ISSUE 02
2026

Medeasy Review

EVIDENCE FOR A BRIGHTER PRACTICE

CLINICAL
PRACTICAL
FORWARD-LOOKING

SPECIAL FEATURE

Colchicine

Why Do the Trials Disagree?

A Medeasy evidence synthesis and updated clinical perspective.

ESC 2026
Five findings worth knowing

SINGLE-AF
Intermediate risk still matters

Baxdrostat
The aldosterone comeback

CARDIO-TTRansform
When target engagement is not enough

TAVI-PCI
Flexibility in sequence

Digital Medicine
Can an app really lower blood pressure?



Colchicum autumnale — the autumn crocus, a historic natural source of colchicine.

A MEDEASY PUBLICATION

medeasy+
Healthcare made easy

Dear Colleagues,

Medicine rarely changes because of one headline. More often, practice evolves when new evidence challenges what we thought we already knew.

In this issue of Medeasy Review, we look beyond whether a trial was simply "positive" or "negative." We ask the questions that matter at the bedside:

How reliable is the result?

Who was actually studied?

How large is the benefit?

Should it change what we do?

Our centerpiece examines low-dose colchicine for secondary cardiovascular prevention. Several landmark trials demonstrated benefit, while newer studies produced less consistent results. We revisit the randomized evidence and ask why the trials may disagree.

We also review important new cardiovascular data from ESC Congress 2026 — from anticoagulation in intermediate-risk atrial fibrillation to transthyretin cardiomyopathy, resistant hypertension and structural heart intervention.

OUR GOAL

Make new evidence easier to understand — and easier to use.



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EVIDENCE UPDATE

FIVE FINDINGS WORTH KNOWING

New data. Different questions. Practical implications.

NEW
EVIDENCE
BRIGHTER
PERSPECTIVE
BETTER
CARE



Science today. Healthier tomorrows.

01 SINGLE-AF

Intermediate-risk AF may deserve another look.

0.5% DOAC vs **1.5%** No anticoagulation

PRIMARY COMPOSITE

HR 0.31
(95% CI 0.10–0.94)
p = 0.028

Absolute difference: about 1 percentage point over 24 months.

A strong relative effect — but a modest absolute separation.

TWO MORE IMPORTANT SIGNALS

02 CARDIO-TTRansform

A familiar question with new data.

29.4% vs **32.2%**

PRIMARY ENDPOINT

Rate ratio 0.89
Primary endpoint neutral • p = 0.28

03 TAVI-PCI

Two strategies. Similar outcomes.

22.2% vs **24.2%**

1-YEAR PRIMARY COMPOSITE

Risk difference −2.0 pp
95% CI −7.4 to 3.4 • Noninferior

THE MEDEASY TAKE

The headline is only the beginning.

Effect size, endpoint design and patient selection still determine what matters clinically.

SAME QUESTIONS. BETTER ANSWERS.

05

TWO MORE STUDIES TO WATCH

Blood pressure. STEMI. Two very different clinical questions.

04 • BAXHTN

HYPERTENSION & CARDIORENAL RISK

DIFFERENT QUESTIONS.
A HEALTHIER TOMORROW.

Aldosterone synthase inhibition moves into focus.

–9.8 mmHg

PLACEBO-CORRECTED SEATED SBP REDUCTION AT 12 WEEKS

Baxdrostat • 1 mg: –8.7 mmHg • 2 mg: –9.8 mmHg

Potassium >6.0 mmol/L: 3.0% at 2 mg vs 0.4% with placebo.



DIFFERENT MECHANISMS.
REAL-WORLD POTENTIAL.

05 • AIR-STEMI

STEMI WITH MULTIVESSEL DISEASE

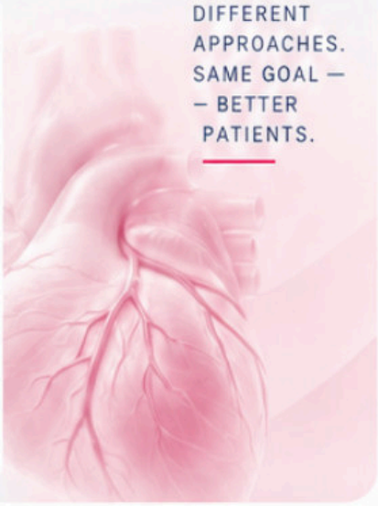
GUIDING CARE
FOR BETTER OUTCOMES.

8.9% vs 13.7%

PRIMARY ENDPOINT

HR 0.62 (95% CI 0.47–0.83) • p<0.001

Functional coronary angiography guidance vs angiography alone.
Median follow-up 17.9 months • all-cause mortality not significantly different.



DIFFERENT APPROACHES.
SAME GOAL — — BETTER PATIENTS.

THE MEDEASY TAKE

Big numbers need the right context.

Magnitude matters — but so do safety, endpoints, follow-up and what was actually compared.

DATA
PERSPECTIVE
BETTER CARE

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EVIDENCE SYNTHESIS + META-ANALYSIS

COLCHICINE: WHY DO THE TRIALS DISAGREE?

Same drug. Different answers.

Low-dose colchicine reduced events in landmark coronary trials, yet newer post-MI and cerebrovascular studies have been neutral. This section asks why benefit may look stronger in some populations than in others.



THE CLINICAL QUESTION

Is colchicine broadly useful in ASCVD — or does benefit depend on the population and clinical setting?

THE COLCHICINE EVIDENCE AT A GLANCE

DIFFERENT CONTEXTS. DIFFERENT OUTCOMES.

ESTABLISHED CAD
LoDoCo2
HR 0.69

RECENT MI
COLCOT
HR 0.77

NEWER TRIALS
CLEAR / CHANCE-3
Neutral

TRIAL	POPULATION (n)	EVENT RATE (colchicine vs placebo)	HAZARD RATIO (HR)
COLCOT	n = 4,745	5.5% vs 7.1%	**HR 0.77** (95% CI 0.61–0.96)
LoDoCo2	n = 5,522	6.8% vs 9.6%	**HR 0.69** (95% CI 0.57–0.83)
CLEAR SYNERGY	n = 7,062	9.1% vs 9.3%	**HR 0.99** (95% CI 0.85–1.16)
CHANCE-3	n = 8,343	6.3% vs 6.5%	**HR 0.98** (95% CI 0.83–1.16)
CONVINCE	n = 3,154	Neutral primary result	**HR ~0.84** (95% CI 0.68–1.05)

WHAT MAY EXPLAIN THE DIFFERENCE?

SAME DRUG. DIFFERENT CONTEXT.

1. POPULATION
Chronic CAD, recent MI, and stroke/TIA are not interchangeable populations.

2. ENDPOINTS
Trials used different composites, event profiles, and follow-up windows.

3. TRIAL CONTEXT
Background therapy, adherence, and event rates also shape the observed effect.

> The totality of evidence suggests a modest reduction in recurrent cardiovascular events, with the clearest signal in established coronary disease.

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METHODS

FROM TRIALS to totality of evidence

Different studies can answer different questions. Understanding how trials are designed helps us interpret the totality of evidence.



SAME QUESTIONS. DEEPER INSIGHTS. BETTER CARE.

THE PICO FRAMEWORK

A CLEARER LENS ON THE EVIDENCE.

P — POPULATION

Established ASCVD

Chronic CAD • recent MI/ACS • ischemic stroke/TIA

I — INTERVENTION

Low-dose colchicine

Commonly 0.5 mg daily

C — COMPARATOR

Placebo / usual care

Randomized evidence

O — OUTCOMES

Events + safety

MACE • MI • stroke • death • revascularization

We also asked: could trial quality explain the disagreement?

CRITICAL APPRAISAL. CLEARER CONCLUSIONS.

Randomization
Was allocation credible?

Blinding
Could knowledge of treatment distort care or reporting?

Missing data
Could attrition change the estimate?

Outcome assessment
Were events captured consistently?

Adherence
Did enough patients remain exposed?

Applicability
Does this resemble real practice?

MEDEASY BOTTOM LINE

A different result is not automatically bias. Population, timing, exposure and endpoint composition can legitimately change the estimate.

SAME EVIDENCE. BETTER DECISIONS.

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MEDICINE. PEOPLE. A HEALTHIER TOMORROW.

EVIDENCE SYNTHESIS + META-ANALYSIS

Colchicine and Major Cardiovascular Events



SCIENCE TODAY, HEALTHIER TOMORROWS.

A contemporary synthesis of randomized trials suggests a modest overall reduction in major cardiovascular events, with important differences across patient populations.

Pooled result

9 RCTs • 30,557 patients
Modest overall reduction in recurrent cardiovascular events.

RR 0.80
(95% CI 0.70–0.92)

20%
relative risk reduction

Selected Major Colchicine Trials

Trial (Year)	Population	Colchicine vs Control	Effect (95% CI)	Interpretation
LoDoCo	stable CAD	–	HR 0.33 (95% CI 0.18–0.59)	Favorable
COLCOT	Recent MI	5.5% vs 7.1%	HR 0.77 (95% CI 0.61–0.96)	Favorable
LoDoCo2	Stable CAD	6.8% vs 9.6%	HR 0.69 (95% CI 0.57–0.83)	Favorable
COPS	ACS	24 vs 38 primary events		Neutral / imprecise
CLEAR SYNERGY	Recent MI	9.1% vs 9.3%	HR 0.99 (95% CI 0.85–1.16)	Neutral
CHANCE-3	Stroke / TIA	6.3% vs 6.5%	HR 0.98 (95% CI 0.83–1.16)	Neutral
CONVINCE	Stroke / TIA	–	HR ~0.84 (95% CI 0.68–1.05) Open-label with blinded endpoint assessment; stopped before	Inconclusive
Overall pooled benchmark		**7.59% vs 8.95%**	**RR 0.80** (95% CI 0.70–0.92)	Modest overall benefit



Selected Major Colchicine Trials

Selected major trials are shown for clinical context. Primary endpoint definitions differ across trials and should not be interpreted as directly interchangeable.

MODEST OVERALL BENEFIT

Pooled randomized evidence supports a modest reduction in recurrent cardiovascular events.

HETEROGENEITY MATTERS

Benefit is clearest in established coronary disease, contemporary post-MI and cerebrovascular trials are less consistent.

SELECTIVE USE

Do not apply one pooled estimate uniformly across every ASCVD population.

MEDEASY BOTTOM LINE: The totality of randomized evidence supports a modest reduction in recurrent cardiovascular events, with the clearest signal in established coronary diseaae. Benefit is less consistent in contemporary post-MI and cerebrovascular populations.

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EVIDENCE SYNTHESIS + META-ANALYSIS

Why Do the Colchicine Trials Disagree?



Different populations. Different endpoints. Different contexts. The results are more consistent than they first appear.

DIFFERENT PIECES. A CLEARER PICTURE.

SELECTED COLCHICINE TRIALS

Trial	Population	Colchicine Dose	Primary Endpoint	Key Result
LoDoCo	Stable CAD	0.5 mg daily	ACS, out-of-hospital cardiac arrest, or non-cardiometabolic stroke	HR 0.33 (0.18–0.59)
COLCOT	Recent MI	0.5 mg daily	CV death, resuscitated cardiac arrest, MI, stroke, or urgent revascularization	5.5% vs 71%; HR 0.77 (0.61–0.96)
LoDoCo2	Stable CAD	0.5 mg daily	CV death, spontaneous MI, ischemic stroke, or ischemia-driven revascularization	6.8% vs 9.6%; HR 0.69 (0.57–0.83)
COPS	ACS	0.5 mg twice daily (1 month); → 0.5 mg daily	ACS, urgent revascularization, stroke, or death	n=795: 24 vs 38 primary events; neutral / imprecise
CLEAR SYNERGY	Post-MI	0.5 mg daily	CV death, MI, stroke, or ischemia-driven revascularization	9.1% vs 9.3%; HR 0.99 (0.85–1.16)
CHANCE-3	Recent stroke/TIA	0.5 mg daily	Recurrent vascular events	6.3% vs 6.5%; HR 0.98 (0.83–1.16)
CONVINCE	Recent stroke/TIA	0.5 mg daily	Recurrent vascular events	HR ~0.84 (0.68–1.05); inconclusive

KEY REASONS FOR DIFFERENT RESULTS

01
Patient population
Recent MI, Stable CAO, ACS, post-MI, or stroke/TIA populations.

02
Endpoint definitions
Not all trials used the same composite outcomes.

03
Dose and duration
Most used 0.5 mg daily, but treatment duration and early dosing strategies differed.

04
Background therapy and era
Differences in statins, revascularization, and contemporary medical therapy may influence outcomes.

05
Event rates and statistical power
Lower event rates in some trials make modest treatment effects harder to detect.

THE MEDEASY TAKE

Different trials, one broader story.

Across randomized trials, colchicine shows a modest overall reduction in recurrent cardiovascular claerest signal in established CAD. Results are less consistent in contemporary post-MI and cerebrovascular settings.

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EVIDENCE SYNTHESIS + META-ANALYSIS

How Much Can We Trust the Evidence?



The colchicine evidence comes from randomized trials of varying size and context. Differences in populations, endpoints, follow-up, and event rates help explain why individual trial results diverge—and how much weight each result should carry in practice.

TRIAL QUALITY AT A GLANCE

Most major randomized colchicine trials are methodologically sound, but differences in design, population, and endpoint selection affect how confidently results can be applied.

Low risk · Some concerns · High risk

Trial	Population	Primary Endpoint	Follow-up	Risk of Bias	Overall Confidence
LoDoCo	Stable CAD	ACS, out-of-hospital cardiac arrest, or non-cardioembolic stroke	3 years	Some concerns	Moderate
COLCOT	Recent MI	CV death, resuscitated cardiac arrest, MI, stroke, or urgent revascularization	22.6 months	Low risk	High
LoDoCo2	Stable CAD	CV death, spontaneous MI, ischemic stroke, or ischemia-driven revascularization	28.6 months	Low risk	High
COPS	ACS	ACS, urgent revascularization, stroke, or death	12 months	Low risk	Moderate
CLEAR SYNERGY	Post-MI	CV death, MI, stroke, or ischemia-driven revascularization	3 years	Low risk	High
CHANCE-3	Recent stroke/TIA	Any new stroke	90 days	Low risk	High
CONVINCE	Recent stroke/TIA	Recurrent ischemic stroke, MI, cardiac arrest, or hospitalization for unstable angina	3 years	Low risk	Moderate

KEY TAKEAWAY

The evidence base is strong—but not uniform.

Most major colchicine trials are methodologically sound, with the clearest and most consistent signal seen in established CAD. Neutral or inconclusive findings in some post-MI and cerebrovascular settings likely reflect differences in population, endpoint choice, follow-up, and event rates rather than a single simple answer.

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CLINICAL INTERPRETATION

Where Does Colchicine Fit in Practice?



The evidence supports a role for colchicine in selected patients, while a more cautious approach is reasonable in others.

Who to Consider?

STRONGER EVIDENCE

Established Stable CAD

The clearest and most consistent benefit has been observed in established coronary disease (e.g., LoDoCo, LoDoCo2).

Selected Post-MI Patients

COLCOT supports benefit after MI, but more contemporary evidence is less consistent.

Patients With Recurrent Atherosclerotic Risk

Consider only after weighing residual cardiovascular risk, tolerability, comorbidity, and competing treatment priorities.

Where to Be More Cautious?

LIMITED OR UNCERTAIN EVIDENCE

Contemporary Post-MI Settings

CLEAR SYNERGY showed no significant reduction in major cardiovascular events.

Cerebrovascular Disease

CHANCE-3 was neutral and CONVINCE remained inconclusive.

Lower-Risk Patients

Absolute benefit may be small when baseline cardiovascular risk is low.

Patients With Safety or Interaction Concerns

Use caution with renal impairment, gastrointestinal intolerance, and clinically important drug interactions.

PRACTICAL CONSIDERATIONS

MAKE IT WORK IN REAL-WORLD PRACTICE

Dose

Most trials used 0.5 mg daily. Some protocols used higher initial doses or different durations depending on clinical context.

Duration

Benefits were observed in both shorter-term and longer-term studies, but duration should match the clinical setting and treatment tolerance.

Safety

Generally well tolerated. Review renal function and potential drug interactions, particularly strong CYP3A4 or P-gp inhibitors; monitor for gastrointestinal and muscle-related adverse effects.

Patient Discussion

Set realistic expectations. Explain potential benefits, uncertainty in some populations, and the importance of adherence and follow-up.

KEY TAKEAWAY

Use colchicine where the evidence is strongest, and individualize in areas of uncertainty.

The totality of randomized evidence supports selective use of low-dose colchicine, with the clearest benefit in established CAD. Treatment should remain individualized rather than applied routinely across all ASCVD populations.

STUDY UNDER THE MICROSCOPE

SINGLE-AF
Do we anticoagulate too late?

A randomized trial in an intermediate-risk AF population.

> *Intermediate-risk is not no risk. The question is not if risk exists, but whether we can reduce it — and improve outcomes for the right patients.*

BETTER TIMING FOR A BRIGHTER TOMORROW

In atrial fibrillation, risk exists on a continuum. SINGLE-AF examines whether anticoagulation can improve outcomes in patients who are at intermediate risk of stroke.



SAME QUESTIONS A BRIGHTER TOMORROW.

TRIAL AT A GLANCE

1,803 patients
with non-valvular AF and intermediate stroke risk

Randomized
Open-label

24 months
median follow-up

Intermediate-risk
atrial fibrillation

DOAC
vs
No anticoagulation

PRIMARY COMPOSITE ENDPOINT

- Stroke
- Systemic embolism
- Major bleeding
- Cardiovascular death

KEY QUESTION

Risk scores create categories, but patients live on a continuum of thromboembolic and bleeding risk. SINGLE-AF tests whether some intermediate-risk patients may still derive net clinical benefit from anticoagulation.

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SINGLE-AF • THE RESULT

ANTICOAGULATION REDUCED THE PRIMARY COMPOSITE ENDPOINT

The result is striking in relative terms — but modest in absolute terms.

PRIMARY COMPOSITE

Stroke • systemic embolism • major bleeding • cardiovascular death

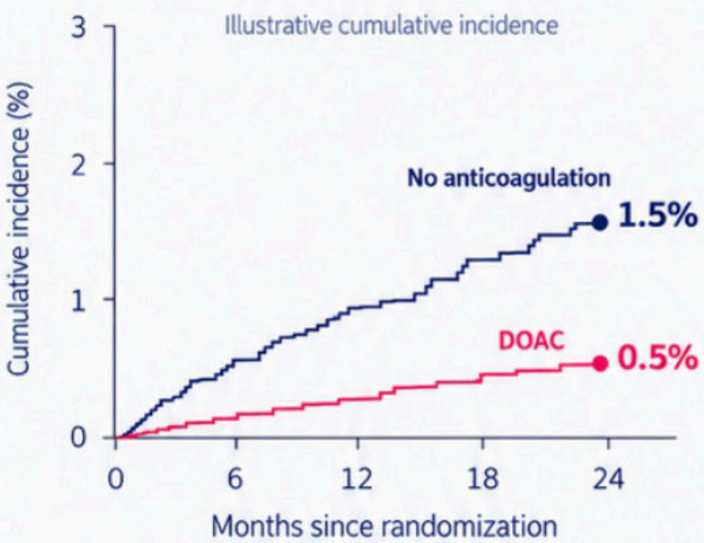
0.5% DOAC

VS

1.5% NO ANTICOAGULATION

HR 0.31
(95% CI 0.10–0.94)
p = 0.028

CUMULATIVE INCIDENCE



READ THE RESULT TWO WAYS

RELATIVE EFFECT

69%
lower hazard
for the primary composite endpoint

ABSOLUTE EFFECT

~1 pp
absolute separation
over 24 months

SELECTED COMPONENTS

ISCHEMIC STROKE

0.1% DOAC VS **1.1%** No anticoagulation

MAJOR BLEEDING

0.3% DOAC VS **0.5%** No anticoagulation

MEDEASY TAKE

A strong relative effect does not mean a large absolute benefit.

The result supports individualized net-clinical-benefit assessment, not automatic anticoagulation for every intermediate-risk patient.

? KEY QUESTION | In patients with intermediate-risk AF, does oral anticoagulation reduce the primary composite endpoint compared with no anticoagulation?

SINGLE-AF TRIAL

A Closer Look at SINGLE-AF



SAME QUESTIONS. BETTER ANSWERS.

Important strengths, but also key limitations to consider when applying the results in practice.

Why Take It Seriously?

REAL AND IMPORTANT SIGNALS

Large, randomized trial
1,803 patients with intermediate-risk AF were randomized to DOAC or no anticoagulation.

Clinically meaningful signal
The primary composite was 0.5% with DOAC versus 1.5% with no anticoagulation (HR 0.31).

Selected component signals
Ischemic stroke was lower with DOAC, while major bleeding remained uncommon in both groups.

Clinically relevant question
Directly tested anticoagulation versus no anticoagulation in an intermediate-risk AF population.

Why Stay Cautious?

IMPORTANT LIMITATIONS

Open-label design
May introduce bias in event reporting and management.

South Korean population
Conducted in South Korea; generalizability to other populations remains uncertain.

Relatively few events
Few outcome events produced a wide confidence interval around the treatment effect.

Composite endpoint
Combined thrombotic and bleeding events, so the overall result may not reflect each component equally.

Not powered for subgroups
Benefit in specific patient subgroups remains uncertain.

THE MEDEASY TAKE

A meaningful signal—but not a one-size-fits-all answer.

SINGLE-AF challenges the assumption that intermediate-risk AF automatically means no anticoagulation. The findings support individualized net-clinical-benefit assessment, but do not justify anticoagulating every patient.

KEY QUESTION

Which intermediate-risk AF patients are most likely to benefit, and how should we individualize the decision?

HYPERTENSION

BaxHTN
The Aldosterone Comeback

Baxdrostat, a next-generation aldosterone synthase inhibitor, lowers blood pressure in treatment-resistant hypertension.



TARGETING EXCESS ALDOSTERONE FOR BETTER BP CONTROL.

KEY RESULT

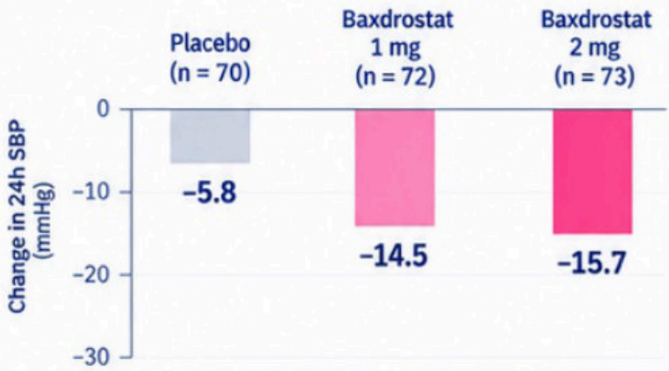
−9.8 mmHg

Greater reduction in systolic blood pressure versus placebo at 12 weeks with baxdrostat 2 mg.

Mean change: −15.7 mmHg vs −5.8 mmHg
Placebo-corrected difference: −9.8 mmHg

12-WEEK SBP CHANGE

Greater BP reduction versus placebo



Mean change in systolic BP from baseline at 12 weeks.

HOW IT WORKS



Baxdrostat inhibits aldosterone synthase (CYP11B2)

↓

Reduced aldosterone production

↓

Reduced sodium retention

↓

Lower blood pressure

KEY SAFETY FINDINGS

Hyperkalemia
Potassium >6 mmol/L occurred in 2.3% with baxdrostat 1 mg, 3.0% with 2 mg, and 0.4% with placebo.

Renal function
No major deterioration in renal function was observed over 12 weeks.

Other adverse events
Overall adverse-event rates were broadly similar across treatment groups.

Ongoing data
Longer-term safety and cardiovascular outcomes remain under evaluation.

WHY IT MATTERS

Addresses an unmet need
Many patients with resistant hypertension remain uncontrolled despite multidrug therapy.

A novel mechanism
Directly inhibits aldosterone synthase, reducing aldosterone production at the source.

Potential to expand options
May offer an additional treatment option for resistant hypertension if longer-term efficacy and safety are confirmed.

THE MEDEASY TAKE

Baxdrostat produced clinically meaningful blood-pressure reduction in resistant hypertension, with greater BP lowering than placebo and an expected potassium-safety signal.

It represents a promising new therapeutic approach, but longer-term safety, cardiovascular outcomes, and comparative effectiveness remain important unanswered questions.

HYPERTENSION

Where Does Baxdrostat Fit?



Positioning, patient selection, and practical considerations for aldosterone synthase inhibition in resistant hypertension.

MORE OPTIONS FOR BETTER BP CONTROL

POTENTIAL PLACE IN THERAPY

01 Resistant hypertension
Patients with uncontrolled blood pressure despite optimized multidrug therapy.

02 Aldosterone-driven phenotype
May be particularly relevant where aldosterone excess is suspected, although validated selection biomarkers are not yet established.

03 Potential future alternative pathway
May become relevant when additional aldosterone-targeted options are needed, but comparative effectiveness versus MRas remains unproven.

04 Potential add-on role
Could complement standard therapy in selected patients if longer-term efficacy and safety are confirmed.

HOW IT COMPARES

Feature	Baxdrostat (ASI)	Spironolactone (MRA)
Target	Aldosterone synthase (CYP11B2)	Mineralocorticoid receptor
Site of action	Upstream — reduces aldosterone production	Downstream — blocks aldosterone receptor signaling
Evidence base	Emerging phase 3 evidence in resistant hypertension	Extensive established evidence in resistant hypertension
Current clinical role	Investigational / emerging option	Established guideline-supported MRA
Key safety consideration	Potassium elevation; longer-term safety still being defined	Hyperkalemia and endocrine adverse effects

Note: Indirect comparison only. Head-to-head comparative efficacy and safety remain unknown.

PRACTICAL CONSIDERATIONS

Patient selection
Focus on resistant hypertension and consider features suggesting aldosterone-driven disease, while recognizing that validated predictive biomarkers are not yet established.

Monitoring
Monitor serum potassium and renal function during therapy.

Dosing
Trial doses included 1 mg and 2 mg daily. Optimal long-term dosing and titration strategies remain to be defined.

KEY UNANSWERED QUESTIONS

Long-term outcomes
Effects on cardiovascular events, mortality, and long-term safety remain under study.

Which patients benefit most?
Better biomarkers or clinical predictors of treatment response are still needed.

Combination strategies
The role of baxdrostat with MRAs and other antihypertensive therapies requires further evaluation.

THE MEDEASY TAKE

Baxdrostat may expand future options for resistant hypertension.

It targets aldosterone production directly and produced meaningful blood-pressure reduction in BaxHTN. Its eventual place in therapy will depend on longer-term safety, cardiovascular outcomes, and direct comparison with established treatments.

KEY QUESTION

Which patients with resistant hypertension **are most likely to benefit from baxdrostat,** and how should it be integrated into future practice?

HYPERTENSION

Key Takeaways and Clinical Pearls

What to remember from BaxHTN and how to apply it in everyday practice.



SMALL CHANGES, BIG IMPACT ON BP.

5 KEY TAKEAWAYS

1. **Meaningful BP reduction**
   Baxdrostat 2 mg produced a placebo-corrected systolic BP reduction of 9.8 mmHg at 12 weeks.
2. **Targets the source**
   Directly inhibits aldosterone synthase (CYP11B2), reducing aldosterone production upstream.
3. **Potential role in resistant hypertension**
   May expand future treatment options for patients with uncontrolled blood pressure despite optimized multidrug therapy.
4. **Potassium requires monitoring**
   Potassium >6 mmol/L occurred in 2.3% with 1 mg, 3.0% with 2 mg, and 0.4% with placebo; renal function was generally stable over 12 weeks.
5. **Expands future possibilities**
   May complement existing therapies, but optimal patient selection and comparative effectiveness remain uncertain.

PRACTICAL CLINICAL PEARLS

Consider an aldosterone-driven phenotype
Clinical features may suggest aldosterone excess, but validated biomarkers for selecting baxdrostat responders are not yet established.

Monitor potassium and renal function
Check serum potassium and renal function during therapy, with closer monitoring when clinically indicated.

Use trial-tested doses cautiously
BaxHTN evaluated 1 mg and 2 mg daily. Optimal long-term dosing and titration strategies remain to be defined.

Individualize therapy
Future use should be guided by resistant-hypertension phenotype, comorbidities, concomitant medications, tolerability, and patient preferences.

CURRENT LIMITATIONS

Short-term data
Blood-pressure efficacy and safety are based primarily on 12-week results; longer-term cardiovascular outcomes remain uncertain.

Selected population
BaxHTN enrolled patients with resistant hypertension, so generalizability to broader hypertension populations is uncertain.

Comparative data needed
Head-to-head trials with MRAs and other established therapies are needed to define baxdrostat's relative place in therapy.

LOOKING AHEAD

Longer-term studies and follow-up will clarify cardiovascular outcomes, durability of blood-pressure lowering, safety, and which patients are most likely to benefit.

THE MEDEASY TAKE

Baxdrostat is a promising emerging approach to resistant hypertension—targeting aldosterone production at the source and producing meaningful blood-pressure reduction.

Its long-term safety, cardiovascular effects, optimal patient selection, and comparative effectiveness versus established therapies still need to be defined.

CARDIAC AMYLOIDOSIS

Target Engagement Is Not Enough

The CARDIO-TTRansform trial: eplontersen reduces transthyretin, but does not significantly improve clinical outcomes in ATTR-CM.



LOWER TTR DOESN'T ALWAYS MEAN BETTER OUTCOMES.

THE TRIAL

CARDIO-TTRansform
Phase 3, randomized, double-blind, placebo-controlled.

Population
1,432 patients with transthyretin amyloid cardiomyopathy (ATTR-CM).

Intervention
Eplontersen 45 mg subcutaneously monthly vs placebo, on top of standard care.

Background therapy
Approximately 57% were receiving a TTR stabilizer at baseline.

Follow-up
Up to 140 weeks.

Primary outcome
Cardiovascular death + recurrent cardiovascular events.

KEY RESULTS

Primary outcome event rate

Eplontersen (n = 709)	Placebo (n = 723)
29.4%	**32.2%**

Rate ratio 0.89
(95% CI 0.73 – 1.09)
p = 0.28

Cardiovascular deaths:
74 vs 70

Background TTR stabilizer
~57%
of participants were receiving a TTR stabilizer at baseline.

SAFETY

Serious adverse events
57.8% vs **59.4%** with eplontersen and placebo, respectively.

Injection-site reactions
More frequent with eplontersen; generally mild to moderate.

Liver-related events
Low and broadly similar between groups.

Treatment discontinuation
Overall discontinuation was similar between groups.

WHAT THIS MEANS

Marked TTR reduction
Eplontersen substantially lowered transthyretin levels.

No significant clinical benefit
The primary cardiovascular outcome was not significantly reduced despite strong target engagement.

Background therapy matters
About 57% of participants were already receiving a TTR stabilizer at baseline.

WHY TARGET ENGAGEMENT MAY NOT TRANSLATE

Clinical disease may be harder to reverse
Reducing TTR does not necessarily reverse established cardiac injury.

Background therapy matters
Concurrent TTR-stabilizer use may influence the incremental clinical effect of an additional TTR-lowering strategy.

Clinical outcomes remain the test
Biological target engagement must ultimately translate into meaningful patient outcomes.

THE MEDEASY TAKE

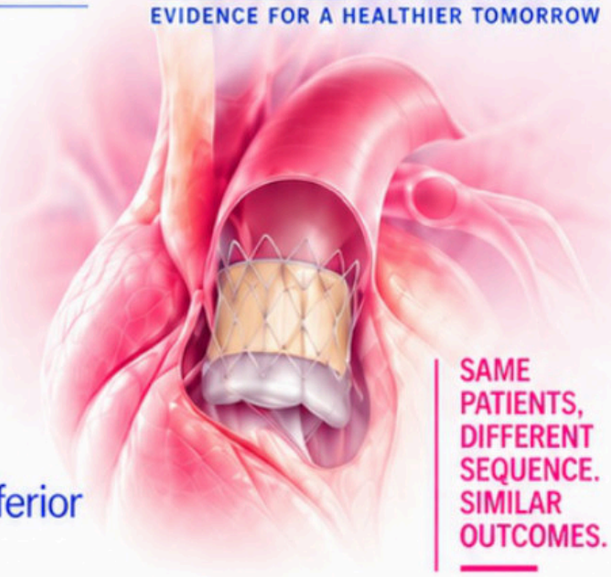
Eplontersen substantially lowers transthyretin, but did not significantly reduce cardiovascular death and recurrent cardiovascular events in CARDIO-TTRansform.

The trial reinforces a central lesson in therapeutics: target engagement does not guarantee clinical benefit.

STRUCTURAL HEART DISEASE

TAVI First or PCI First?

In patients with severe aortic stenosis and coronary artery disease, TAVI-first was noninferior to PCI-first for 1-year outcomes.



SAME PATIENTS, DIFFERENT SEQUENCE. SIMILAR OUTCOMES.

THE TRIAL

Population
986 patients with severe aortic stenosis and significant coronary artery disease.

Randomization
TAVI-first (n = 498) vs PCI-first (n = 488).

Follow-up
1 year.

Primary endpoint
Composite of all-cause death, nonfatal myocardial infarction, ischemia-driven revascularization, valve-, procedure-, or heart-failure-related rehospitalization, and life-threatening, disabling, or major bleeding.

KEY RESULTS

Primary endpoint at 1 year

22.2%	24.2%
TAVI-first (n = 498)	PCI-first (n = 488)

Risk difference
−2.0 percentage points
(95% CI −7.4 to 3.4)

p < 0.001 for noninferiority

SAFETY

Serious adverse events
Overall serious adverse events were similar between groups.

No clear procedural penalty
TAVI-first did not show a clinically important excess in major procedural complications.

Sequence remained flexible
The trial supports more than one acceptable sequencing strategy rather than a single mandatory approach.

CLINICAL IMPLICATIONS

Individualize the sequence
Choose TAVI-first or PCI-first according to coronary anatomy, ischemic burden, clinical stability, anticipated future coronary access, and Heart Team assessment.

Optimize medical therapy
Guideline-directed medical therapy remains essential regardless of procedural sequence.

KEY CONSIDERATIONS

Noninferiority is not superiority
TAVI-first was noninferior to PCI-first, but the trial did not establish that TAVI-first is better.

Patient selection matters
Results may not apply equally to every anatomy or clinical scenario, particularly complex coronary disease or situations where future coronary access is a major concern.

LOOKING AHEAD

Refining patient selection
Further work should identify which patients benefit most from a TAVI-first versus PCI-first sequence.

Longer-term outcomes
Longer follow-up will help clarify durability, coronary access, repeat revascularization, and late clinical outcomes.

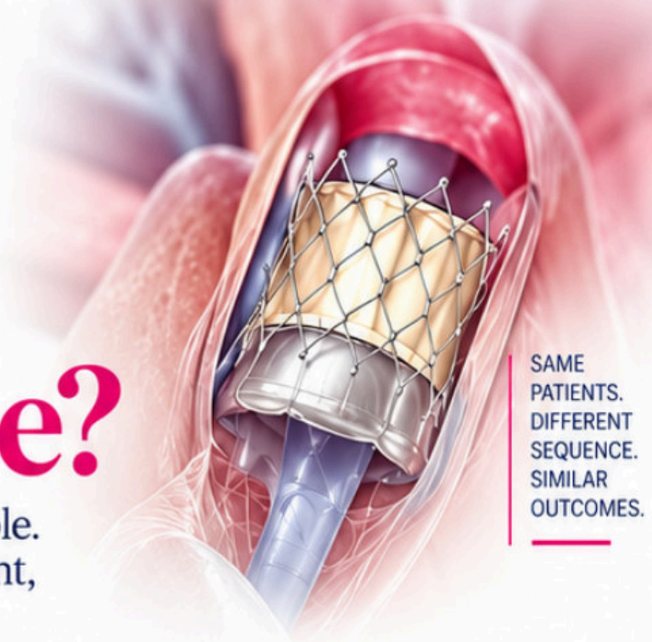
THE MEDEASY TAKE

In patients with severe aortic stenosis and coronary artery disease, a TAVI-first strategy was **noninferior** to PCI-first for 1-year outcomes. The findings support flexible, individualized sequencing based on coronary anatomy, ischemia, clinical stability, future coronary access, and Heart Team assessment—not routine preference for one strategy in every patient.

STRUCTURAL HEART DISEASE

How Should We Choose the Sequence?

Both TAVI-first and PCI-first are reasonable. The right sequence depends on the patient, not a one-size-fits-all approach.



SAME PATIENTS. DIFFERENT SEQUENCE. SIMILAR OUTCOMES.

A QUICK TRIAL REMINDER

Population
986 patients with severe aortic stenosis and significant coronary artery disease.

Comparison
TAVI-first (n = 498) vs PCI-first (n = 488).

Primary Endpoint at 1 Year

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TAVI-first (n = 498)	PCI-first (n = 488)

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KEY CONSIDERATIONS IN PRACTICE

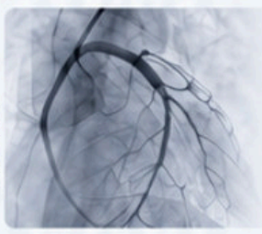
INDIVIDUALIZE. DISCUSS. PLAN TOGETHER.

01 Valve severity and clinical status
Consider symptom burden, hemodynamic stability, and urgency for valve intervention.



Severe AS often drives the timing.

02 Coronary anatomy and ischemic burden
Assess lesion complexity, ischemic significance, technical feasibility of PCI, and how the coronary strategy may affect procedural sequencing.



Complex CAD may favor PCI-first in selected patients.

03 Patient and procedural factors
Consider age, comorbidities, surgical risk, anticipated future coronary access, life expectancy, local expertise, and Heart Team assessment.



Heart Team assessment remains essential.

✓ WHAT THE RESULTS ALLOW US TO SAY

In patients with severe aortic stenosis and coronary artery disease, a TAVI-first strategy was noninferior to PCI-first for 1-year outcomes, supporting flexibility in sequencing based on coronary anatomy, ischemic burden, clinical stability, anticipated future coronary access, and Heart Team assessment.

✕ WHAT THE RESULTS DO NOT MEAN

The trial does not prove that TAVI-first is superior, nor that it should be used in all patients. Results may not apply equally to complex coronary anatomy, specific clinical scenarios, or situations where future coronary access is a major concern.



THE MEDEASY TAKE

The best sequence is the one that fits the patient.

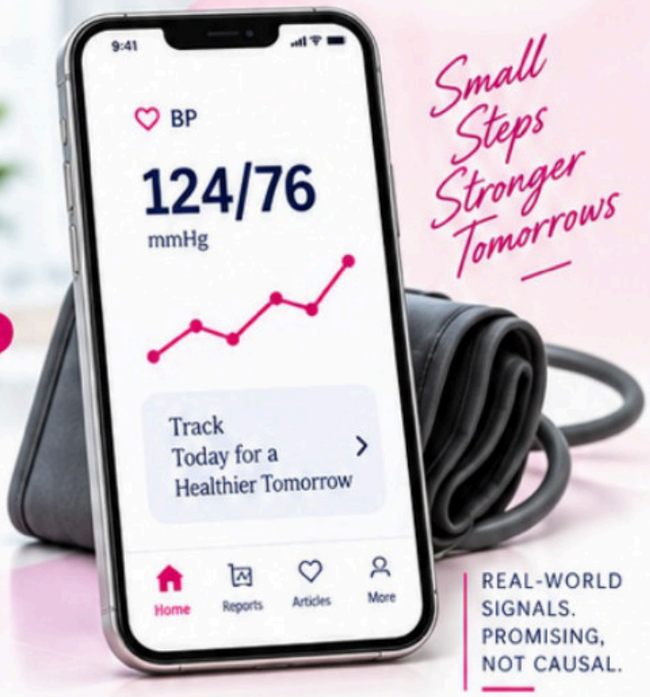
Use a Heart Team approach to individualize sequencing according to valve severity, coronary anatomy, ischemic burden, clinical stability, anticipated future coronary access, and patient-specific factors.

medeasy REVIEW

DIGITAL MEDICINE

Can an App Really Lower Blood Pressure?

Real-world data suggest meaningful blood-pressure improvement among users of a digital hypertension platform — but observational evidence cannot prove causation.



REAL-WORLD SIGNALS. PROMISING, NOT CAUSAL.

WHAT WAS OBSERVED

22,901 USERS

−9.5 mmHg SBP

−6.3 mmHg DBP

34.4% → 51.6% reported BP control
Month 1 → Month 12

What Makes This Interesting

01 THE MAGNITUDE

The observed blood-pressure reductions were clinically notable, but causality cannot be inferred from an observational analysis.

02 THE ENGAGEMENT

The platform may support self-monitoring, reminders, education, and engagement between visits, but the analysis cannot isolate which components drove the observed change.

03 THE LIMITATION

Users were not randomized, and treatment changes, selection effects, regression to the mean, and engagement may explain part of the observed improvement.

THE MEDEASY TAKE

Digital tools may support blood-pressure improvement — but observational results cannot establish causation.

Use them to extend care, not replace clinical judgment or validated treatment pathways.

BETTER QUESTIONS BRIGHTER CARE

Observational real-world analysis • 22,901 users • 12 months • user-reported data
Funding disclosed: Servier Affaires Médicales.

medeasy REVIEW

DIGITAL MEDICINE

Beyond the Download

What Makes Digital Health Worth Using?

The most useful tools do more than collect blood-pressure readings. They support accurate measurement, sustained engagement, and better clinical decisions.



THREE PRINCIPLES FOR REAL-WORLD IMPACT

01

MEASUREMENT QUALITY

Reliable home BP begins with validated devices, correct cuff size, proper technique, and consistent timing. Good data is the foundation of good decisions.

> *Bad data cannot become good care.*

02

SUSTAINED ENGAGEMENT

The tool has to remain useful after the novelty wears off. Simple reminders, clear feedback, and low-friction workflows help patients keep tracking and stay involved.

> *Simple workflows beat feature overload.*

03

CLINICAL INTEGRATION

A digital tool should help patients and clinicians review trends together, recognize patterns, and act on what matters. Data without action is just more data.

> *Technology should extend clinical judgment, not compete with it.*

WHAT GOOD DIGITAL CARE LOOKS LIKE

- Reliable measurements
- Consistent use
- Actionable trends
- Shared decision-making

THE MEDEASY TAKE

The best digital tool is not the one with the most features – it is the one that improves real care.

Technology should extend clinical judgment, not replace it.

BETTER QUESTIONS BRIGHTER CARE

PRACTICAL INSIGHTS

Monday Morning Medicine

Eight practical lessons to carry into the week ahead.

A quick round-up of practical insights from this issue — so you can turn evidence into smarter everyday decisions for your patients.



01 Don't Stop at Positive or Negative

A trial result is only the start. Look at effect size, confidence intervals, event rates, endpoint design, and clinical context.

02 Colchicine Is Not a Universal ASCVD Drug

The overall signal is modest, with the clearest benefit in established CAD and less consistent results in contemporary post-MI and cerebrovascular settings.

03 Intermediate-Risk AF Is Still Risk

SINGLE-AF challenges the assumption that intermediate-risk AF automatically means no anticoagulation—but treatment still requires individualized net-clinical-benefit assessment.

04 Aldosterone Is Back in the Conversation

Baxdrostat produced meaningful blood-pressure lowering in resistant hypertension, while potassium monitoring and longer-term evidence remain important.

05 Target Engagement ≠ Clinical Benefit

CARDIO-TTRansform reminds us that lowering a biological target does not guarantee better patient outcomes.

06 TAVI vs PCI Sequence Should Be Individualized

Noninferiority supports flexibility, not superiority. Coronary anatomy, ischemia, stability, future coronary access, and Heart Team assessment still matter.

07 Digital Health Needs Clinical Standards

Useful tools need reliable measurements, sustained engagement, clinical integration, and evidence that goes beyond observational association.

08 Ask Whether the Evidence Applies to Your Patient

Population, baseline risk, comorbidity, treatment context, and patient preferences determine how evidence should influence practice.

MEDEASY TAKEAWAY

Better decisions begin with better interpretation.

Read beyond the headline, understand the context, and apply the evidence to the patient in front of you.

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FOR A HEALTHIER TOMORROW

EVIDENCE SNAPSHOT

Four Studies. Real Impact.

Key findings. Clearer context. Practical takeaways.

A quick look at four recent studies spanning rhythm management, stroke prevention, coronary assessment, and the evolving role of AI in medicine.



01

CARDIOLOGY · ATRIAL FIBBILLATION

AVANT-GUARD

PFA First vs Antiarrhythmic Drug



In patients with persistent atrial fibrillation, pulsed field ablation (PFA) was compared with antiarrhythmic drug therapy as an initial rhythm-control strategy. 310 randomized: 207 PFA / 103 AAD

Key Result

56% vs 30%

Freedom from treatment failure

HR 0.46 (95% CI 0.33–0.65)
p < 0.001

Takeaway

- PFA improved rhythm-control efficacy versus AAD, but neurological safety events remain an important part of interpretation.
- Neurological events prompted a pause and workflow modification.

02

CARDIOLOGY · STROKE PREVENTION

CHAMPION-AF

Left Atrial Appendage Closure vs NOAC



In 3,000 patients with non-valvular atrial fibrillation at increased stroke risk, left atrial appendage closure was compared with continued NOAC therapy.

Key Result

5.7% vs 4.8%

Primary composite outcome at 3 years
Met noninferiority criteria

Nonprocedural bleeding: 10.9% vs 19.0%; HR 0.55
Ischemic stroke: 3.2% vs 2.0%

Takeaway

- LAAC met noninferiority for the primary composite and reduced nonprocedural bleeding, but ischemic stroke was numerically higher.

03

CARDIOLOGY · CORONARY IMAGING

FUSION

FFRct and Invasive Angiography Without Obstructive CAD



In 528 patients with suspected coronary artery disease, an FFRct-guided strategy was compared with standard care.

Key Result

18% vs 33%

Invasive angiography without obstructive CAD

OR 0.46 (p < 0.001)

Takeaway

- FFRct reduced invasive angiography without obstructive CAD, supporting a more selective diagnostic strategy.

04

HEALTHCARE · ARTIFICIAL INTELLIGENCE

AI in Medicine

From "Can AI Do It?" to "Does It Improve Decisions?"



Recent studies and real-world evaluations are moving beyond technical performance to ask whether AI meaningfully improves clinical decisions and patient outcomes.

Key Message

The focus is shifting from "Can AI do it?" to "Does AI improve decisions and outcomes?"

Takeaway

- AI shows greatest value when integrated into clinical workflows, with human oversight.
- The real goal is not just accurate answers, but better, safer, and more informed care.

MEDEASY TAKEAWAY

Evidence Today. Better Care Tomorrow.

Different questions.
One goal — better decisions for healthier patients.

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MEDEASY PICKS

Use Medeasy. Make Everyday Practice More Rewarding.



Earn Medeasy Points through eligible activities across the app — while keeping clinical decisions exactly where they belong: with you and your patient.

EXPLORE • EARN • STAY CONNECTED

HOW IT WORKS

01

USE

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02

EARN

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03

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THE PRINCIPLE

Clinical decisions remain based solely on professional judgment and patient needs.

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Before You Go...

A few final reminders.

Better Clinicians Brighter Tomorrows.

CLINICAL
TRUSTED
INDEPENDENT
IMPACTFUL

This issue brought together practical lessons from cardiometabolic prevention, rhythm care, hypertension, structural heart disease, and digital medicine. The goal is simple: help clinicians see the signal, interpret it wisely, and bring what matters back to practice.

01

Read beyond the headline.

Look past the abstract and ask what the trial truly means for patients in front of you.

DEEPER UNDERSTANDING.

02

Absolute benefit matters.

Relative reductions are useful, but treatment decisions depend on baseline risk and real clinical context.

REAL-WORLD PERSPECTIVE.

03

Fit the evidence to the patient.

Population, safety, comorbidities, and preferences still shape whether evidence belongs in practice.

PATIENTS AT THE CENTER.

04

Keep the conversation going.

Good medicine evolves through continued learning, reflection, and thoughtful application.

A STRONGER TOMORROW.

> **Stay curious.**
> **Stay updated.**
> **Keep caring.**
>
> — Medeasy Review Team



Same Commitment. Brighter Care.

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Together for a healthier tomorrow.