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Pharmacological and Nonpharmacological Interventions for Prevention and Management of Pulmonary Embolism in Patients with Cardiac Comorbidities: A Literature Review

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ABSTRACT

Pulmonary embolism (PE) presents significant healthcare challenges and potentially life-threatening consequences, demanding a nuanced approach to management. This systematic review aimed to critically evaluate available pharmacological and nonpharmacological interventions for PE, analyzing their efficacy and safety in patients with cardiac comorbiditie, ranging from traditional anticoagulants like warfarin to novel direct oral anticoagulants (DOACs) or thrombolytic therapies is commercially available options with varying benefits and limitations. Nonpharmacological interventions, including catheter-directed therapies and embolectomy, provide alternative avenues, especially when thrombolysis is contraindicated despite advancements, and gaps persist, particularly in reconciling efficacy with safety profiles and optimizing resource allocation. Addressing these challenges necessitates a multidisciplinary approach, integrating clinical expertise with evidence-based practices. By exploring these interventions' intricacies and implications, this review aims to inform clinicians, researchers, and policymakers, fostering improved care pathways and better outcomes for patients facing the complex landscape of PE management.

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INTRODUCTION

A pulmonary embolism (PE) arises from a blockage within the lung's arteries, often caused by a blood clot originating from a vein, typically deep vein thrombosis (DVT). PE is potentially life-threatening; prompt diagnosis and treatment can improve outcomes. PE symptoms include worsening of breath and chest pain exacerbated by movement, coughing, eating, and fainting. If PE is developed, it can be detected with signs that may include leg swelling, dizziness, coughing up blood, irregular heartbeat, and excessive sweating, but proper diagnosis tests are needed. For treatment, Anticoagulant therapy, like warfarin, is frequently prescribed post-diagnosis to prevent future clotting events. PE recurrence risks remain uncertain, but research indicates over 22% recurrence upon cessation of anticoagulants. A PE can precipitate cardiac arrest due to disruptions in the heart's electrical signals. Immediate intervention, often utilizing tissue plasminogen activator (tPA), is crucial for restoring normal heart rhythm and dissolving clots[1]. Statics suggest that in the United States, pulmonary embolism affects 1 in 1000 persons annually. Autopsy studies reveal up to 900,000 fatal and nonfatal events yearly. It ranks third among causes of hospital deaths and contributes to 70% of cases. Pediatric incidence remains low but is still associated with significant morbidity, contributing to 30% of deaths in affected children. Black individuals exhibit higher incidence rates compared to

whites, but Asian/Pacific Islanders/American Indian patients have shown lower risk. Male mortality rates are 20-30% higher, though pulmonary embolism incidence in younger patients is higher in females. Anticoagulant treatment reduces mortality to under 5% [2]. The economic burden of pulmonary embolism (PE) in the USA is substantial, with annual costs estimated between $7 to $12 billion[3], which is quite challenging. Incident cases of venous thromboembolism (VTE), including PE, result in preventable medical costs averaging around $20,000 per case[3]. Mean reimbursed costs for a PE event in the USA are approximately $9,566[4]. The study aims to evaluate pharmacological and nonpharmacological interventions for preventing and managing pulmonary embolism (PE) in cardiac patients, analyzing treatment options with their efficacy and safety.

METHODOLOGY

We employed a systematic approach to evaluate pharmacological and nonpharmacological interventions for the management of pulmonary embolism (PE) in cardiac patients. We identified relevant literature from three electronic databases: PubMed, Embase, and Scopus.

Keywords: We utilized primary and secondary keywords for pulmonary embolism (PE) management in cardiac patients. Primary keywords *were pulmonary embolism, anticoagulants, thrombolytics, nonpharmacological interventions, and cardiac patients*, which formed the core of the search strategy. We supplemented it with secondary keywords, including *pulmonary thromboembolism, deep vein thrombosis, and specific medication names like heparin and warfarin*. Boolean operators such as AND, OR, and NOT were employed to refine search results, ensuring relevance while avoiding irrelevant studies. Medical Subject Headings (MeSH) terms were integrated, encompassing concepts like anticoagulants/therapeutic use and thrombolytic therapy/methods.

Mesh Terms:

("Pulmonary Embolism"[Mesh]) AND ("Anticoagulants"[Mesh] OR "Thrombolytic Therapy"[Mesh] OR "Anticoagulant Therapy"[Mesh])

("Pulmonary Embolism"[Mesh]) AND ("Treatment"[Mesh] OR "Evidence-Based Medicine "[Mesh] OR "Pharmacological OR Non Pharmacological interventions"[Mesh])

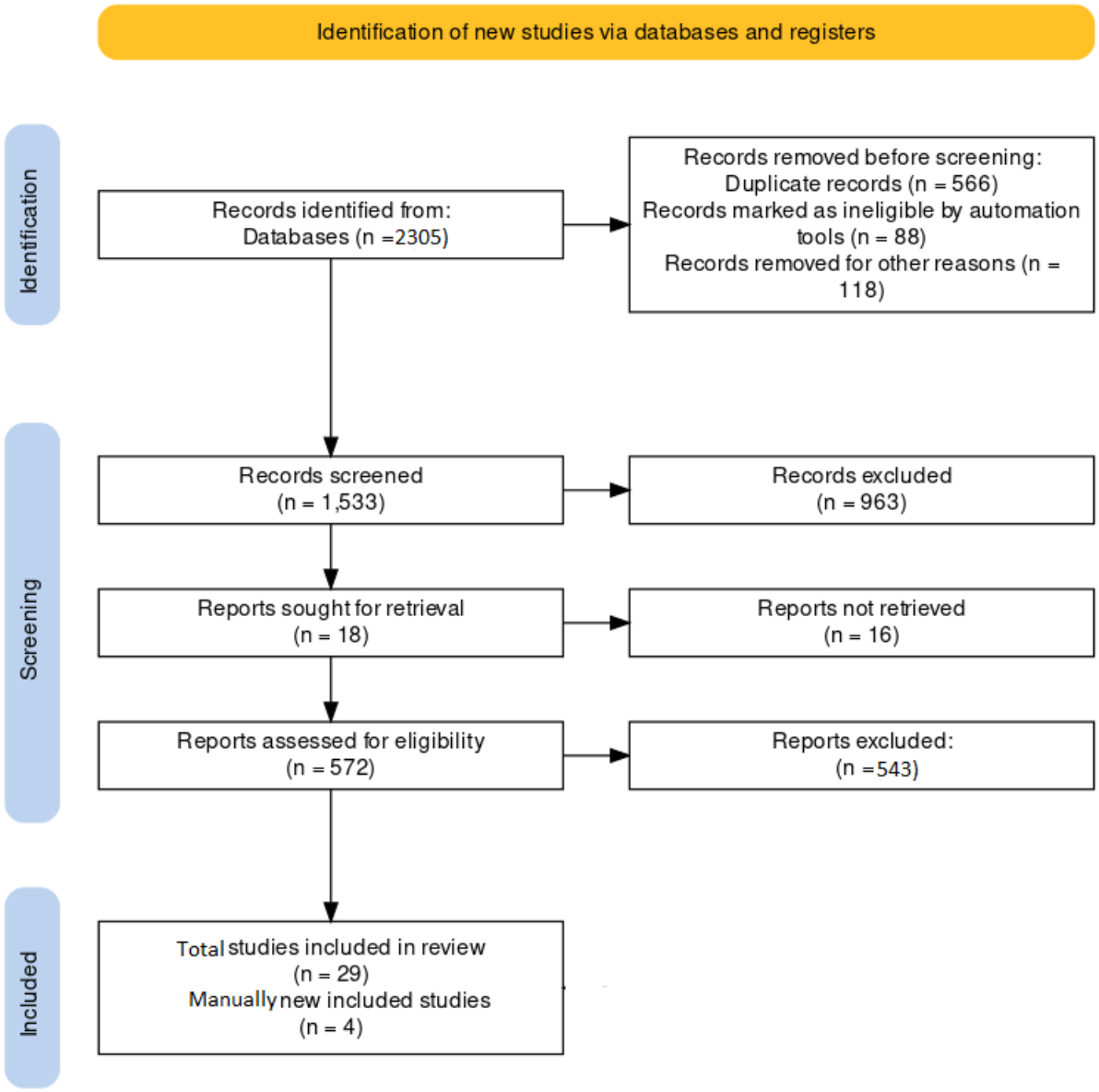
("Pulmonary Embolism"[Mesh]) AND ("NONPHARMACOLOGICAL OR PHARMACOLOGICAL interventions "[Mesh])

Following the above method, we identified and explored diverse literature sources across an electronic database and captured a broad spectrum of medications and evidence while evaluating the efficacy and safety of pharmacological and non-pharmacological treatments in PE management among cardiac patients.

Inclusion and Exclusion Criteria

This paper includes peer-reviewed literature published in English between 2010 and 2024, focusing on medications, clinical trials, literature reviews, systematic reviews, meta-analyses, and observational studies. We evaluated titles where we identified pharmacological and nonpharmacological interventions being discussed in the paper for pulmonary embolism (PE) in cardiac patients. We included those papers with interventions that included anticoagulants, thrombolytics, and nonpharmacological strategies such as catheter-directed therapies, embolectomy, and inferior vena cava filter placement. The scope of the inclusion criteria encompassed both acute and chronic management of PE. All other papers that do not discuss treatments and management were discarded. Incomplete papers, grey literature, unpublished papers, and reports were excluded.

RESULTS



Description: Above PRISMA flow diagram illustrates the study selection process, by searching on 3 different databses, 2,305 citations were found. After removing duplicates and automated exclusions, 1,533 records were screened and in this step, we made judgements bases on titles. Following exclusions, 572 reports were assessed for eligibility and we accessed abstract and then readed full body text when required and we ended up with 543 exclusions. Ultimately, 29 studies from databases were included in this process and 4 manually included studies were considered. This paper contains 33 total studies.

Pharmacological Agents for PE

a. Anticoagulants

Anticoagulants are medicines that do not allow blood clot formation and thus prevent the occurrence of such a dangerous condition as thrombosis, which is clot formation in blood vessels[6]. They exert their action by interrupting the complicated homeostasis process involving protein clotting factors. Such drugs can be field-specific as they inhibit the activity of particular clotting factors such as thrombin (factor IIa) or factor Xa, or, alternatively, they can prevent these factors from being produced altogether. Fibrin is the structure-forming protein that is the cornerstone of blood clots, and anticoagulants' effect is an inhibition of the chain of events that cause the protein formation[7]. Anticoagulants like warfarin or apixaban are commonly prescribed for heart conditions and pulmonary embolism to prevent blood clots.

b. Low Molecular Weight Heparins (LMWHs)

LMWHs are low-weight compounds, and it has been demonstrated that they reduce thrombus formation in patients[8]. A group of LMWHs, including enoxaparin and dalteparin, have a constant anticoagulant effect due to their ATWs (Average Thousand Molecular Weight) being relatively uniform and less than 50% of UFH (Unfractionated

Heparin) [9]. While subcutaneously administered and weight-based dosed, they are particularly recommended for cancer activity and pregnancy, although recent research now shows that Direct Oral Anticoagulants (DOACs) can potentially treat cancer-related venous thromboembolism (VTE) [10]. It is routine for antiXa to monitor the effect of enoxaparin in severe renal impairment or obesity. Regarding the levels of anti−Xa for dalteparin, it is not that dissimilar, especially in morbidly obese or renal compromised patients[10, 11]. Although LMWHs have a lower potential for HIT than UFH, one should still be careful and monitor the situation as well as, particularly in patients with a HIT history. Although they are not HIT antibodies by UFH as powerful triggers, LMWHs also play an efficient role in platelet activation [12]. LMWHs such as enoxaparin and dalteparin are utilized in pulmonary embolism and heart conditions for their predictable anticoagulant effect, weight-based dosing, and lower risk of complications compared to UFH.

c. Direct Oral Anticoagulants DOACs

The emergence of new Direct Oral Anticoagulants (DOACs) has rearranged the whole concept of anticoagulant therapy, making alternatives to traditional oral agents, like warfarin. DOACs such as apixaban, rivaroxaban, dabigatran, and edoxaban, along with different pharmacokinetics and clinical applications, come to the forefront. Apixaban, the oral direct factor Xa inhibitor, is approved for other diseases, including health risk reduction after patients have had knee and hip replacement surgery, prophylaxis of deep vein thrombosis, and treatment of deep vein thrombosis and pulmonary embolism. It also performs a vital role with antithrombotic therapy following acute coronary syndrome (ACS) or percutaneous coronary intervention (PCI) and in atrial fibrillation and heparin-induced thrombocytopenia (HIT) too [14]. Apixaban displays an oral bioavailability of about 50%, mainly the tiny intestine absorption [15]. The metabolism activity in the intestine and liver is carried out through CYP3A4 pathways. Prolonged downstream of this process leads to an increased risk of thrombotic events. To highlight, patients taking apixaban can develop an epidural or spinal hematoma while being subjected to neuraxial anesthesia or spinal puncture, which can bring about permanent paralysis.

Like the direct factor Xa inhibitor, Rivaroxaban is highly competitive, with high oral bioavailability ranging from 80% to 100%. This is characterized by a terminal halflife ranging from 5 to 9 hours, which may be prolonged in elderly patients. Rivaroxaban elimination takes place through renal filtration. Assistance should be given to those with impaired renal function. It also shares a similar site of metabolism via the CYP3A4 systems and ekes out a black box warning regarding the risk of thrombotic events upon early discontinuation [16]. The patients receiving therapy with rivaroxaban during neuraxial anesthesia, meningeal blockage, or spinal puncture are increasing the chance of epidural or spinal hematomas with conceivable long-term consequences. Dabigatran, a direct thrombin (IIa) inhibitor, may be used alone; however, some indications require bridging with unfractionated heparin or low molecular weight heparin. So, the problem with drug interactions, especially with those drugs that increase bleeding risk or change P-glycoprotein (Pgp) activity,, is that they should be considered very cautiously. Renal impairment and Pgp inhibition impact dabigatran exposure, warranting dose adjustments and cautious use, especially in elderly patients. Dabigatran is contraindicated in patients with mechanical heart valves due to increased thromboembolic risk observed in clinical trials. Edoxaban is approved for the treatment of PE and prophylaxis against stroke and systemic embolism in nonvalvular atrial fibrillation because it offers comparable efficacy to warfarin with reduced bleeding risk. Its approval stems from the Hokusai VTE study, which demonstrates noninferiority to warfarin. Unlike other DOACs, it lacks indications for specific prophylactic measures in joint replacement surgeries[9].

d. Unfractionated Heparin (UFH)

Unfractionated Heparin (UFH) is a parental anticoagulant obtained from porcine or bovine tissues. It shows its effects by inhibiting thrombin (IIa) and factor Xa via antithrombin. UFH is given using 80 units/kg IV bolus. After that, 18 units/kg/hour infusion takes place. Short HALFLIFE, 0.5 to 1.5 hours, of this drug is incredibly beneficial for patients with pulmonary embolism (PE), specifically when high bleeding risk, critical illness, or surgery is concerned [6]. Through metabolism by the reticuloendothelial system and clearance in patients with poor renal function (creatinine clearance of < 30 mL/min), UFH is efficient. Yet, the side effects of heparin-induced thrombocytopenia (HIT), which is relevant to heparin and affects up to 7% of patients with a high mortality rate – 20% to 30% – have been reported. LMWH is associated with a higher risk of HIT compared to UFH. HIT stems from IgG fabrication towards the heparin/PF4 complex on platelets, which is the reason for platelet activation and thrombi formation. Platelet assessment is highly advised with UFH administration[9].

Therefore, UFH can not only affect patients by causing reactions such as thrombocytopenia and major bleeds, including intracranial and gastrointestinal bleeds. In addition, endocrine diseases cause a substantial reduction in bone density that results in adult patients having experimental bone fractures in some cases. Anticoagulants are distinctive in terms of the effectiveness and disadvantages they offer in managing PE patients with cardiac comorbidities. Unfractionated heparin (UFH) has a rapid onset of action but could be problematic for patients who may develop HIT. Enoxaparin and Dalteparin give up the same molecular weight but still increase HIT risk. For instance, oral anticoagulants like Edoxaban, Apixaban, Dabigatran, and Rivaroxaban propose oral dosage and only have a few monitoring requirements, but they do have the risk of

bleeding. On the other hand, taking kidney function and bleeding risk into account is most important. Careful surveillance of complications like bleed and thrombocytopenia is imperative, especially for those with preexisting cardiac disorders[17].

Vitamin K Antagonists (VKAs)

Vitamin K Antagonists (VKAs), exemplified by warfarin, are pivotal in treating pulmonary embolism (PE), especially in patients with severe renal insufficiency or financial constraints precluding Direct Oral Anticoagulants (DOACs) [16]. Warfarin therapy necessitates International Normalized Ratio (INR) monitoring due to its variable response. Initial treatment may induce a transient hypercoagulable state, mandating bridging with UFH, LMWH, or fondaparinux until INR stabilizes within the therapeutic range of 2 to 3 doses[18]. Warfarin's efficacy is influenced by vitamin Krich foods and numerous drug interactions, necessitating patient education and close monitoring. Additionally, its interaction with CYP2C9, 2C19, 1A2, and 3A4 underscores the importance of medication reconciliation. Conversely, Fondaparinux selectively inhibits factor Xa, making it a practical alternative with no need for routine monitoring but lacking a specific reversal agent[13].

Table 1. Anticoagulants Types, Names, Dosages Limitations and Considerations

Name(s)	Type	Dose	Special Considerations
Unfractionated Heparin (UFH)	Parenteral	80 unit/kg IV bolus, followed by an 18unit/kg/hour infusion	Rapid onset and short halflife; preferred in PE with high bleeding risk, critical illness, or need for surgery. Metabolism and clearance through reticuloendothelial system; suitable for patients with poor renal function (CrCL < 30 mL/min). Limited drug interactions. IV route is preferred in shock/hypotension due to absorption variability from subcutaneous tissues.
Enoxaparin	Subcutaneous	1 mg/kg sub Q twice daily	Standard dosing for thromboprophylaxis and treatment of DVT/PE.
Dalteparin	Subcutaneous	200 IU/kg/day sub Q for one month, followed by 150 IU/kg/day subcutaneously for months 2 through 6	Maximum of 18,000 IU per day.
Fondaparinux	Subcutaneous	<50 kg: 5 mg sub Q daily 50–100 kg: 7.5 mg sub Q daily >100 kg: 10 mg sub Q daily	Initiate warfarin within 72 h and give concomitantly for at least 5 days.
Edoxaban	Oral	60 mg PO once daily; 30 mg once daily if body weight ≤60 kg	Not for use in patients with CrCl > 95 mL/min. Dose adjustment after 5 to 10 days of initial therapy with parenteral anticoagulant.
Apixaban	Oral	10 mg PO twice daily for 7 days, followed by 5 mg twice daily	
Rivaroxaban	Oral	15 mg PO twice daily × 3 weeks, then 20 mg once daily × at least 6 months	Take with food to improve absorption.
Dabigatran	Oral	150 mg PO BID; 110 mg BID for patients ≥80 years	Dose adjustment after 5 to 10 days of initial therapy with parenteral anticoagulant. Reduce dose to 110 mg BID for patients ≥80 years or ≥75 years with at least one bleeding risk factor.

Monitoring of Anticoagulants

Once the appropriate anticoagulant medication has been chosen, its effectiveness and safety must be monitored. Factor Xa assay (antiXa) uses other methods for measuring unfractionated heparin (UFH), while aPTT is the nutritional one. The international normalized ratio (INR) is the

recommended monitoring of warfarin therapy. DOACs (Direct oral anticoagulants) are usually not required for routine monitoring, but the anti-factor Xa activity can help exclude clinically significant levels. Dabigatran may be assessed using qualitative and quantitative methods through thrombin time (TT) and aPTT. The selection of the monitoring method depends on a patient's traits and the anticoagulant drug being used [19].

Bleeding is the most salient issue of the anticoagulation therapy use, among them that is intracranial and gastrointestinal bleeds. The importance of anticoagulants targeted reversal agents must be considered. Protamine sulfate provides a UFH and LMWH reversal and is administered cautiously due to possible side effects [20]. The short life span of UFH will see it normalize itself without the reversal drugs. Nonetheless, the activation of LMWH is only partly reversed by protamine. Fondaparinux is made of larger molecules; thus, protamine does not affect it. For bleeding of warfarin type, there are advantages such as potency and practicality of 4FPPCC contrary to FFP. It can effectively substitute the Kdependent vitamin clotting factors and be given in no time. Idarucizumab is dabigatran reversal specific, and andexanet alfa is the complete factor Xa inhibitor antidote. If idarucizumab or andexanet alfa is unavailable, PCC or pack may be used as a substitute. The charcoal might be activated in a recent DOAC ingestion [21]

For fondaparinux inhibition, both factor VII recombinant or aPCC present positive results, but it is still necessary to advance the human data. Lysis of thrombolysis for PE, especially in emergency cases, is not advisable due to the high risks of bleeding and good outcomes. Alteplase is considered the FDA-authorised thrombolytic therapy with weight-based regimens. Intraarterial directed alteplase, although reported effective, is not yet FDA-approved. For off-label use, patients with PE are being treated with tenecteplase and reteplase [21].

Thrombolytic Therapy For Pulmonary Embolism

Thrombolysis is a therapeutic option that is both disputed and vital in pulmonary embolism (PE), particularly in unstable or high-risk subjects. The primary use is treating acute massive or submassive PE with RV strain. Criteria for using it are low blood pressure or even rapid risk of it, which is caused by some clinical signs. According to recent studies, thrombolysis could reduce mortality by a few percentage points in intermediate-risk pulmonary embolisms. However, major bleeding should also be studied. Strategies including thrombolytic doses of lower specific markers like CKMB levels can help refine treatment and predict outcomes. In that regard, choosing the thrombolytic agent that acts the fastest is crucial, often relying on off-label drugs that aim to treat patients faster. The coadministration of unfractionated heparin (UFH) and thrombolytics are not recommended, and anticoagulation follows thrombolysis; then, UFH should be delayed till clotting periods are regular. Advanced cases attended by cardiac arrest and rapid administration of thrombolytic agents, including alteplase, can represent a lifesaving option. At present, there are three thrombolytic FDA-approved agents indicated for rapidly resolving clots related to PE, namely, alteplase, urokinase, and streptokinase, with more investigations being carried out to discover additional options such as Tenecteplase [5,22,23]

Table 2. Thrombolytic Regimens: Names, Types, Doses, and Special Considerations

Name	Type	Dose	Special Considerations
Alteplase	Intravenous	100 mg continuous infusion over 2 hours	Heparin drip must be discontinued during alteplase infusion.
Accelerated	Intravenous	<67 kg: 15 mg IV bolus, then 0.75 mg/kg over 30 mins (max 50 mg), followed by 0.50 mg/kg over 60 mins (max 35 mg). >67 kg: 15 mg IV bolus, then 50 mg over 30 mins, followed by 35 mg over 60 mins.	Some centers prefer accelerated 90-minute regimens for faster action, safety, and efficacy.
Urokinase	Intravenous	4400 U/kg loading dose over 10 mins, then 4400 U/kg/hr continuous infusion over 12 hours	Suitable for patients with poor renal function (CrCL < 30 mL/min).
Streptokinase	Intravenous	250,000 U loading dose over 30 mins, then 100,000 U/hr continuous infusion over 1224 hours	Notably, its use may be limited by potential allergic reactions and a higher risk of non-antibody mediated adverse effects than other thrombolytic agents.
Reteplase	Intravenous	Two IV boluses of 10 U each, administered 30 minutes apart	Not FDA approved for PE; dosing similar to AMI treatment [5]

Antiplatelet Agents

Platelet inhibitors are also prescribed to patients to block the platelet clumping process happening in coronary artery disease (CAD)and to prevent stroke in atrial fibrillation conditions or during acute coronary syndromes. One example is an antiplatelet agent comprising medications like aspirin, clopidogrel, ticagrelor, and prasugrel.

This condition, however, cannot be managed well, and in cases where both the pulmonary embolism and the coronary artery disease coexist, the condition becomes more complex and often requires the usage of both anticoagulant and antiplatelet drugs. They share the same mechanism of clot prevention as aspirin, which can result in treating pulmonary embolism (PE). Studies indicate that these medications can diminish the recurrence of PE and offer some therapeutic approaches for patients with cardiac pathologies by targeting the partial thrombosis mechanism. In contrast, antiplatelets are thought to bring fewer benefits for the acute management of PE because their mechanism is geared more towards arterial thrombosis, the primary cause of PE, other than venous clots. Despite that, in particular clinical situations, for people who are not taking blood thinning medications, antiplatelet therapy might be used alone or together with anticoagulants in the treatment of PE [24].

Table 3 Antiplatelet Agents for Pulmonary Embolism

Medication	Dosage	Route	Considerations and Limitations
Aspirin	75 mg once daily	Oral	Considered less effective for venous thromboembolism compared to arterial thrombosis [25]
			It may attenuate pulmonary vasoconstriction and bronchospasm associated with PE by inhibiting thromboxane A2.
Clopidogrel	75 mg once daily	Oral	Genetic factors may have delayed the onset of action and variability in response.
			Potential for increased bleeding, particularly when combined with other antiplatelet agents or anticoagulants.
Ticagrelor	Loading dose: 180 mg once,	Oral	Requires caution in patients with a history of intracranial bleeding or severe hepatic impairment.
	Maintenance dose: 90 mg twice daily		Associated with a higher risk of bleeding compared to clopidogrel.
Prasugrel	Loading dose: 60 mg once,	Oral	Contraindicated in patients with prior stroke or transient ischemic attack (TIA) due to increased bleeding risk.
	Maintenance dose: 10 mg once daily		Requires caution in patients with a history of intracranial bleeding or severe hepatic impairment.
			Limited data is available regarding its use in pulmonary embolism; further research is warranted.

Non-Pharmaceutical Approach Pulmonary Embolism

1. **CatheterDirected Therapies:** direct therapy techniques engage clot dissolve in the arteries of the lungs to dislodge the blood clots. The thrombus can be eliminated with a jet injection of saline, ultrasonic waves, or direct mechanical destruction using catheters inserted into the concerned artery. On the other hand, catheter-directed therapies require the use of smaller doses of thrombolytics than systemic thrombolysis for optimal results, hence reducing the risk of bleeding, especially where the bleeding risk is higher, such as in older people [26].

2. Suctioning: Suctioning, indeed thrombectomy, is frequently used to remove a clot or clot fragment in the control part of the small blood vessels. This approach cleans the obstruction and enhances artery blood flow, especially in patients with acute pulmonary embolism [27].

3. Inferior Vena Cava Filter (IVCF) Placement: Conversely, IVCFs are fitted where other anticoagulation therapy is contraindicated in patients with acute venous thromboembolism (VTE). The usefulness of the IVCF is in the fact that this device catches big emboli from the feet to the pulmonary circulation and thus prevents pulmonary embolism. The application of IVCF in acute pulmonary embolism patients having already taken anticoagulant drugs is not recommended on account of insufficient evidence of favorable outcomes of such treatment and also possible complications of deep vein thrombosis (DVT) without any reduction in mortality [28].

4. Percutaneous Transcatheter Interventions: The fundamental ones are significant for people with sudden pulmonary embolisms of the submassive or massive sort. They are generally taken when there is no room for one when thrombolysis doesn't work or cannot be used, when no

surgical thrombectomy is available, or when it is not the first choice. A mixture of these approaches by implementing both techniques is also evolving. Catheter-directed thrombolysis or pharmacomechanical thrombolysis is noted for patients in good condition to take thrombolysis, which typically reduces pulmonary artery pressure, lowers proper ventricle burden, improves systemic circulation, and enhances adequate ventricle recovery. Many options fall under the percutaneous intervention category, including thrombus aspiration for removal, fragmentation, or hemolytic thrombectomy, which all utilize a different way of clearing the blockage and reducing thrombus load [29].

5. Surgical Embolectomy: Emergency pulmonary embolectomy with cardiopulmonary bypass was first undertaken and successfully performed on a patient in 1924. This procedure has gained greater prominence as a proven approach to thrombolysis and other treatments for this condition, which are not feasible in patients with various contraindications. It is indicated for patients with RAT thrombus and walked pneumonia under right atrial thrombolysis. In cases of thrombolytic agent resistance among patients, immediate surgical embolectomy is shown; this is preferably timed before the shock stage of cardiogenic. Despite this, surgical mortality (loss of life) remains high in certain instances, and it is even possible for a patient to stay in recuperation for up to several weeks following the procedure [30].

6. Pulmonary Endarterectomy (PEA): PEA remains the preferred chronic thromboembolic pulmonary hypertension (CTEPH) treatment option. The imperative requirement for operability embraces the wound accessibility and operability of the blood clots of the patients before the operation. Despite the progress in endovascular advancement, PEA is the current gold standard that sustainably reduces symptoms and helps in immediate cardiac output surge. Patients who are not eligible for surgery or suffering persistent pulmonary hypertension, even after pulmonary endothelial angioplasty, usually are not promising. Therefore, alternative methods like balloon pulmonary angioplasty are still in development [31].

7. Other therapies Proper oxygen supplementation helps these patients compensate for oxygen demand in their body tissues because they are at a higher risk for hypoxemia. Oxygen therapy serves to reduce the effects of hypoxia, and it helps the heart avoid further strain and prolong cardiac arrest. Patients with acute PE are likely to be put on bed rest as the situation can worsen if they get involved in activities that can increase embolization and cause the heart muscles to work hard. Nevertheless, the initial movement is recommended according to patients' condition improvement when they can be stable to avoid complications like deep vein thrombosis (DVT), pulmonary complications, and many more associated with immobilization. Compression stockings and pneumatic compression devices that alternate the compression at intervals are typically used in patients prone to DVT development who have PE. These devices help relieve the stagnant blood in the veins of the lower limbs through the reduction in the occurrence of clots. Personal efforts to achieve behaviors that result in weight loss, smoking secession, regular exercising, and physical activity are among strategies that can substantially decrease the risk factors for PE, such as smoking, obesity, and a sedentary lifestyle. The awareness of the patients is the factor that keeps transient phlebitis from becoming chronic and helps in controlling the existing complications. Patient education about PE signs and symptoms, proper compliance with anticoagulant therapy, lifestyle adaptation, and DVT recurrence prevention strategies can give them complete control over their care and optimize results. It is paramount that there be frequent aspect of followup with healthcare providers. Simultaneously, treatment procedures should be adjusted if any medical concerns arise, and complications should also be addressed. No matter how good surgical treatment is for patients with PE, an appropriate diet is essential in their recovery process. Nutritional supplementation may be needed for individuals who are reported to be experiencing weight loss, malnutrition, and poor dietary status secondary to the absence of appetite, higher metabolic rates, or other underlying health complications. Nutritionists or dietitians, for instance, possess the skillfulness to tailor individualized nutrition plans that are designed in such a way that they can meet the needs of PE patients. To survive through the physical and emotional effects of stroke becomes a complicated task both for the patients and their relatives. Such psychological care services as counseling, support groups, and referrals to professional psychologists are meant to assist people in managing any psychological concerns such as stress, anxiety, depression, and problems associated with the diagnosis and treatment of postexposure stress [32, 33.]

CONCLUSION

Overall, the management of PE patients with cardiac comorbities becomes more complicated with various interventions, ranging from emergency to long-term monitoring. While pharmacological interventions like anticoagulants and thrombolytics present several promising options, their efficacy can have to be weighed against possible risks, particularly among high-risk groups, such as cardiac patients. Nonpharmacological approaches, though hopeful, may face some challenges that need careful investigation regarding identifying their place in the overall treatment interventions. Furthermore, the cost linked to PE demonstrates why it should be done through efficient work and low-cost methods. While making progress, the gap between evidence-based practices and clinical use should be the focus of future deliberations. A multidisciplinary collaboration involving cardiologists, pulmonologists, hematologists, and interventional radiologists must seek

personalized treatment solutions, prioritizing patient interest and results. Besides that, the ongoing investigation should always be remembered because this is the only way to fill the gaps, regulate the treatment algorithms, and search for new medications. Through this, the patient-centric approach that considers clinical evidence and is guided by expertise is the primary resource for the team embarking on the complex maze of PE management. Novel strategies can improve results, lower morbidity and mortality, and increase the quality of life for people facing this disease.

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