8	11.4 ± 8.5
Resting LVOT-G, mmHg	9.1 ± 6.1	8.2 ± 4.6
LVEF, %	67.9 ± 4.0	68.4 ± 3.4
Maximal LV wall thickness, mm	22.3 ± 5.2	23.1 ± 5.7
LAVI, mL/m^2	36.8 ± 10.9	37.5 ± 11.9

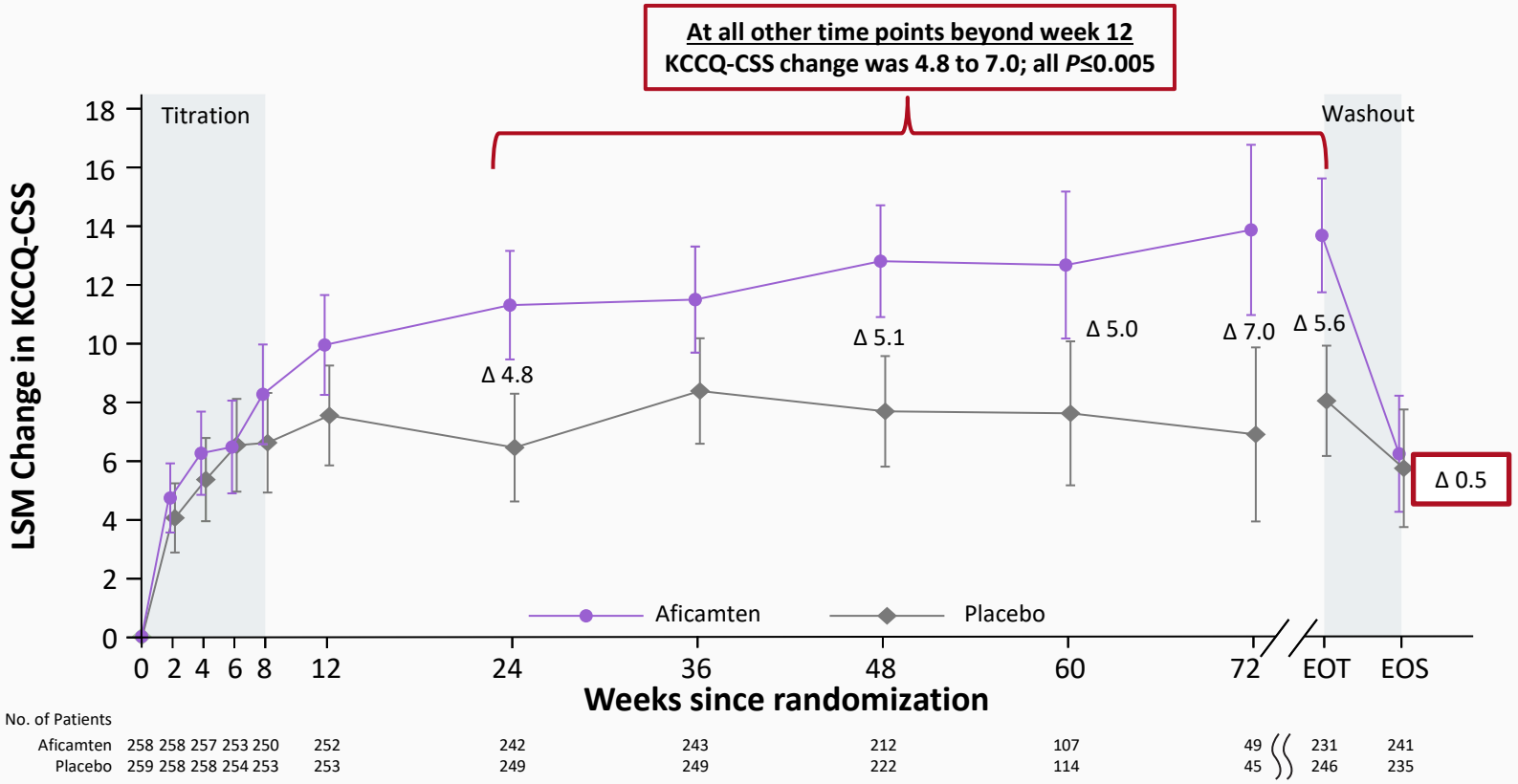
Data are n (%), mean ± SD or median [IQR]. Percentages may not total 100 because of rounding. [a]Race reported by the patient. [b]Defined as positive family history, known HCM mutation (site-reported) or genotype positive as part of ACACIA-HCM genetic study
AF/F, atrial fibrillation/flutter; CCB, calcium channel blocker; CPET, cardiopulmonary exercise testing; HCM, hypertrophic cardiomyopathy; hs-cTnI, high-sensitivity cardiac troponin I; KCCQ-CSS, Kansas City Cardiomyopathy Questionnaire Clinical Summary Score; LAVI, left atrial volume index; LV, left ventricular; LVEF, left ventricular ejection fraction; LVOT-G, left ventricular outflow tract gradient; NT-proBNP, N-terminal pro-B-type natriuretic peptide; NYHA, New York Heart Association; pVO_2, peak oxygen uptake; RER, respiratory exchange ratio.

Results: KCCQ-CSS (Dual Primary Endpoint at Week 36)



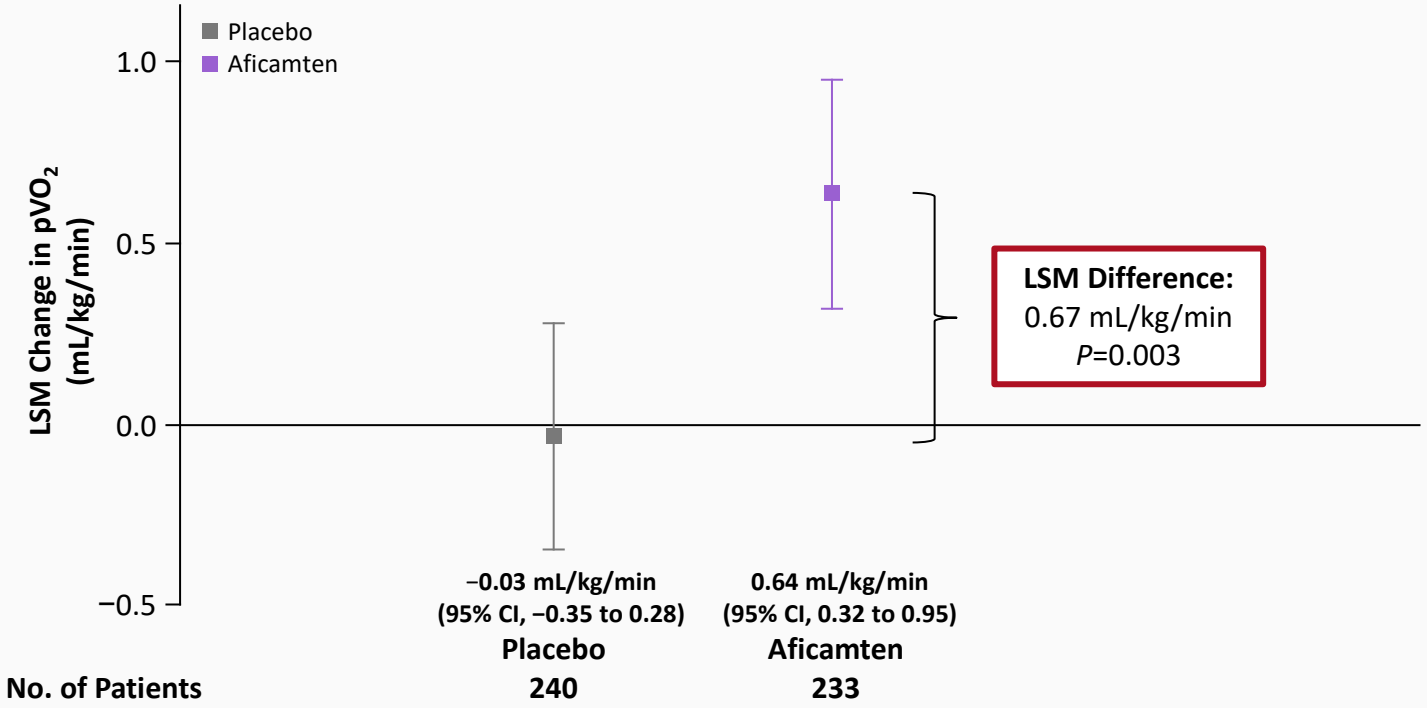
EOS, end of study; EOT, end of treatment; KCCQ-CSS, Kansas City Cardiomyopathy Questionnaire Clinical Summary Score; LSM, least squares mean.

Results: KCCQ-CSS (Dual Primary Endpoint at Week 36)



EOS, end of study; EOT, end of treatment; KCCQ-CSS, Kansas City Cardiomyopathy Questionnaire Clinical Summary Score; LSM, least squares mean.

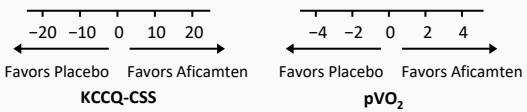
Results: Change in pVO_2 (Dual Primary Endpoint at Week 36)



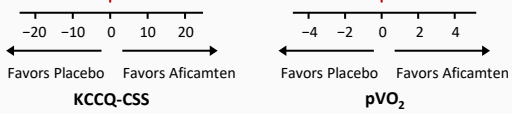
LSM, least squares mean; pVO_2, peak oxygen uptake.

Consistent Results for Both Endpoints Across Prespecified Subgroups

Subgroup	Aficamten (no. of patients)	Placebo (no. of patients)	KCCQ-CSS Estimate (95% CI)	pVO_2 Estimate (95% CI)
Overall	258	259	3.0 (0.5, 5.5)	0.7 (0.2, 1.1)
Sex				
Male	118	122	5.2 (1.7, 8.7)	1.0 (0.3, 1.7)
Female	140	137	1.1 (−2.5, 4.8)	0.4 (−0.2, 1.0)
Race				
White	200	188	2.8 (−0.2, 5.7)	0.5 (0.0, 1.1)
Asian	30	39	2.9 (−1.6, 7.3)	1.4 (0.1, 2.7)
Others	28	32	8.6 (−0.3, 17.5)	0.9 (−0.5, 2.4)
Ethnicity				
Hispanic or Latino	44	30	0.8 (−7.1, 8.8)	−0.1 (−1.3, 1.1)
Not Hispanic or Latino	210	207	2.8 (0.1, 5.6)	0.7 (0.2, 1.2)
Age group				
< 65 Years	168	175	3.7 (0.7, 6.7)	0.9 (0.3, 1.5)
≥ 65 Years	90	84	1.4 (−3.2, 6.1)	0.2 (−0.5, 0.8)
Baseline body-mass index				
≤ 35	229	244	2.7 (0.1, 5.3)	0.7 (0.2, 1.2)
> 35	29	15	8.6 (−3.0, 20.2)	0.3 (−0.9, 1.5)
Baseline NYHA class				
II	171	183	2.7 (−0.1, 5.5)	0.8 (0.3, 1.4)
III	84	74	4.7 (−0.8, 10.2)	0.3 (−0.4, 1.0)
Baseline KCCQ-CSS				
≤ Median (67.7)	126	140	1.8 (−2.5, 6.0)	0.3 (−0.3, 0.9)
> Median (67.7)	132	119	4.4 (1.7, 7.0)	1.0 (0.4, 1.7)
Baseline KCCQ-CSS				
≤ 85	229	239	3.5 (0.7, 6.2)	0.5 (0.0, 1.0)
> 85	29	20	−2.7 (−8.2, 2.9)	2.6 (0.9, 4.3)
Baseline LVEF				
< 65%	46	35	0.4 (−6.3, 7.1)	1.2 (0.0, 2.3)
≥ 65%	212	224	3.4 (0.7, 6.2)	0.6 (0.1, 1.1)

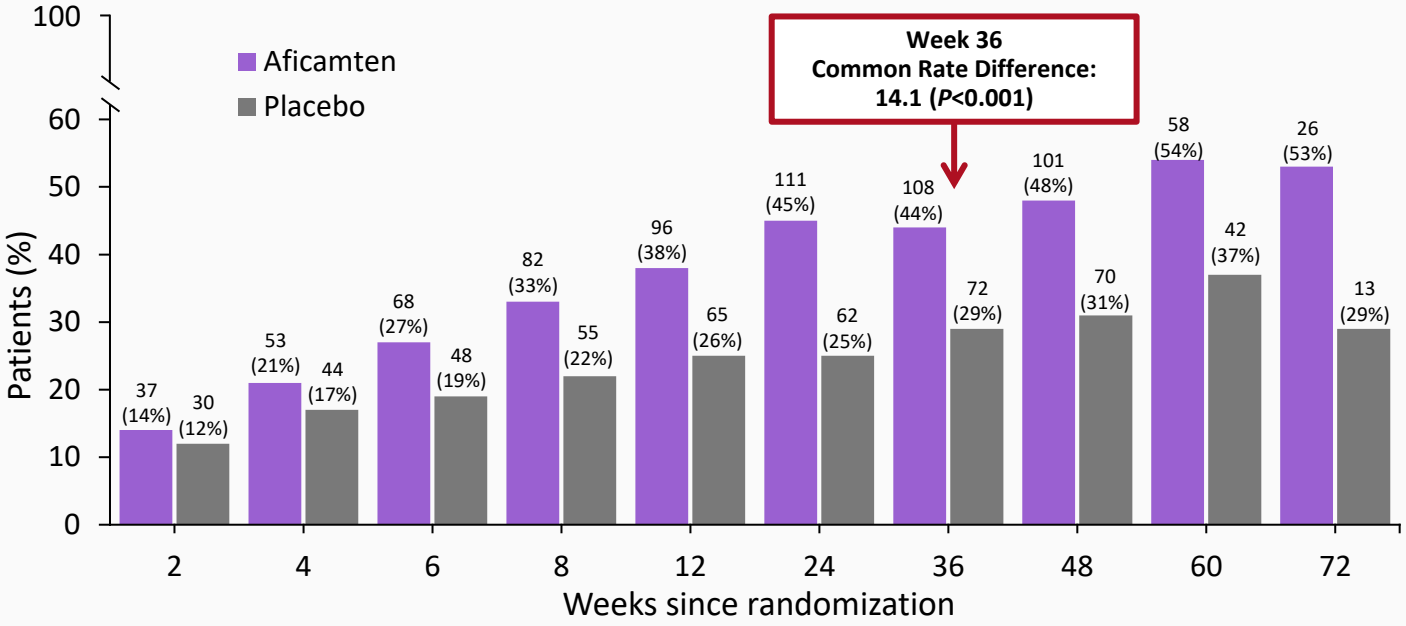


Subgroup	Aficamten (no. of patients)	Placebo (no. of patients)	KCCQ-CSS Estimate (95% CI)	pVO_2 Estimate (95% CI)
Baseline NT-proBNP				
≤ Median (893.0 pg/mL)	129	130	3.6 (−0.1, 7.3)	0.4 (−0.3, 1.0)
> Median (893.0 pg/mL)	129	129	2.4 (−1.0, 5.9)	1.0 (0.4, 1.6)
CPET				
Bicycle	107	126	3.5 (−0.2, 7.2)	0.7 (0.1, 1.4)
Treadmill	151	133	1.8 (−1.6, 5.2)	0.6 (0.0, 1.3)
Baseline pVO_2				
≤ Median (17.3 mL/kg/min)	126	134	1.9 (−2.0, 5.8)	0.8 (0.2, 1.3)
> Median (17.3 mL/kg/min)	132	125	3.9 (0.6, 7.2)	0.6 (−0.1, 1.3)
Beta blocker				
No use	55	62	5.2 (−0.4, 10.9)	0.4 (−0.6, 1.4)
Use	203	197	2.2 (−0.6, 5.1)	0.7 (0.2, 1.2)
Persistent or permanent AF				
Yes	20	19	11.9 (3.3, 20.6)	1.2 (−0.2, 2.7)
No	238	240	2.3 (−0.3, 5.0)	0.6 (0.2, 1.1)
Presence of intracavitary obstruction				
Yes	22	26	5.9 (−2.3, 14.0)	1.3 (−0.2, 2.7)
No	236	233	2.7 (0.0, 5.4)	0.6 (0.1, 1.1)
Prior septal reduction therapy				
Yes	37	23	5.3 (−3.7, 14.3)	0.8 (−0.5, 2.1)
No	221	236	2.8 (0.2, 5.4)	0.6 (0.2, 1.1)
Genotype				
Negative	47	49	5 (−1.3, 11.3)	0.6 (−0.4, 1.5)
Positive	124	107	2.9 (−1.0, 6.8)	0.7 (0.0, 1.4)
Variant of uncertain significance	26	37	6.2 (−1.3, 13.7)	0.9 (−0.2, 1.9)



AF, atrial fibrillation; CPET, cardiopulmonary exercise testing; KCCQ-CSS, Kansas City Cardiomyopathy Questionnaire Clinical Summary Score; LVEF, left ventricular ejection fraction; NT-proBNP, N-terminal pro-B-type natriuretic peptide; pVO_2, peak oxygen uptake.

Results: Improvement in ≥1 NYHA Functional Class

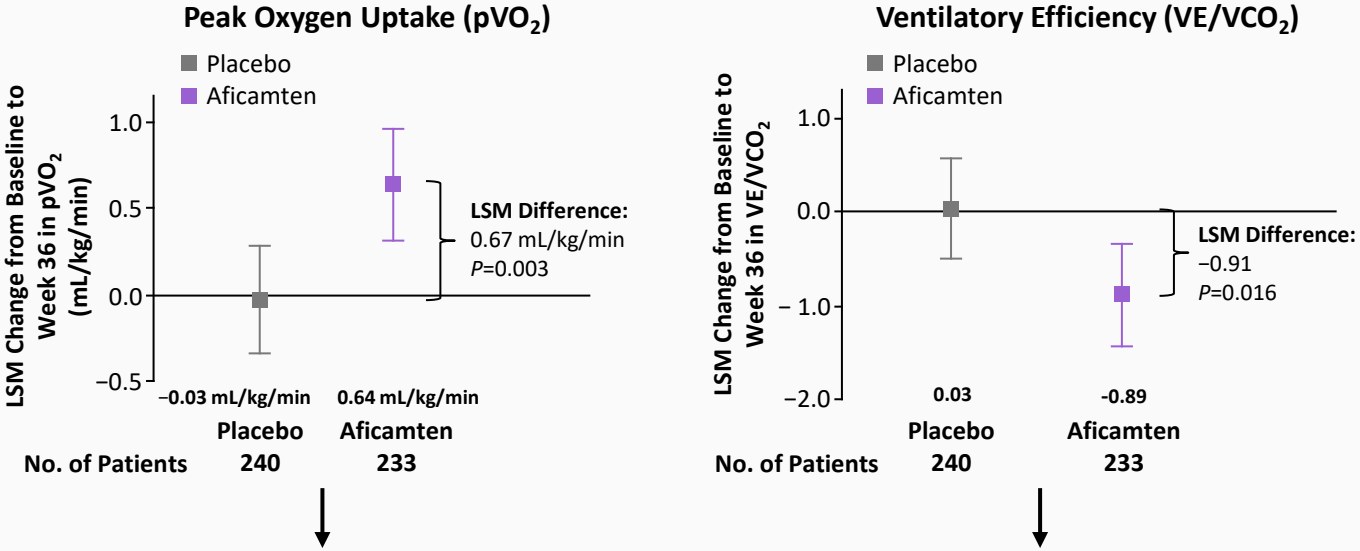


No. of Patients										
Aficamten	256	257	253	251	253	244	243	212	108	49
Placebo	257	257	254	251	253	249	247	223	114	45

First secondary endpoint tested.
NYHA, New York Heart Association.

Results: Improvement in Comprehensive Exercise Performance

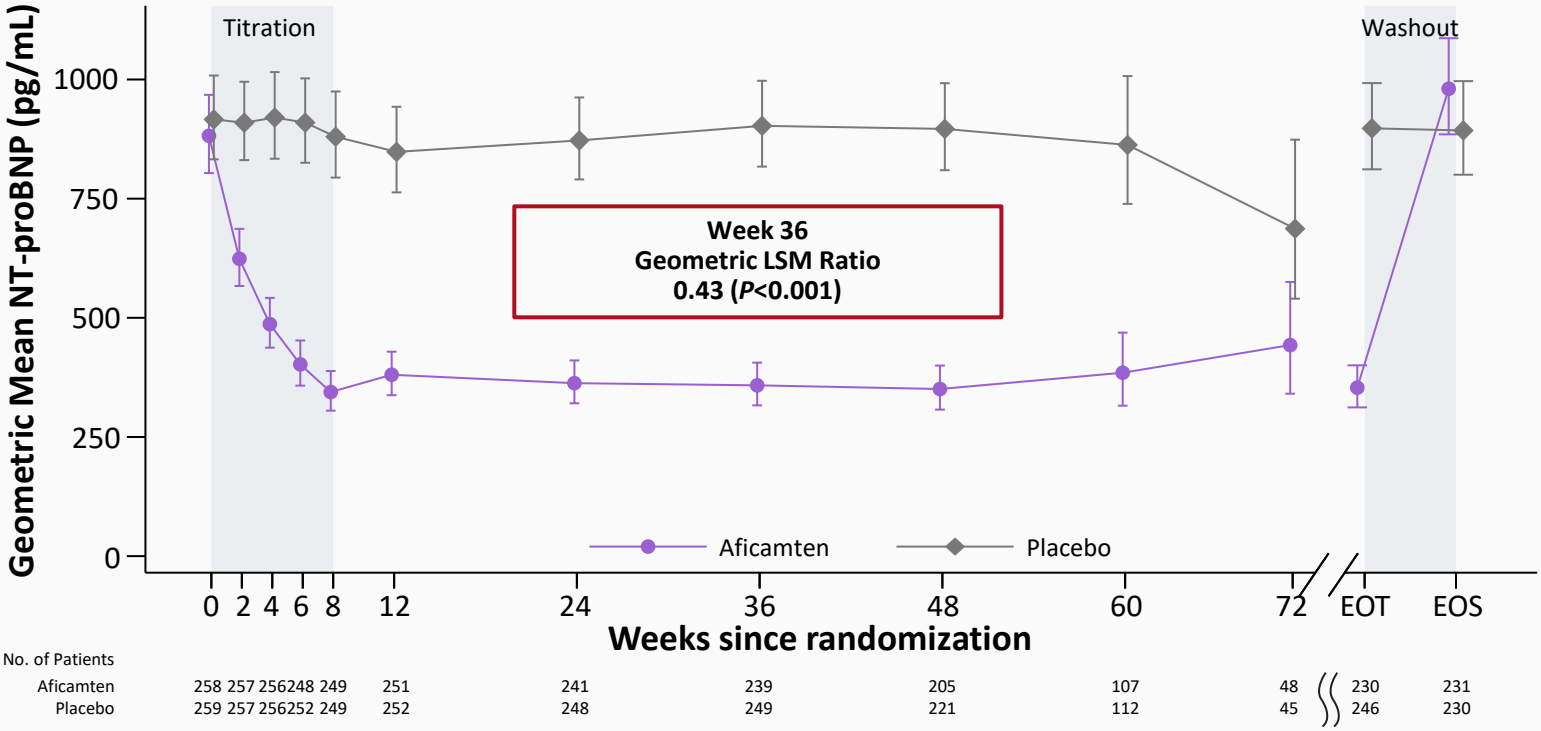
CPET Z-Score



Aficamten: 0.05 (95% CI, –0.01 to 0.11) vs. Placebo: –0.09 (95% CI, –0.14 to –0.04)
Mean difference 0.14 (95% CI, 0.07 to 0.22), *P*<0.001

Second secondary endpoint tested.
LSM, least squares mean, CPET, Cardiopulmonary Exercise Test

Results: NT-proBNP Concentration Over Time

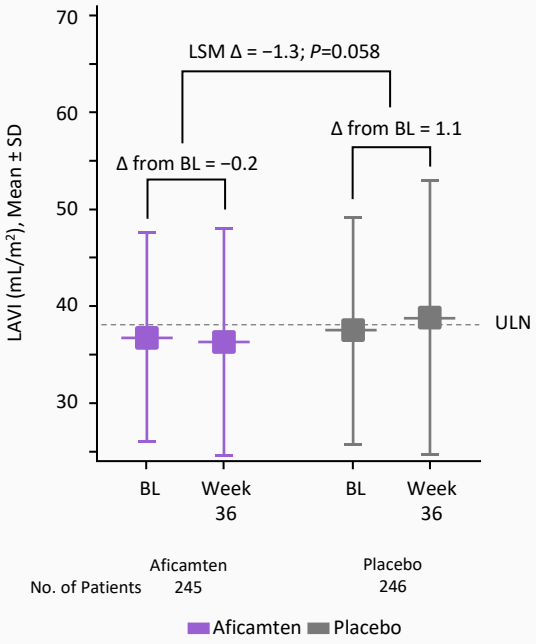


Third secondary endpoint tested.
EOS, end of study; EOT, end of treatment; LSM, least squares mean; NT-proBNP, N-terminal pro-B-type natriuretic peptide.

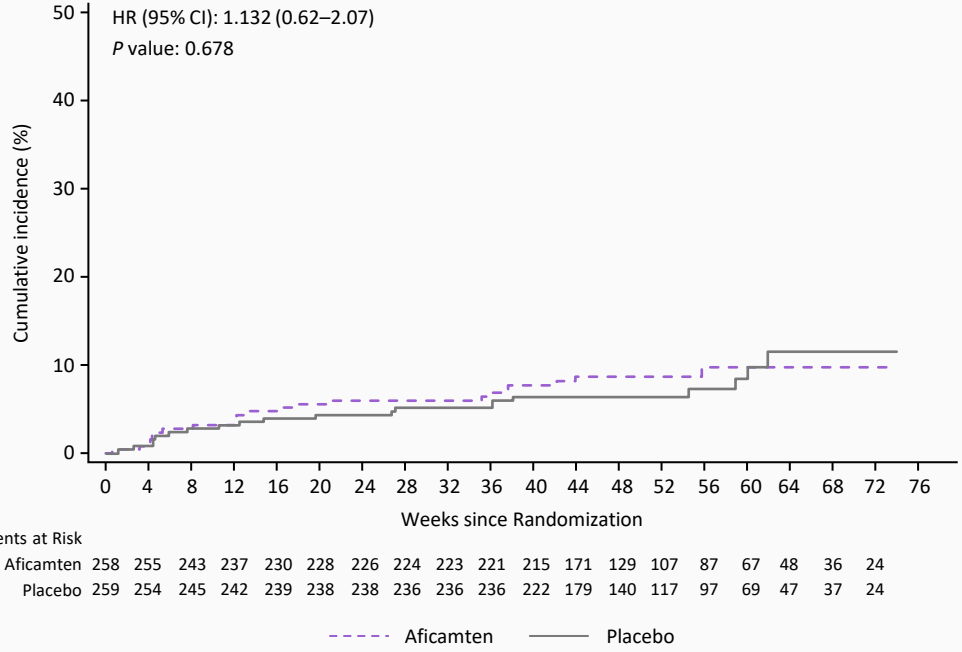
Results: LAVI and Time to First Cardiovascular Event

ACACIA HCM

Change from BL at Week 36 in LAVI



Time to First Composite Cardiovascular Event[a]



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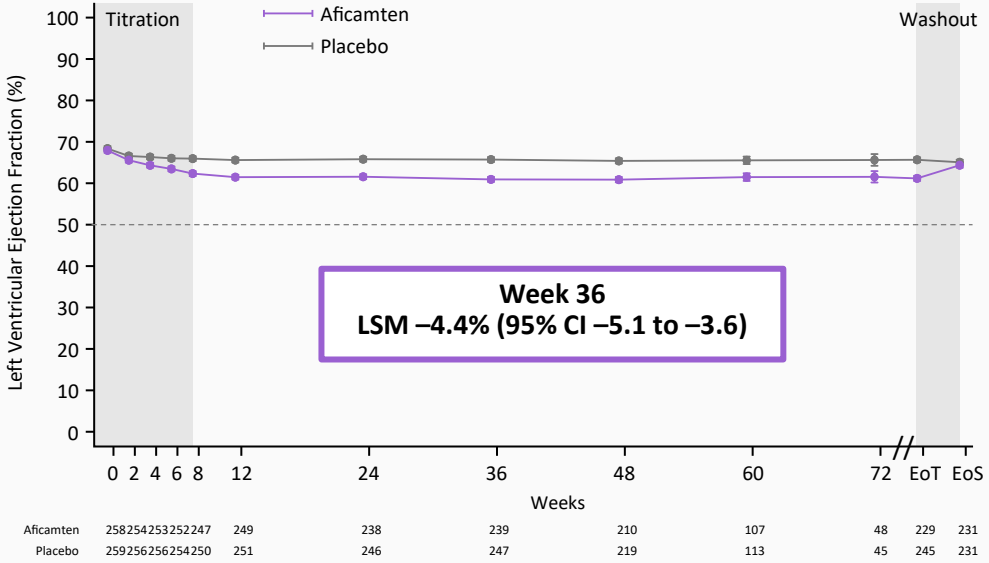
Primary analysis included patients not in AF at BL.
Δ, change; AF, atrial fibrillation; BL, baseline; HR, hazard ratio; LAVI, left atrial volume index; LSM, least squares mean; ULN, upper limit of normal.
[a]CV Events: Time to first event of cardiovascular death, heart transplantation or left ventricular assist device, aborted sudden cardiac death, non-fatal stroke, heart failure hospitalization, or cardiac arrhythmia (atrial fibrillation or ventricular tachyarrhythmia) requiring treatment or hospitalization.

Results: Safety Outcomes

- Aficamten was generally well-tolerated without new safety signals
  - Aficamten was titrated to maximum tolerated doses as guided by LVEF
  - 80.7% of patients were on 15-20 mg by the end of the titration period, and 79.4% of patients were on 15-20 mg by the end of the treatment period.

- At week 36, the LVEF LSM difference between aficamten and placebo was −4.4% (95% CI, −5.1 to −3.6).
  - Only 3% of the patients who received aficamten had treatment interruption due to reduced left ventricular ejection fraction



EOS, end of study; EOT, end of treatment; LSM, least squares mean; LVEF, left ventricular ejection fraction.

Results: Safety Outcomes

Event, n (%)	Aficamten (n=258)	Placebo (n=259)
Any AE	207 (80.2)	217 (84.1)
Any nonfatal AE that led to treatment discontinuation	18 (7.0)	5 (1.9)
Any nonfatal AE that led to early study discontinuation	9 (3.5)	2 (0.8)
Any SAE	52 (20.2)	38 (14.7)
Death	0 (0)	3 (1.2)
Heart Failure	12 (4.7)	3 (1.2)
New AF	4 (1.6)	3 (1.2)
Incidence of LVEF <50%	27 (10.5)	2 (0.8)
Incidence of LVEF <50% with any serious cardiac failure AE[a]	2 (0.8)	0 (0)
Incidence of LVEF <40%	7 (2.7)	0 (0)

All heart failure events in aficamten treated patients occurred during titration/prior to Week 12 and were generally responsive to a short course of diuretic therapy.

Of the 27 LVEF <50% events, 21 completed treatment while receiving the same or lower doses of aficamten.

AE, adverse event; AF, atrial fibrillation; LVEF, left ventricular ejection fraction; SAE, serious adverse event

Global Efficacy of Aficamten in Nonobstructive Hypertrophic Cardiomyopathy

A Pre-specified Analysis

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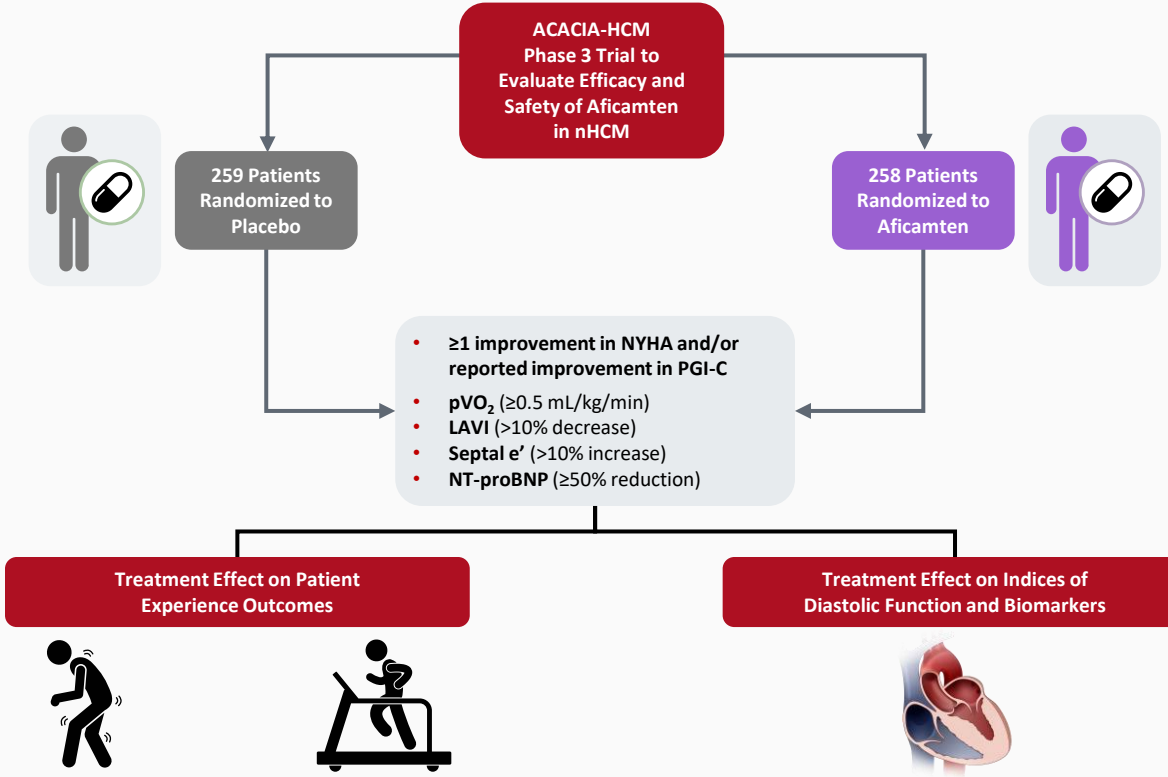
METHODS



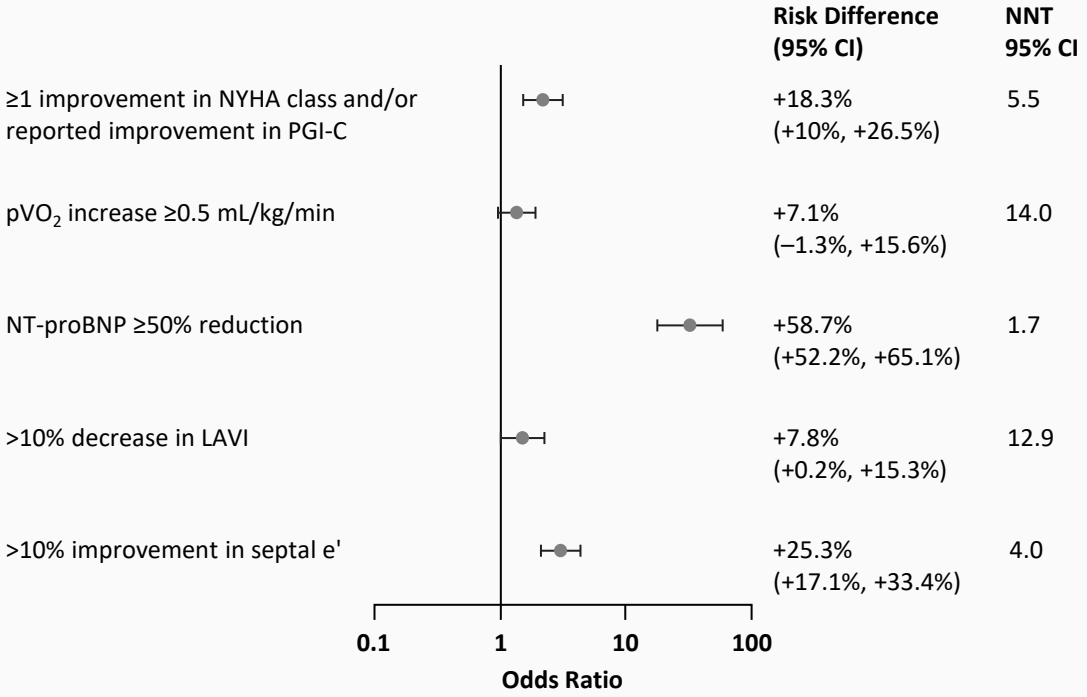
Patient Global Impression of Change (PGI-C)

Please choose the response below that best describes the overall change in your symptoms and overall health since you started taking the study medication.

- ☐ Very much better
- ☐ Moderately Better
- ☐ A Little Better
- ☐ No Change
- ☐ A Little Worse
- ☐ Moderately Worse
- ☐ Very Much Worse

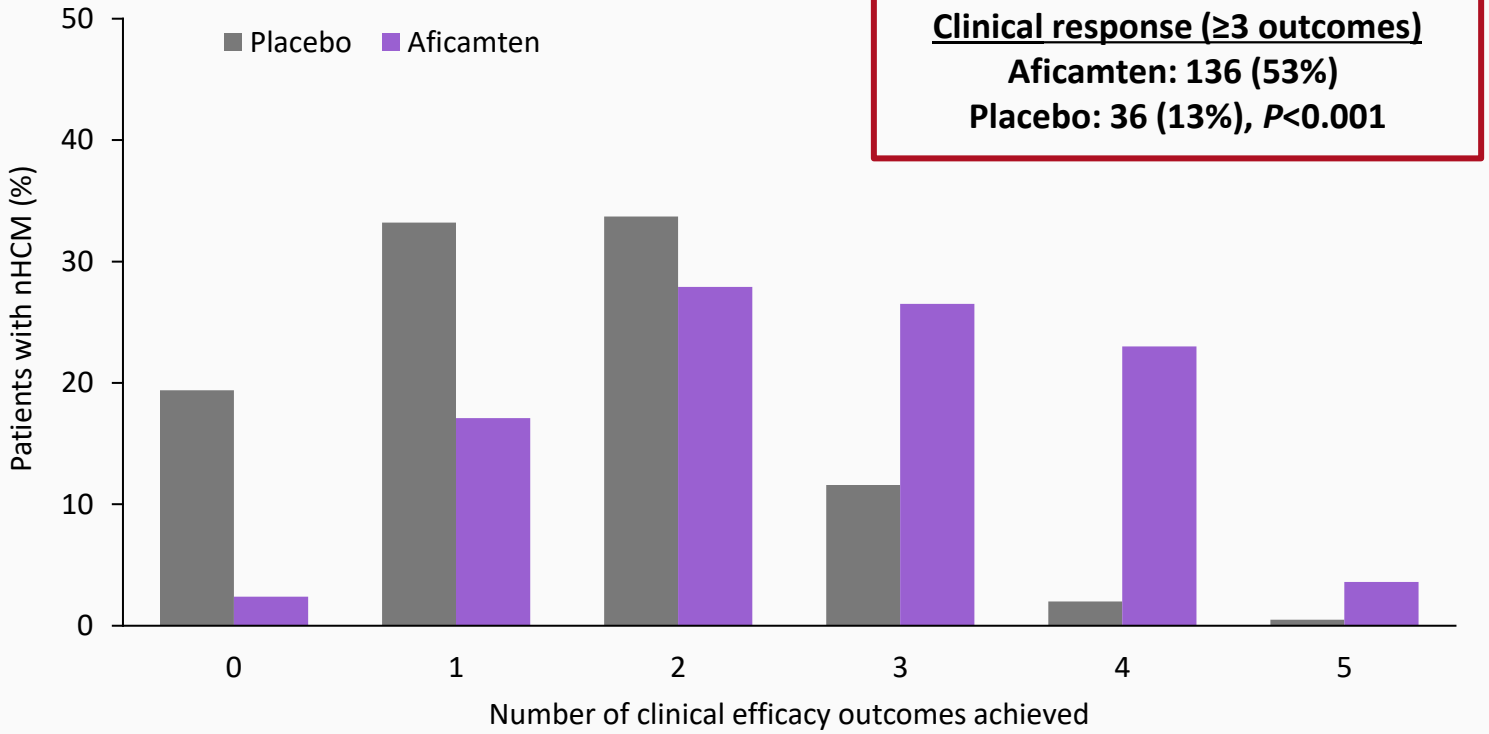
LAVI, left atrial volume index; NT-proBNP, N-terminal pro–B-type natriuretic peptide; nHCM, nonobstructive hypertrophic cardiomyopathy; NYHA, New York Heart Association; pVO_2, peak oxygen uptake.

Results: Effect of Aficamten vs Placebo on Clinical Efficacy Outcomes



LAVI, left atrial volume index; NNT, number needed to treat; NT-proBNP, N-terminal pro–B-type natriuretic peptide; NYHA, New York Heart Association; PGI-C, Patient Global Impression of Change; pVO_2, peak oxygen uptake.

Global Efficacy across Five Domains at Week 36



Clinical response (≥3 outcomes)
Aficamten: 136 (53%)
Placebo: 36 (13%), *P*<0.001

nHCM, nonobstructive hypertrophic cardiomyopathy.

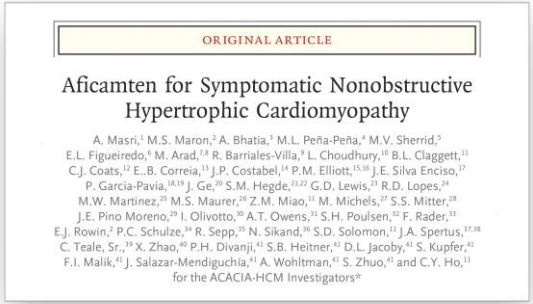
Conclusions

- Aficamten improved exercise capacity, patient-reported health status, and symptoms, as compared with placebo in patients with symptomatic nHCM.
- Over 36 weeks of treatment, aficamten yielded statistically significant improvements in efficacy as compared with placebo:
  - Dual primary endpoints (KCCQ-CSS and pVO_2).
  - Three of five secondary endpoints (NYHA class, exercise performance [Z-score], and NT-proBNP).
- Aficamten was generally well-tolerated, and the majority of reductions of LVEF < 50% were managed by simple down titration.
- In a prespecified responder analysis, aficamten positively affected a wide range of relevant clinical endpoints.
- These findings show that aficamten is an effective treatment for patients with nHCM.
- The 5-year ongoing FOREST-HCM trial will provide further data on the longer-term efficacy and safety of aficamten in nHCM, where >80% of eligible participants from ACACIA-HCM are currently enrolled.

KCCQ-CSS, Kansas City Cardiomyopathy Questionnaire Clinical Summary Score; nHCM, nonobstructive hypertrophic cardiomyopathy; NT-proBNP, N-terminal pro-B-type natriuretic peptide; NYHA, New York Heart Association; pVO_2, peak oxygen uptake.

Simultaneous Publications in *New England Journal* of Medicine and in *Circulation*

The NEW ENGLAND JOURNAL of MEDICINE



ORIGINAL ARTICLE

Aficamten for Symptomatic Nonobstructive Hypertrophic Cardiomyopathy

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JOURNALS

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Global Efficacy of Aficamten in Nonobstructive Hypertrophic Cardiomyopathy: Results from ACACIA-HCM

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