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Antiplatelet Therapy After Coronary Artery Bypass Grafting: Current Status and Future Perspectives

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Abstract: Antiplatelet therapy is a critical component in managing patients undergoing coronary artery bypass grafting (CABG) to prevent graft occlusion and reduce thrombotic events. Current treatment options primarily include monotherapy with aspirin, dual antiplatelet therapy (DAPT), combining aspirin with a P2Y12 inhibitor, and, in some cases, triple therapy incorporating an anticoagulant. Recent advancements have focused on optimizing treatment regimens to balance the efficacy of antithrombotic protection with the associated risks of bleeding. Personalized treatment approaches, driven by patient-specific factors and genetic profiling, are increasingly recognized as essential for improving patient outcomes. Ongoing research aims to identify biomarkers that can predict patient responses to antiplatelet therapy, enabling more precise and effective treatment strategies. Additionally, novel therapeutic targets and new antiplatelet agents are being investigated to enhance the safety and efficacy of treatment. The results of these studies will shape the future of antiplatelet therapy and contribute to better clinical practices. The integration of genetic and pharmacogenomic data is anticipated to play a significant role in the personalization of antiplatelet therapy, ultimately leading to improved patient care in the context of CABG. This review article provides a comprehensive analysis of the current status and future perspectives of antiplatelet therapy in CABG.

Keywords: Aspirin, antiplatelet therapy, clopidogrel, coronary artery bypass grafting, dual antiplatelet therapy

INTRODUCTION

Coronary artery bypass grafting (CABG) is a well-established surgical intervention for the treatment of coronary artery disease (CAD), which remains a leading cause of morbidity and mortality worldwide [1]. CABG involves the creation of alternative pathways for blood to bypass occluded coronary arteries, thereby improving myocardial perfusion and reducing the risk of ischemic events [2]. Despite the success of CABG in restoring adequate blood flow, patients remain at risk for graft occlusion and subsequent cardiovascular events, necessitating the implementation of adjunctive medical therapies [3].

Antiplatelet therapy plays a critical role in the management of patients post-CABG [4]. Platelets contribute to thrombus formation within the coronary arteries and grafts, potentially leading to graft failure and adverse clinical outcomes [5]. Therefore, the use of antiplatelet agents is crucial to inhibit platelet aggregation, maintain graft patency, and improve long-term survival [4]. Commonly prescribed antiplatelet agents include aspirin, clopidogrel, and newer agents such as ticagrelor, each with their own efficacy and safety profiles [6].

The purpose of this review is to provide an in-depth analysis of the current status of antiplatelet therapy following CABG, highlighting the evidence supporting existing

practices, as well as identifying the challenges and limitations associated with these therapies. Additionally, this article aims to explore future perspectives in the field, including emerging therapies, personalized medicine approaches, and ongoing research endeavors. By synthesizing the latest available data, this review seeks to inform clinical practice and guide future research efforts in optimizing antiplatelet therapy for CABG patients.

CURRENT STATUS OF ANTIPLATELET THERAPY POST-CABG

Standard Antiplatelet Therapy Protocols

Post- CABG, standard antiplatelet therapy protocols typically involve dual antiplatelet therapy (DAPT) or single antiplatelet therapy (SAPT). DAPT usually consists of aspirin (75-100 mg daily) and a P2Y12 inhibitor such as clopidogrel, ticagrelor, or prasugrel [7] (Table 1). The duration of DAPT varies but is often around 12 months, followed by SAPT with aspirin alone [8]. The choice of therapy and duration depends on the patient's risk of thrombotic events versus bleeding complications.

Table 1: Currently prescribed antiplatelet agents post-CABG

Antiplatelet Agent	Mechanism of Action	Common Dosage	Duration	Side Effects	Important Interactions
Aspirin	COX-1 inhibitor	75-100 mg daily	Long-term	Bleeding, upset stomach, bruising, gastrointestinal ulcers	NSAIDs (e.g., ibuprofen), anticoagulants (e.g., warfarin), ACE inhibitors
Clopidogrel	P2Y12 ADP receptor inhibitor	75 mg daily	12 months (DAPT), Long-term (SAPT)	Bleeding, bruising, gastrointestinal issues	NSAIDs, proton pump inhibitors (PPIs), anticoagulants
Ticagrelor	Direct-acting P2Y12 receptor antagonist	90 mg twice daily	12 months (DAPT), Long-term (SAPT)	Bleeding, dyspnea, bruising	PPIs, anticoagulants
Prasugrel	Thienopyridine P2Y12 inhibitor	10 mg daily	12 months (DAPT), Long-term (SAPT)	Bleeding, gastrointestinal issues, rash	NSAIDs, PPIs, anticoagulants
Cangrelor	Intravenous P2Y12 receptor blocker	IV infusion	Short-term (acute setting)	Bleeding, hypotension	Anticoagulants, NSAIDs

DAPT involves the use of two antiplatelet agents to provide a more potent anti-thrombotic effect. The combination of aspirin and a P2Y12 inhibitor is particularly effective in preventing platelet aggregation and thrombus formation [9]. The most commonly used P2Y12 inhibitors in DAPT are clopidogrel, ticagrelor, and prasugrel. The duration of DAPT is typically around 12 months, but it can be extended based on the patient's risk profile and

clinical judgment [10]. After the initial period of DAPT, patients are often transitioned to SAPT, which involves the use of aspirin alone. This approach is continued for long-term management to maintain antiplatelet effects while minimizing the risk of bleeding [11].

The choice of antiplatelet therapy and its duration is tailored to each patient based on their individual risk factors. Factors such as age, history of bleeding, comorbid conditions, and the type of graft used in CABG are considered when determining the appropriate therapy [12]. For example, patients with a higher risk of bleeding may have a shorter duration of DAPT, while those with a higher risk of thrombotic events may require extended therapy [13]. Regular monitoring and follow-up are essential to assess the efficacy and safety of antiplatelet therapy. Platelet function tests and clinical evaluations are used to ensure that the therapy is achieving the desired outcomes without causing significant adverse effects [14]. Adjustments to the therapy may be made based on the patient's response and any new risk factors that arise during follow-up.

Evidence Supporting Current Practices

Current practices are supported by several recent clinical trials and studies [4,15-18] (Table 2). The DACAB trial demonstrated that ticagrelor plus aspirin significantly reduced the risk of major adverse cardiovascular events (MACE) compared to aspirin alone or ticagrelor monotherapy over a five-year follow-up. Additionally, guidelines from organizations such as the American College of Cardiology (ACC) and the American Heart Association (AHA) recommend DAPT for patients undergoing CABG to improve graft patency and reduce thrombotic complications [19].

Table 2: Key studies comparing SAPT and DAPT for CABG

Study	Year	Type of Study	Number of Patients	Key Findings
DACAB Trial[4]	2023	Randomized Controlled Trial	500	Ticagrelor plus aspirin reduced MACE compared to aspirin alone or ticagrelor monotherapy over five years.
Kim et al.[15]	2023	Propensity score matching	671	Patients who received DAPT and remained stable for 1 year, SAPT maintenance with clopidogrek or aspirin did not show any significant differences in 4-year outcomes such as all-cause mortality, major adverse events, and newly occurring graft occlusion. However, more patients taking aspirin required changes in antiplatelet regimens to other antiplatelet or anticoagulation therapies.
Nei et al.[16]	2022	Retrospective	2341	DAPT was not associated with an increase in composite bleeding compared to SAPT.
Hess et al.[17]	2021	Propensity score matching	3562	DAPT was associated with higher rate of postoperative transfusion but similar

				overall survival and rates of MACCE compared with aspirin.
Rocha-Gomes et al.[18]	2020	Retrospective	351	DAPT showed a non-significant impact on long-term survival and demonstrated to be a safe option compared with aspirin.

DAPT = dual antiplatelet therapy; MACE = major adverse cardiovascular events; MACCE = major adverse cardiovascular and cerebrovascular events; SAPT = single antiplatelet therapy

A meta-analysis by Solo et al. highlighted the benefits of DAPT in reducing the risk of graft occlusion and major adverse cardiovascular events post-CABG [12]. The study emphasized the importance of individualized therapy based on patient-specific factors such as age, bleeding risk, and comorbid conditions.

The ACC/AHA guidelines provide a framework for tailoring antiplatelet therapy duration based on the balance between ischemic and bleeding risks. These guidelines recommend a minimum of 6-12 months of DAPT, with the possibility of extending therapy in patients with a higher risk of thrombotic events and shorter duration for those with a higher bleeding risk [19].

Clinical Outcomes and Efficacy

Clinical outcomes post-CABG with antiplatelet therapy have shown significant benefits. DAPT has been associated with improved vein graft patency and reduced rates of myocardial infarction, stroke, and coronary revascularization. However, it is essential to balance these benefits with the risk of bleeding, particularly in elderly patients or those with a history of bleeding disorders [20]. Studies have indicated that while DAPT reduces the risk of MACE, it also increases the risk of major bleeding events [21].

CHALLENGES AND LIMITATIONS

Antiplatelet Resistance

Antiplatelet resistance refers to the reduced effectiveness of antiplatelet medications, such as aspirin and clopidogrel, in inhibiting platelet aggregation. This phenomenon can lead to recurrent cardiovascular events despite treatment [22]. The prevalence of antiplatelet resistance varies, with an overall prevalence of 39% in patients who underwent CABG [23]. The impact on clinical outcomes is significant, as resistant patients are at a higher risk of adverse events, including stroke and myocardial infarction [24].

Several factors contribute to antiplatelet resistance, including genetic polymorphisms, drug interactions, and patient-specific factors (Table 3). Genetic variations in the CYP2C19 enzyme, which is responsible for metabolizing clopidogrel, can lead to reduced activation of the drug and decreased platelet inhibition [25]. Additionally, concomitant use of other medications, such as proton pump inhibitors, can interfere with the absorption and effectiveness of antiplatelet drugs [26].

Table 3: Factors associated with antiplatelet resistance

Factor	Description
Genetic Polymorphisms	Variations in genes such as CYP2C19 can affect drug metabolism and reduce the effectiveness of antiplatelet medications.
Drug Interactions	Concomitant use of other medications, such as proton pump inhibitors, can interfere with the absorption and effectiveness of antiplatelet drugs.
Patient Age	Older patients may have altered drug metabolism and increased risk of resistance.
Comorbid Conditions	Conditions such as diabetes and renal impairment can impact drug efficacy and contribute to resistance.
Adherence to Therapy	Poor patient compliance with medication regimens can lead to suboptimal drug levels and resistance.
Bioavailability Issues	Variations in drug absorption and distribution can affect the therapeutic levels of antiplatelet medications.
Platelet Function Variability	Individual differences in platelet reactivity can influence the response to antiplatelet therapy.

The presence of antiplatelet resistance poses a significant challenge in the management of cardiovascular diseases. Patients with resistance are more likely to experience MACE, including recurrent ischemic stroke and myocardial infarction [27]. To address this issue, healthcare providers may consider alternative antiplatelet agents, such as ticagrelor or prasugrel, which have shown better efficacy in patients with clopidogrel resistance. Additionally, platelet function tests can be used to monitor the effectiveness of antiplatelet therapy and guide treatment decisions [28] (Table 4).

Table 4: Strategies to counter antiplatelet resistance

Strategy	Description
Platelet Function Tests	Monitoring platelet function to assess the effectiveness of antiplatelet therapy and guide treatment decisions.
Alternative Antiplatelet Agents	Use of more potent antiplatelet agents such as ticagrelor or prasugrel, which have shown better efficacy in patients with clopidogrel resistance.
Genetic Testing	Identifying genetic polymorphisms, such as CYP2C19 variants, to tailor antiplatelet therapy based on individual genetic profiles.
Dose Adjustments	Modifying the dosage of antiplatelet medications to achieve optimal therapeutic levels.
Combination Therapy	Using dual antiplatelet therapy (DAPT) with aspirin and a P2Y12 inhibitor to enhance platelet inhibition.
Patient Education	Educating patients about the importance of adherence to medication regimens to improve treatment outcomes.

Bleeding Risks and Management

Antiplatelet therapy, essential for preventing thrombotic events, inherently increases the risk of bleeding. This risk is influenced by several factors, including incomplete surgical hemostasis, residual heparin effects, platelet dysfunction, hypothermia during surgery, hemodilution, and postoperative hypertension. Age, comorbidities, and concomitant medications also significantly contribute to bleeding risks [29]. Therefore, a thorough assessment of these patient-specific factors is vital for effective management (Figure 1).

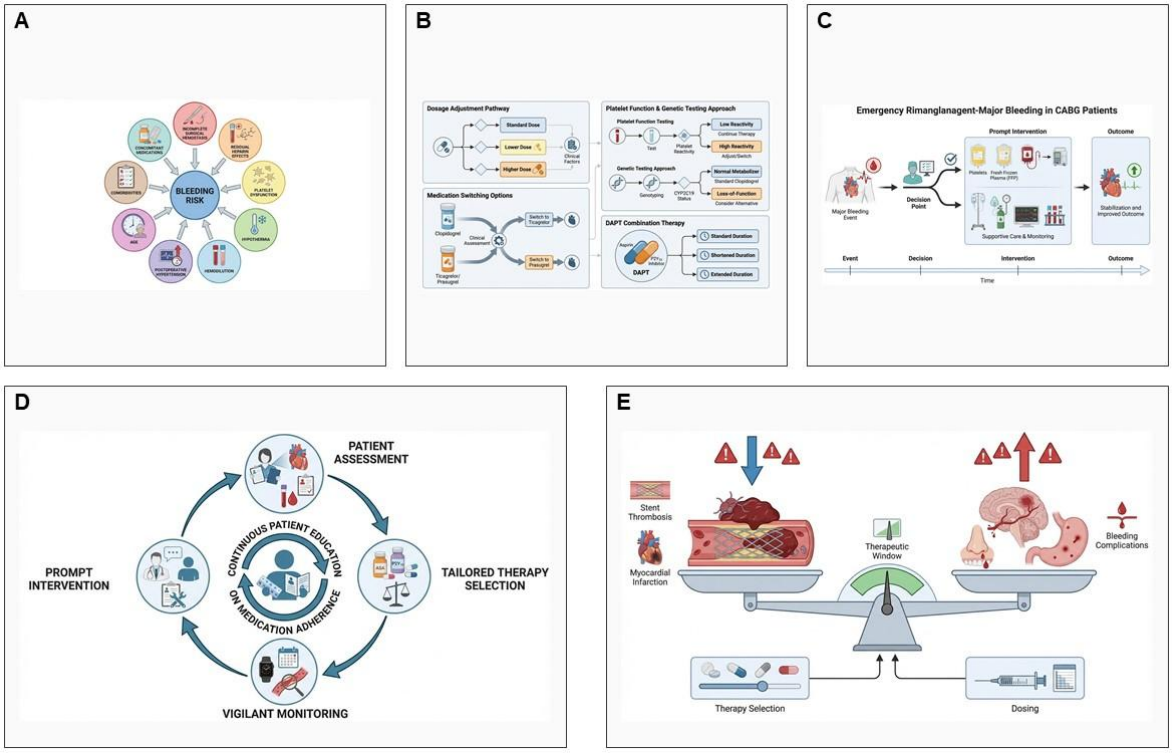

Figure 1. Antiplatelet Therapy Management and Bleeding Risk in CABG Patients

(A) Multiple factors influence bleeding risk in antiplatelet therapy patients.
(B) Personalized antiplatelet therapy optimization approaches and alternatives.
(C) Emergency intervention protocol for major bleeding management.
(D) Comprehensive management framework integrating assessment, therapy, monitoring, and intervention.
(E) Balancing thrombotic prevention benefits against bleeding risks.

To mitigate bleeding risks, various strategies can be employed. These include adjusting the dosage of antiplatelet medications to balance the benefits of preventing thrombosis with the risks of bleeding, and switching to alternative agents such as ticagrelor or prasugrel for patients exhibiting resistance to clopidogrel [30]. Platelet function tests and genetic testing can further personalize antiplatelet therapy, ensuring optimal effectiveness. Moreover, combination therapy with DAPT can enhance platelet inhibition, although it requires careful monitoring to avoid excessive bleeding [31]. Educating patients about the importance of adherence to their medication regimen is also crucial for improving treatment outcomes [32].

In instances of major bleeding, prompt intervention is essential. This includes the immediate administration of blood products such as platelets and fresh frozen plasma, along with supportive care [33]. Effective management of bleeding risks in CABG patients requires

a comprehensive approach that includes careful patient assessment, tailored antiplatelet therapy, vigilant monitoring, and prompt intervention when necessary [7]. By addressing these factors, healthcare providers can minimize bleeding complications and improve patient outcomes.

Patient Compliance Issues

Patient compliance, or adherence to prescribed treatment regimens, is a critical factor in the effectiveness of antiplatelet therapy [34]. Non-adherence can result from various factors, including complex dosing schedules, side effects, and a lack of understanding of the treatment's importance [35]. For instance, patients may struggle with the frequency and complexity of dosing schedules, leading to missed doses or discontinuation of therapy. Additionally, side effects such as gastrointestinal discomfort or bleeding can deter patients from adhering to their prescribed regimens [6]. Misunderstanding the importance of continued antiplatelet therapy, especially in preventing serious cardiovascular events, further exacerbates non-adherence.

Addressing these compliance issues requires a patient-centered approach, which involves educating patients about their condition and the critical role of antiplatelet therapy in their overall treatment plan [36]. Clear and comprehensive communication from healthcare providers can help patients understand the benefits and potential risks of their medications. This education should be tailored to the individual patient's needs, considering their literacy levels, language barriers, and cultural contexts [37]. Simplifying treatment regimens, such as reducing the number of daily doses or providing combination pills, can also improve adherence by making it easier for patients to follow their prescribed therapy [38].

Moreover, providing support systems to enhance adherence is crucial. This can include regular follow-ups, either in-person or via telemedicine, to monitor the patient's progress and address any concerns or side effects they may experience [39]. Involving family members or caregivers in the patient's treatment plan can also provide additional support and encouragement [40]. Utilizing reminders, such as pillboxes, smartphone apps, or automated calls, can help patients remember to take their medications consistently [41]. Financial support or assistance programs may also be necessary to ensure patients can afford their medications and do not skip doses due to cost constraints [42].

Ultimately, improving patient compliance with antiplatelet therapy requires a multifaceted and individualized approach [31]. By addressing the various barriers to adherence and providing continuous support and education, healthcare providers can enhance the effectiveness of antiplatelet therapy and improve patient outcomes [43]. This comprehensive strategy not only helps prevent recurrent cardiovascular events but also empowers patients to take an active role in managing their health.

Variability in Treatment Protocols

There is considerable variability in treatment protocols for antiplatelet therapy across different healthcare settings and patient populations. This variability can stem from differences in clinical guidelines, physician preferences, and patient-specific factors [44]. For example, while some healthcare providers might follow the guidelines issued by the

American College of Cardiology (ACC) or the European Society of Cardiology (ESC), others may adhere to local or regional guidelines that could have varying recommendations. Additionally, physicians' personal experiences, comfort levels, and familiarity with certain medications can influence their choice of treatment. Patient-specific factors such as age, comorbidities, genetic makeup, and risk of bleeding further add to this variability. As a result, patients receiving antiplatelet therapy may experience different outcomes based on the treatment protocols they follow [45].

Standardizing treatment protocols and promoting evidence-based practices can help reduce this variability and improve patient outcomes. By adopting standardized guidelines that are regularly updated with the latest research, healthcare providers can ensure that patients receive the most effective and safe treatments [46]. Moreover, training and education programs for healthcare professionals can facilitate the adoption of these guidelines, thereby minimizing the influence of individual preferences and ensuring consistency in care delivery. Collaborative efforts among healthcare institutions, professional organizations, and policymakers are essential to achieve this goal. Ultimately, reducing variability in treatment protocols can lead to better adherence to therapy, fewer adverse events, and improved overall cardiovascular health for patients on antiplatelet therapy [44,47].

Limitations of Current Studies and Data Gaps

Current studies on antiplatelet therapy face several limitations, which can hinder the robustness and generalizability of their findings. One significant limitation is the small sample sizes used in many studies. Smaller samples may not accurately represent the broader patient population, leading to biased results and limited statistical power [48]. Additionally, short follow-up periods are another common issue. Short-term studies may miss long-term outcomes and adverse events, making it challenging to fully understand the prolonged effects and safety of antiplatelet therapy [49]. Furthermore, there is often a lack of diversity in study populations. Most clinical trials tend to focus on specific demographic groups, which can lead to results that may not be applicable to a more diverse patient population. This lack of diversity can be particularly problematic when considering factors like age, sex, race, and comorbidities, which can influence treatment response and outcomes [50].

Moreover, significant data gaps remain in understanding the long-term effects of antiplatelet therapy. While short-term efficacy and safety are often well-documented, there is a need for more extensive research into the extended use of these therapies. The optimal duration of antiplatelet treatment is another area where data is lacking. Prolonged use of antiplatelet agents can increase the risk of bleeding, while shorter durations may not provide adequate protection against thrombotic events. Consequently, determining the ideal treatment duration requires more comprehensive studies that balance these risks and benefits [4]. Additionally, the impact of genetic factors on treatment response is an emerging area of interest. Genetic variations can significantly influence how patients metabolize and respond to antiplatelet medications, but current research in this area is still in its infancy. Understanding these genetic factors could lead to more personalized and effective treatment strategies, but further investigation is necessary to fully elucidate these relationships [32,51].

Addressing these limitations and data gaps is crucial for advancing the field of antiplatelet therapy. Larger, more diverse, and longer-term studies are needed to provide a more comprehensive understanding of the benefits and risks associated with these treatments. By doing so, healthcare providers can make more informed decisions and offer tailored treatment plans that improve patient outcomes and minimize adverse effects. Additionally, exploring the role of genetic factors in treatment response could pave the way for personalized medicine, ensuring that each patient receives the most appropriate and effective therapy based on their unique genetic makeup.

FUTURE PERSPECTIVES

Emerging Antiplatelet Agents and Therapies

The landscape of antiplatelet therapy is rapidly evolving with the development of novel agents aimed at improving efficacy and safety. Recent advancements include the introduction of new P2Y12 receptor antagonists, such as prasugrel and ticagrelor, which offer a more rapid onset and reversible effects compared to older agents like clopidogrel [52]. These newer agents have demonstrated improved patient outcomes, particularly in reducing the risk of major adverse cardiovascular events. By providing more consistent and potent platelet inhibition, these agents can potentially minimize the risk of thrombotic events such as heart attacks and strokes [53].

In addition to improvements in P2Y12 receptor antagonists, research is focusing on targeting alternative pathways involved in platelet activation and aggregation. For instance, the protease-activated receptor pathway and the thrombin receptor are being investigated as potential targets for new antiplatelet therapies [54,55]. These emerging therapies hold the promise of reducing thrombotic events while minimizing the risk of bleeding complications, which is a significant challenge with current treatments. By selectively targeting different mechanisms of platelet activation, these novel agents aim to provide more effective and safer treatment options for patients at high risk of thrombotic events.

These advancements in antiplatelet therapy reflect the ongoing efforts to enhance patient outcomes by developing more effective and safer treatment options. As research continues, it is likely that new therapies targeting various pathways of platelet activation will emerge, offering patients better protection against thrombotic events with fewer side effects. This progress underscores the importance of staying up-to-date with the latest developments in the field to ensure that patients receive the best possible care.

Personalized Medicine Approaches

Personalized medicine is revolutionizing the field of antiplatelet therapy by tailoring treatments to individual patient characteristics and risk profiles [56]. One key component of personalized medicine is the use of genetic testing to identify variations in genes that affect drug metabolism [57]. For instance, pharmacogenomic testing can detect specific genetic polymorphisms in the CYP2C19 gene, which influence how patients metabolize clopidogrel. Patients with certain polymorphisms are classified as poor metabolizers, meaning they do not effectively convert clopidogrel into its active form, thereby reducing its efficacy. By identifying these patients through genetic testing, healthcare providers can

prescribe alternative antiplatelet agents, such as prasugrel or ticagrelor, which do not rely on the same metabolic pathways and are more effective for these individuals [58]. This targeted approach helps ensure that patients receive the most appropriate and effective therapy, reducing the risk of thrombotic events and improving overall outcomes (Figure 2).

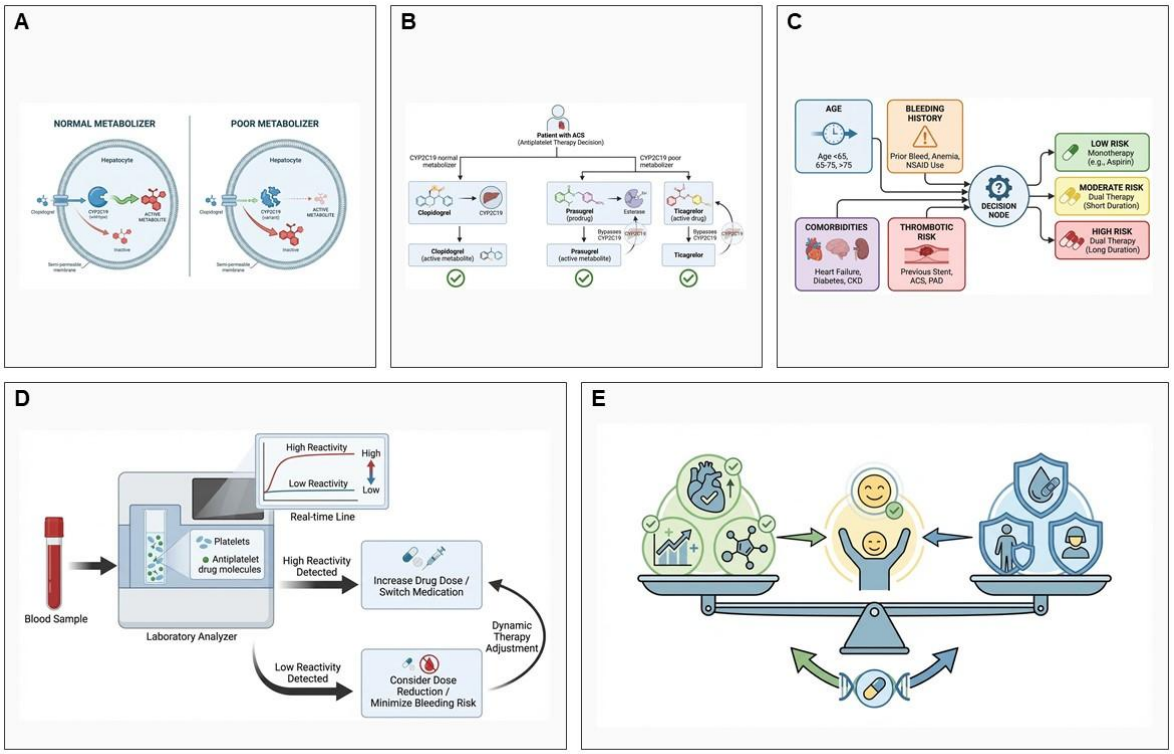

Figure 2. Personalized Medicine Approaches in Antiplatelet Therapy

(A) CYP2C19 polymorphisms determine clopidogrel metabolic efficiency.
(B) Personalized drug selection based on metabolizer phenotype.
(C) Multi-factor risk stratification guides personalized treatment planning.
(D) Real-time platelet function assays enable dynamic therapy optimization.
(E) Personalized approach optimizes therapeutic benefit-risk balance.

In addition to genetic testing, personalized medicine in antiplatelet therapy involves comprehensive risk stratification and the use of platelet function assays. Risk stratification considers various patient-specific factors, such as age, comorbidities, and history of bleeding, to determine the most suitable treatment plan. Platelet function assays measure the effectiveness of antiplatelet medications in real-time, allowing for adjustments in therapy based on individual patient response. This dynamic approach enables clinicians to balance the benefits of antiplatelet therapy with the risks of bleeding, providing a customized treatment strategy that maximizes efficacy while minimizing adverse events [59]. By incorporating these personalized medicine approaches, healthcare providers can enhance the safety and effectiveness of antiplatelet therapy, ultimately improving patient adherence and outcomes.

Advances in Pharmacogenomics

Pharmacogenomics has emerged as a critical field in the advancement of antiplatelet therapy, providing the foundation for more personalized and effective treatment strategies.

By analyzing genetic variations that influence drug metabolism and response, researchers and clinicians can tailor antiplatelet therapy to individual patients, maximizing therapeutic benefits while minimizing risks [60]. For example, specific alleles in the CYP2C19 gene can affect how well a patient metabolizes clopidogrel. Those with loss-of-function alleles may not convert the drug into its active form efficiently, leading to suboptimal platelet inhibition and a higher likelihood of thrombotic complications [61]. Recognizing these genetic variations enables the selection of more effective antiplatelet agents for patients who do not efficiently metabolize clopidogrel.

Furthermore, ongoing pharmacogenomic research continues to identify novel genetic markers that can impact the effectiveness and safety of antiplatelet therapies. For instance, polymorphisms in genes like ABCB1 and PON1 have been found to influence drug absorption, distribution, metabolism, and excretion [62,63]. By integrating these genetic insights into clinical practice, healthcare providers can make more informed decisions about antiplatelet therapy, ensuring that each patient receives the most appropriate and effective treatment based on their unique genetic profile. This personalized approach not only improves patient outcomes by reducing the risk of thrombotic events but also helps to minimize adverse effects, enhancing overall treatment adherence and satisfaction. As the field of pharmacogenomics continues to evolve, it holds the promise of further refining antiplatelet therapy, paving the way for more precise and individualized medical care.

Potential for Combination Therapies

Combination therapies are being explored as a means to enhance the antithrombotic effects of antiplatelet agents while mitigating their limitations in the context of CABG. DAPT, which involves the use of aspirin in combination with a P2Y12 inhibitor, is a common approach for patients undergoing CABG to prevent graft occlusion and reduce the risk of thrombotic events [64]. Recent studies have also investigated the potential benefits of combining antiplatelet agents with anticoagulants, such as direct oral anticoagulants (DOACs), in patients undergoing CABG with additional indications for anticoagulation, such as atrial fibrillation [65]. These combination therapies aim to provide comprehensive protection against thrombotic events while minimizing the risk of bleeding complications.

In addition to DAPT, recent research has focused on the use of triple therapy, which combines an antiplatelet agent, an anticoagulant, and aspirin, for patients undergoing CABG with multiple thrombotic risks. This approach is particularly beneficial for patients with complex cardiovascular conditions, as it provides a more robust antithrombotic effect [66]. However, the increased risk of bleeding associated with triple therapy necessitates careful patient selection and monitoring. Studies have shown that shorter durations of triple therapy, followed by dual therapy, can effectively balance the benefits of antithrombotic protection with the risks of bleeding [4]. This tailored approach to combination therapy is crucial for optimizing patient outcomes and ensuring the safe and effective management of thrombotic conditions in CABG patients.

Ongoing Clinical Trials and Research Directions

The field of antiplatelet therapy is continuously evolving, with numerous clinical trials and research initiatives underway. These studies are focused on evaluating the efficacy and

safety of new antiplatelet agents, optimizing personalized treatment strategies, and exploring novel therapeutic targets. For example, ongoing trials are investigating the use of factor XI inhibitors, which have shown promise in reducing thrombotic events without significantly increasing bleeding risk [67]. Additionally, research is being conducted to identify biomarkers that can predict patient responses to antiplatelet therapy, enabling more precise and effective treatment approaches [68]. The results of these trials will shape the future of antiplatelet therapy and contribute to improved patient care.

CONCLUSION

Antiplatelet therapy for CABG has evolved significantly, with DAPT and even triple therapy being the primary strategies to prevent graft occlusion and reduce thrombotic events. Recent studies have shown that DAPT, combining aspirin with a P2Y12 inhibitor, is effective in improving graft patency and reducing MACE. However, triple therapy, which includes an antiplatelet agent, an anticoagulant, and aspirin, is being explored for patients with multiple thrombotic risks, such as those with atrial fibrillation. The balance between antithrombotic efficacy and bleeding risk remains a critical consideration in optimizing treatment strategies[4].

The findings from recent clinical trials emphasize the importance of personalized treatment approaches in antiplatelet therapy for CABG patients. Clinicians should consider patient-specific factors, such as the presence of additional thrombotic risks and bleeding tendencies, when selecting the appropriate antiplatelet regimen. Shorter durations of triple therapy followed by dual therapy have shown promise in balancing the benefits of antithrombotic protection with the risks of bleeding. This tailored approach can help optimize patient outcomes and reduce the incidence of adverse events.

Ongoing research is focused on identifying biomarkers that can predict patient responses to antiplatelet therapy, enabling more precise and effective treatment approaches. Additionally, the development of new antiplatelet agents and the exploration of novel therapeutic targets are critical areas of investigation. Future studies should aim to provide long-term data on the efficacy and safety of different antiplatelet regimens, as well as to refine guidelines for the optimal duration and combination of therapies. The integration of genetic and pharmacogenomic data may also enhance the personalization of antiplatelet therapy, leading to better patient care and outcomes.

DECLARATIONS

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process, including reviewers' selection, manuscript handling, and decision making, while the other authors have declared that they have no conflicts of interest.

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