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Therapy After Percutaneous Coronary Intervention in Chronic Coronary Disease: A Practical Review for Internal Medicine and Interventional Cardiology

Akramov Bekhzod Bakhromjon o'gli
Assistant, Department of Internal Diseases, Nephrology and Hemodialysis, Tashkent State Medical University, Tashkent, Uzbekistan
Research Applicant at The Republican Specialized Scientific and Practical Medical Center of Nephrology and Kidney Transplantation, Uzbekistan

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Abstract

Percutaneous coronary intervention (PCI) is a central procedure in modern cardiology, but the long-term prognosis of patients with chronic coronary disease is determined not only by mechanical revascularization. Durable benefit after PCI depends on the systematic control of atherosclerotic risk, correct selection and duration of antiplatelet therapy, intensive lipid lowering, blood pressure and glycemic optimization, antianginal treatment, rehabilitation, and patient adherence. Recent European and American recommendations increasingly frame coronary artery disease as a chronic, multidisciplinary condition that requires coordinated follow-up after the catheterization laboratory. This review summarizes current evidence and provides a practical outpatient algorithm for therapy departments and interventional cardiology services managing adults after PCI. The article focuses on five clinically important domains: antithrombotic therapy, lipid-lowering escalation, antianginal and hemodynamic treatment, cardiometabolic risk modification, and structured post-discharge follow-up. A stepwise checklist is proposed for visits at 7-14 days, 1-3 months, and 6-12 months after PCI. The practical aim is to reduce fragmentation of care, improve documentation of treatment goals, and help physicians translate guideline recommendations into everyday practice. The proposed approach is particularly relevant for patients with diabetes, hypertension, chronic kidney disease, obesity, previous myocardial infarction, multivessel coronary disease, and high bleeding risk, in whom a single uniform post-PCI prescription is insufficient.

Keywords: Chronic coronary disease; chronic coronary syndrome; percutaneous coronary intervention; interventional cardiology; therapy; dual antiplatelet therapy; LDL-C; secondary prevention; outpatient follow-up; guideline-directed medical therapy.

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1. Introduction

Percutaneous coronary intervention has changed the clinical course of obstructive coronary artery disease by rapidly relieving ischemia, improving symptoms, and reducing acute complications in appropriately selected patients. However, successful stent implantation is only one component of cardiovascular care. A patient who leaves the catheterization laboratory with an optimal angiographic

result may still be exposed to recurrent myocardial infarction, stent thrombosis, progressive non-culprit plaque disease, heart failure, uncontrolled angina, bleeding complications, or poor quality of life if long-term pharmacological and lifestyle management is weak.

This point is especially important for physicians working in internal medicine and therapy departments. After discharge, many post-PCI patients return not to the interventional cardiologist but to the therapist, family physician, endocrinologist, nephrologist, or outpatient cardiologist. The procedural report may mention the treated artery, the stent type, and the recommended antiplatelet plan, but the long-term risk reduction strategy often requires multiple adjustments over several months. Lipid targets must be reached, blood pressure must be controlled without hypotension, diabetes and obesity treatment should be selected with cardiovascular benefit in mind, angina must be reassessed after revascularization, and the bleeding risk should be weighed against ischemic risk rather than treated as a fixed category.

Recent recommendations from the European Society of Cardiology and the American Heart Association/American College of Cardiology emphasize that chronic coronary disease is not simply a stable condition after an acute episode. It is a continuing systemic atherosclerotic process. In this framework, PCI should be integrated with guideline-directed medical therapy rather than viewed as an alternative to it. The most practical question for everyday medicine is therefore not whether the patient underwent PCI, but whether post-PCI therapy is complete, understandable, documented, tolerated, and periodically re-evaluated.

The present article offers a publication-ready practical review at the interface of therapy and interventional cardiology. It does not report patient-level outcomes. Instead, it translates contemporary guideline principles into an outpatient pathway that can be used for clinical practice, teaching, and future real-world audits. The review is written for physicians who need a structured approach to patients after PCI, particularly in settings where specialist visits, laboratory monitoring, medication access, and patient adherence may vary.

Aim of the article

The aim of this review is to summarize current principles of medical therapy after PCI in chronic coronary disease and to propose a structured outpatient management algorithm applicable to internal medicine, therapy, and interventional cardiology practice. The specific objectives are to define key post-PCI therapeutic priorities, describe individualized antithrombotic and lipid-lowering strategies, clarify the role of cardiometabolic treatment, and provide a follow-up checklist that can be implemented in routine outpatient care.

2. Methods and Scope of The Review

This manuscript is a narrative clinical review with practical recommendations. The core sources were contemporary guideline documents and major randomized or practice-changing studies relevant to chronic coronary disease, PCI, secondary prevention, lipid lowering, antithrombotic therapy, diabetes and obesity pharmacotherapy, and cardiac rehabilitation. The review gives priority to recommendations published by the European Society of Cardiology, the American Heart Association, the American College of Cardiology, and other major cardiovascular societies. Randomized trials and high-quality reviews were used to support practical statements where appropriate.

The article is not a systematic review and does not use meta-analytic methods. No individual patient data were collected, analyzed, or reproduced. Therefore, ethics committee approval and informed consent are not required for the manuscript in its present form. If the proposed pathway is later evaluated in real patients as a clinical audit or observational study, local ethical review, anonymization, and institutional permission should be obtained according to national and journal requirements.

Why therapy remains decisive after PCI

The biological substrate of coronary disease usually extends beyond the stented segment. PCI treats flow-limiting stenoses, but it does not remove systemic endothelial dysfunction, dyslipidemia, inflammation, insulin resistance, hypertension, smoking-related injury, obesity, sedentary behavior, or chronic kidney disease. A technically successful PCI can therefore coexist with high residual risk. This residual risk is particularly visible in patients with multivessel disease, diabetes, chronic kidney disease, previous myocardial infarction, recurrent revascularization, peripheral artery disease, or persistent inflammatory and metabolic abnormalities.

The stent itself also creates a temporary therapeutic obligation. Endothelial healing requires correct antiplatelet therapy, careful education, and avoidance of premature discontinuation. At the same time, modern practice recognizes that prolonged antithrombotic therapy is not automatically better for every patient. Bleeding risk is not a

secondary issue; it is a prognostic factor. The clinician must therefore individualize the balance between ischemic protection and bleeding avoidance.

Post-PCI therapy can be understood as five parallel tasks: protection of the stent, protection of the remaining coronary tree, relief of symptoms, treatment of comorbidities that accelerate atherosclerosis, and education that converts a prescription into long-term adherence. Fragmentation occurs when these tasks are handled separately without a written pathway. A patient may receive aspirin and a P2Y12 inhibitor but remain far from LDL-C goals; another may receive intensive lipid therapy but no clear plan for the duration of dual antiplatelet therapy; another may have persistent angina after PCI but no systematic assessment for residual ischemia, microvascular angina, vasospasm, anemia, or uncontrolled hypertension.

Table 1. Core therapeutic priorities after PCI in chronic coronary disease

Domain	Clinical purpose	Practical outpatient action
Antithrombotic therapy	Prevent stent thrombosis and recurrent ischemic events while limiting bleeding.	Document the exact antiplatelet plan, expected duration, gastroprotection when indicated, bleeding warning signs, and actions before surgery or dental procedures.
Lipid lowering	Reduce progression of atherosclerosis and future cardiovascular events.	Use high-intensity or maximally tolerated statin, check LDL-C response, add ezetimibe early if target is not reached, and consider PCSK9 inhibitor or bempedoic acid where appropriate and available.
Blood pressure and hemodynamics	Reduce recurrent ischemia, stroke, heart failure, and renal risk.	Set individualized BP goal, avoid hypotension in elderly/frail patients, prioritize ACE inhibitor/ARB when hypertension, diabetes, CKD, or LV dysfunction is present.
Diabetes and obesity	Lower cardiometabolic and atherosclerotic risk beyond glucose numbers alone.	Prefer agents with cardiovascular benefit when indicated, especially SGLT2 inhibitors and GLP-1 receptor agonists in eligible patients.
Angina and functional status	Confirm that PCI improved symptoms and detect residual disease or non-obstructive mechanisms.	Assess angina class, exercise tolerance, nitrate use, adherence, and consider non-invasive or invasive reassessment if symptoms persist.
Lifestyle and rehabilitation	Convert procedural success into sustained risk reduction.	Prescribe smoking cessation, diet, physical activity, cardiac rehabilitation, vaccination, and clear follow-up responsibilities.

Antithrombotic therapy: from routine DAPT to individualized risk

Antithrombotic therapy is the most visible medication issue after PCI. The traditional approach of prescribing dual antiplatelet therapy for a fixed period remains useful, but contemporary practice is more individualized. The decision depends on clinical presentation, stent characteristics, bleeding risk, ischemic risk, need for oral anticoagulation, renal function, age, history of gastrointestinal bleeding, anemia, and planned surgery. A patient after elective PCI for chronic coronary disease differs from a patient treated for acute coronary syndrome, and a frail patient with anemia differs from a younger patient with complex multivessel PCI and low bleeding risk.

A practical post-PCI prescription should include the names and doses of antiplatelet drugs, the intended minimum duration, the plan after DAPT completion, the reason for any shortened or prolonged course, and the warning that therapy should not be stopped without medical consultation. This simple documentation step prevents many avoidable errors. Patients often know that they received a stent but cannot name the drug that protects it. Some stop the P2Y12 inhibitor because of bruising, epigastric discomfort, dental work, or misunderstanding of the prescription. Others continue DAPT indefinitely because no one reassessed the plan.

In chronic coronary disease after PCI, aspirin plus clopidogrel is a common initial strategy, while more potent

P2Y12 inhibition is generally reserved for specific high-risk contexts, particularly acute coronary syndrome. In high bleeding risk patients, shorter DAPT followed by single antiplatelet therapy may be appropriate if ischemic risk is acceptable and the PCI result is uncomplicated. Conversely, prolonged intensified antithrombotic therapy may be considered in selected patients with high ischemic risk and low bleeding risk. Patients with atrial fibrillation or another indication for oral anticoagulation require a separate strategy that minimizes the duration of triple therapy and avoids unnecessary bleeding.

The therapist should not treat antiplatelet therapy as a fixed instruction that belongs only to the interventional cardiologist. Instead, outpatient follow-up should verify adherence, bleeding, drug interactions, hemoglobin if clinically indicated, proton-pump inhibitor need in gastrointestinal risk, and exact timing of de-escalation. Any planned invasive procedure must trigger communication with the cardiologist, especially during the early months after stent implantation.

Table 2. Practical antithrombotic decisions after PCI

Clinical situation	Usual direction of therapy	Key outpatient check
Elective PCI for chronic coronary disease, average bleeding risk	DAPT for the guideline-recommended period, commonly aspirin plus clopidogrel, followed by single antiplatelet therapy.	Confirm adherence, date of PCI, stent type if available, and planned date for transition to monotherapy.
High bleeding risk after uncomplicated PCI	Consider shorter DAPT when supported by the procedural report and ischemic risk is not high.	Document hemoglobin history, prior bleeding, age/frailty, renal function, and gastroprotection.
Complex PCI or high ischemic risk with low bleeding risk	Longer or intensified antithrombotic strategy may be considered individually.	Look for diabetes, multivessel disease, recurrent MI, long stent length, bifurcation PCI, peripheral artery disease.
PCI after acute coronary syndrome	More intensive antiplatelet strategy is usually required unless bleeding risk is prohibitive.	Do not shorten therapy without reviewing ACS diagnosis, culprit lesion, bleeding risk, and cardiology recommendation.
Atrial fibrillation requiring oral anticoagulation	Avoid prolonged triple therapy; use an anticoagulant-centered strategy with the shortest safe combination period.	Check renal function, anticoagulant dose, bleeding risk, and drug interactions.
Upcoming surgery, endoscopy, or dental procedure	Never stop antiplatelet therapy automatically; decision depends on timing after PCI and urgency of procedure.	Contact interventional cardiology if procedure is planned within the high-risk post-stent period.

Lipid-lowering therapy: reaching the target rather than prescribing a statin

Lipid management after PCI should be goal-directed. The clinical question is not whether the patient is taking a statin, but whether LDL-C has reached the recommended target and whether the reduction from baseline is sufficient. Patients after PCI are usually considered at very high cardiovascular risk. For many such patients, an LDL-C goal below 55 mg/dL together with at least a 50% reduction from baseline is recommended in European guidance. Very high residual risk or recurrent events may justify even more intensive targets according to specialist judgment.

High-intensity statin therapy remains the foundation. Atorvastatin and rosuvastatin at evidence-based doses are commonly used, but the term "high-intensity" should not be replaced by vague wording such as "statin therapy." If LDL-C remains above target after several weeks of adherence, ezetimibe should be added rather than waiting for many months. If the target is still not achieved, PCSK9 inhibitors can provide substantial additional LDL-C reduction where accessible. Bempedoic acid is an important option for patients with statin intolerance or insufficient response despite statin and ezetimibe, particularly after evidence showing reduction of cardiovascular events in statin-intolerant patients.

Common errors include prescribing a moderate-intensity statin after PCI without a reason, failing to repeat the lipid profile, interpreting partial LDL-C reduction as success despite remaining above target, attributing all muscle pain to statins without assessment, and not adding ezetimibe because the patient is already "on treatment." A therapy-led pathway should include a lipid check at approximately 4-12 weeks after initiation or dose adjustment and again after treatment escalation. Liver enzymes and creatine kinase should be assessed when clinically indicated rather than used as a reason for avoiding therapy in asymptomatic patients.

Patients should be told that lipid-lowering therapy is not a short course after stenting. The stent treats one lesion, whereas LDL-C lowering protects the entire arterial system. Explaining this difference often improves adherence because the patient understands why tablets continue even after chest pain has disappeared.

Table 3. Stepwise lipid-lowering escalation after PCI

Step	Action	When to escalate
1	Start or continue high-intensity statin at the highest tolerated dose.	Immediately after PCI unless contraindicated or truly not tolerated.
2	Check LDL-C response and adherence.	Usually 4-12 weeks after initiation or dose change.
3	Add ezetimibe.	If LDL-C remains above target or expected reduction is insufficient.
4	Consider PCSK9 inhibitor.	If target is not reached despite maximally tolerated statin plus ezetimibe, especially in very high-risk patients.
5	Consider bempedoic acid.	In statin intolerance or insufficient response where appropriate and available.
6	Continue long-term monitoring.	Repeat lipid profile after each escalation and at routine follow-up; reinforce adherence at every visit.

Blood pressure, renin-angiotensin system blockade and beta-blockers

Hypertension is one of the most frequent comorbidities in patients undergoing PCI. Blood pressure control reduces recurrent ischemic events, stroke, heart failure, renal progression and mortality risk. A target around 130/80 mmHg is often appropriate when tolerated, but the clinician must individualize therapy in elderly patients, patients with orthostatic symptoms, advanced chronic kidney disease, or low diastolic pressure with persistent angina. The aim is stable control rather than episodic aggressive reduction.

ACE inhibitors or angiotensin receptor blockers are particularly important when hypertension coexists with diabetes, chronic kidney disease, left ventricular dysfunction, or previous myocardial infarction. They should be titrated thoughtfully with monitoring of renal function and potassium. Mineralocorticoid receptor antagonists are reserved for appropriate heart failure or post-MI indications and require electrolyte monitoring.

Beta-blocker therapy requires a more nuanced interpretation than in older practice. Beta-blockers remain important for symptom control, heart rate reduction, prior myocardial infarction in selected periods, arrhythmias, hypertension, and reduced left ventricular ejection fraction. However, indefinite beta-blocker therapy solely to improve outcomes is not automatically necessary in all patients with chronic coronary disease and normal left ventricular function, especially when there has been no recent myocardial infarction and no other indication. Reassessment is therefore part of good care, not withdrawal of cardioprotection. In symptomatic angina, beta-blockers, calcium-channel blockers and long-acting nitrates remain useful according to blood pressure, heart rate, left ventricular function, vasospastic suspicion, and tolerance.

In daily practice, the therapist should document heart rate, blood pressure sitting and standing when needed, angina frequency, nitrate use, dyspnea, edema, bradycardia, fatigue, and medication interactions. This prevents two common extremes: leaving the patient undertreated because

PCI was performed, or overmedicating the patient with multiple hemodynamic drugs despite dizziness and low blood pressure.

Diabetes, obesity and chronic kidney disease: cardiometabolic therapy after PCI

Diabetes, obesity and chronic kidney disease markedly increase residual risk after PCI. Their treatment should not be limited to glucose, weight or creatinine values alone. Cardiometabolic therapy now includes drug classes with proven cardiovascular or renal benefit. In patients with chronic coronary disease and type 2 diabetes, SGLT2 inhibitors and GLP-1 receptor agonists with demonstrated cardiovascular benefit should be considered according to comorbidities, renal function, body weight, cost, access and contraindications. In patients with heart failure, SGLT2 inhibitors have a particularly important role across diabetic and non-diabetic populations, depending on ejection fraction and guideline indications.

GLP-1 receptor agonists are relevant not only for glycemic control but also for weight management and cardiovascular risk reduction in selected patients. Semaglutide has shown cardiovascular outcome benefit in patients with established cardiovascular disease and overweight or obesity without diabetes, expanding the discussion beyond traditional diabetic indications. This does not mean that every post-PCI patient requires injectable therapy. It means that obesity should be treated as a cardiovascular disease modifier rather than a cosmetic issue.

Chronic kidney disease affects antithrombotic safety, lipid therapy choices, blood pressure targets, contrast exposure history and medication dosing. After PCI, renal function should be reviewed, especially in older patients, those with diabetes, those receiving diuretics or renin-angiotensin system blockers, and those exposed to repeated contrast procedures. Albuminuria, if available, adds prognostic information and may guide renal-protective therapy.

A practical cardiometabolic post-PCI visit should therefore include HbA1c or recent glycemic assessment, renal function, electrolytes, body mass index, waist circumference where feasible, smoking status, physical activity, and medication affordability. The most evidence-based prescription is ineffective if the patient cannot obtain it or does not understand why it was added.

Antianginal therapy and persistent symptoms after PCI

Absence of chest pain after PCI is reassuring but should not be assumed. Some patients have persistent or recurrent symptoms due to incomplete revascularization, restenosis, stent thrombosis, progression of non-culprit lesions, microvascular dysfunction, vasospasm, anemia, uncontrolled hypertension, tachyarrhythmia, gastroesophageal disease, anxiety, or musculoskeletal pain. The outpatient physician should ask specifically about exertional chest discomfort, dyspnea as an anginal equivalent, nitrate response, exercise tolerance, and changes compared with the pre-PCI period.

Antianginal therapy should be selected according to phenotype. Beta-blockers may be preferred when heart rate is high or there is previous myocardial infarction or reduced ejection fraction. Calcium-channel blockers are useful in hypertension and suspected vasospastic components, while non-dihydropyridine agents require caution in bradycardia or left ventricular dysfunction. Long-acting nitrates may be helpful but require nitrate-free intervals and attention to hypotension. Ranolazine can be considered in patients with persistent symptoms despite standard antianginal drugs, particularly when blood pressure or heart rate limits further titration.

Persistent angina after technically successful PCI should not be dismissed as psychological. It should trigger a structured evaluation: adherence to antiplatelet therapy, adequacy of antianginal medication, electrocardiographic changes, anemia, blood pressure, heart rate, diabetes control, echocardiographic function when needed, and non-invasive ischemia testing or repeat coronary assessment if clinical risk is significant. In women and in patients with diffuse atherosclerosis, microvascular disease and vasospasm deserve particular attention.

Cardiac rehabilitation and patient education

Cardiac rehabilitation is often underused after PCI, especially when the procedure was elective and hospitalization was short. Yet rehabilitation provides a structured opportunity to address physical activity, diet, smoking cessation, weight, psychological recovery, return to work, medication adherence, and recognition of warning symptoms. The therapist should present rehabilitation not as optional exercise but as part of secondary prevention.

Patient education should be short, repeated, and specific. A useful discharge explanation includes five messages: do not stop antiplatelet drugs without medical advice; lipid-lowering therapy protects all arteries and is long-term; chest pain, black stool, syncope, severe dyspnea or neurological symptoms require urgent care; follow-up blood tests are part of treatment; and lifestyle changes are medications without

tablets. Written instructions should name the stented artery when known, the date of PCI, the planned antiplatelet duration, and the next visit date.

Education is also necessary for relatives. In many families, medication decisions are influenced by spouses or adult children who may advise stopping drugs when symptoms improve or when bruising appears. Including relatives in the explanation can prevent dangerous interruptions.

A practical outpatient algorithm after PCI

The proposed pathway divides follow-up into three clinically realistic time points. The first visit focuses on safety and understanding; the second on escalation toward targets; the third on stability and long-term prevention. The exact timing may vary according to local practice, but the logic should remain consistent.

Table 4. Follow-up checklist after PCI for therapy and interventional cardiology practice

Time point	Main purpose	Minimum checklist
7-14 days	Safety, adherence, early complications.	Review discharge summary, confirm antiplatelet drugs and doses, check bleeding/bruising, ask about recurrent chest pain, review BP/HR, assess access-site complaints, correct prescription errors, explain warning symptoms.
1-3 months	Therapy escalation and target achievement.	Check lipid profile, renal function and glucose status; intensify statin/ezetimibe pathway; reassess BP and antianginal therapy; address smoking, weight, rehabilitation; document antiplatelet duration.
6-12 months	Long-term prevention and de-escalation decisions.	Decide on transition from DAPT to single antiplatelet therapy when appropriate; reassess LDL-C target; review beta-blocker indication; evaluate angina and functional status; plan annual follow-up.
Any unscheduled visit	Detect danger signs.	Evaluate chest pain, dyspnea, syncope, major bleeding, neurological symptoms, medication interruption, planned surgery, or inability to obtain drugs.

Risk-triggered actions in high-priority groups

A single post-PCI protocol should be flexible enough to identify subgroups that need more intensive follow-up. Table 5 summarizes risk-triggered actions that can be incorporated into the medical record or outpatient checklist.

Table 5. Risk-triggered actions after PCI

Patient feature	Why it matters	Action
Diabetes mellitus	Higher risk of recurrent ischemia, restenosis, kidney disease and diffuse atherosclerosis.	Check HbA1c, renal function and albuminuria if available; consider SGLT2 inhibitor or GLP-1 receptor agonist with cardiovascular benefit.
Chronic kidney disease	Higher bleeding risk, medication dosing concerns, increased cardiovascular mortality.	Monitor creatinine/eGFR and potassium; adjust doses; avoid unnecessary nephrotoxins; individualize antithrombotic intensity.
Previous MI or multivessel disease	Higher ischemic risk beyond the stented lesion.	Ensure intensive lipid lowering, strong adherence work, careful antithrombotic review and low threshold for cardiology reassessment if symptoms recur.
Anemia or previous bleeding	Higher risk of harm from prolonged or intensive antithrombotic therapy.	Investigate reversible causes, consider gastroprotection, review DAPT duration, and coordinate before procedures.

Patient feature	Why it matters	Action
Persistent angina	May reflect residual ischemia, microvascular disease, vasospasm or non-cardiac causes.	Assess adherence, ECG, BP/HR, anemia, antianginal therapy; refer for further testing if symptoms are exertional or progressive.
Obesity	Associated with hypertension, diabetes, inflammation and lower functional capacity.	Use structured weight plan; consider GLP-1 receptor agonist in eligible patients; prescribe rehabilitation and nutrition counseling.

3. Discussion

The main practical message of this review is that PCI creates a therapeutic opportunity. Patients are often more motivated immediately after a coronary procedure, and this period should be used to establish durable secondary prevention. A clear pathway helps prevent loss of momentum after discharge. It also reduces the false separation between "interventional" and "therapeutic" cardiology. The intervention opens the artery; therapy keeps the patient stable.

A structured post-PCI pathway is particularly valuable in healthcare systems where follow-up may be distributed across several clinicians. Without a shared checklist, important decisions can be missed because each physician assumes that another specialist has addressed them. The interventional cardiologist may assume that the therapist will intensify lipid therapy; the therapist may assume that the cardiologist has already selected the final antiplatelet duration; the patient may assume that medications are temporary because the stent has solved the problem. A written algorithm assigns clinical tasks to specific visits rather than to vague responsibility.

The proposed model emphasizes early lipid escalation because delayed achievement of LDL-C targets is common in real practice. The addition of ezetimibe is simple, generally well tolerated and often underused. PCSK9 inhibitors and bempedoic acid may be limited by availability and cost, but their role should still be documented for eligible high-risk patients. Documentation is useful even when the drug is not immediately accessible because it identifies unmet therapeutic need.

The second key point is individualized antithrombotic care. Modern drug-eluting stents and trial evidence allow shorter DAPT in selected high bleeding risk patients, but this should not be interpreted as permission for random interruption. Conversely, routine indefinite DAPT is not a marker of better care. The best approach is explicit documentation of ischemic risk, bleeding risk, clinical presentation and planned duration.

The third point is cardiometabolic treatment. Diabetes and obesity should not be managed separately from coronary disease. The availability of SGLT2 inhibitors and GLP-1 receptor agonists with cardiovascular outcome evidence means that the post-PCI medication review should include more than antiplatelet drugs and statins. In high-risk patients, the choice of glucose-lowering or weight-loss medication can be part of cardiovascular prevention.

Finally, symptom assessment remains essential. PCI may improve epicardial flow, but persistent angina is not rare. A patient with ongoing symptoms should not simply receive repeated reassurance. The clinician should confirm adherence, evaluate hemodynamics, optimize antianginal therapy, and consider residual ischemia, microvascular dysfunction or vasospasm. This is especially relevant in patients whose angiographic result was satisfactory but whose functional limitation remains significant.

4.Limitations

This article is a narrative review and practical synthesis rather than a systematic review. It does not provide pooled effect estimates, and it does not test the proposed pathway in a real clinical cohort. Medication access, reimbursement, patient education level, laboratory availability and specialist referral pathways differ substantially between healthcare systems. Therefore, local adaptation is necessary. The algorithm should be considered a structured clinical tool that supports decision-making, not a substitute for individualized physician judgment.

For future research, the pathway can be evaluated prospectively in an outpatient department by comparing target achievement, medication adherence, recurrent angina, bleeding, LDL-C levels, blood pressure control and unplanned hospital visits before and after implementation. Such a study would require appropriate ethical approval, anonymized data collection and transparent reporting.

5.Conclusion

Post-PCI care should be understood as a long-term therapeutic process rather than a short procedural aftercare period. In chronic coronary disease, the durability of PCI benefit depends on correct antithrombotic therapy, intensive lipid lowering, blood pressure control, cardiometabolic treatment, symptom reassessment, rehabilitation and adherence. Internal medicine and therapy departments play a decisive role because they often supervise the patient after discharge.

A structured outpatient algorithm can help translate contemporary guideline recommendations into everyday practice. The most important steps are to document antiplatelet duration, actively pursue LDL-C targets, reassess beta-blocker and antianginal indications, treat diabetes and obesity with cardiovascular benefit in mind, and schedule follow-up that checks outcomes rather than merely renews prescriptions. Integration between interventional cardiology and therapy is therefore not optional; it is the practical foundation of secondary prevention after PCI.

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