CHEST

Interhospital Transfer of Patients With Acute Pulmonary Embolism

Challenges and Opportunities

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Acute pulmonary embolism (PE) is associated with significant morbidity and mortality. The management paradigm for acute PE has evolved in recent years with wider availability of advanced treatment modalities ranging from catheter-directed reperfusion therapies to mechanical circulatory support. This evolution has coincided with the development and implementation of institutional pulmonary embolism response teams (PERT) nationwide and internationally. Because most institutions are not equipped or staffed for advanced PE care, patients often require transfer to centers with more comprehensive resources, including PERT expertise. One of the unmet needs in current PE care is an organized approach to the process of interhospital transfer (IHT) of critically ill PE patients. In this review, we discuss medical optimization and support of patients before and during transfer, transfer checklists, defined roles of emergency medical services, and the roles and responsibilities of referring and receiving centers involved in the IHT of acute PE patients. CHEST 2021; 160(5):1844-1852

KEY WORDS: interhospital transfer; pulmonary embolism transfer; transition of pulmonary embolism care; transfer of pulmonary embolism; patients

ABBREVIATIONS: BACS = bleeding, age, cancer, syncope; CTPA = computed tomographic pulmonary angiography; ESC = European Society of Cardiology; HFNC = high-flow nasal cannula; IHT = interhospital transfer; LMWH = low-molecular-weight heparin; PE = pulmonary embolism; PEEP = positive end-expiratory pressure; PERT = pulmonary embolism response team; PESI = Pulmonary Embolism Severity Index; PVR = pulmonary vascular resistance; RV = right ventricular; sPESI = Simplified Pulmonary Embolism Severity Index; STEMI = ST-elevation myocardial infarction; UFH = unfractionated heparin

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Venous thromboembolism has long been recognized as a major public health issue.[1,2] Pulmonary embolism (PE) is the third leading cause of cardiovascular mortality in the United States.[3] However, unlike acute myocardial infarction, stroke, and trauma, strategies for initial care and support of PE patients have not been standardized. For example, thrombolytic use in rural settings is employed significantly less compared with its use in metropolitan hospitals.[4] The Centers for Disease Control and Prevention Wide-Ranging Online Data for Epidemiologic Research database demonstrates mortality disparities in rural vs urban settings, especially in the South.[5-7] Survival of the most critically ill PE patients is often dependent on early recognition and diagnosis, expedient decision-making, and appropriate execution of interventions. Management must be expeditious, beginning at triage and continuing through the point of definitive care.[8,9] The pulmonary embolism response team (PERT) aims to provide time-sensitive, multidisciplinary individualized care to critically ill patients with acute PE.[10] Data from large-scale multisite observational cohorts suggest that patients with acute PE benefit (ie, short-term 30-day mortality) when treated at high-volume centers and by experienced physicians.[11-14] The recent European Society of Cardiology (ESC) guidelines for the diagnosis and management of acute PE highlights the value of PERTs for high- and intermediate-risk PEs.[15] One of the main challenges in treating these patients is the lack of access to locoregional expert PERT centers. Many patients who present to rural or community hospitals have to be transferred to receive advanced care, often guided by the receiving centers' PERTs.[16]

Literature from other acute medical conditions (ie, ST-elevation myocardial infarction [STEMI] and stroke) suggests that patients requiring interhospital transfer (IHT) have significantly higher morbidity and mortality compared with patients admitted directly to hospitals with expertise and experience.[17,18] The outcomes of transferred patients are directly affected by many factors, including the care rendered and the duration of time spent at the initial hospital. A timely transfer may be a key element that improves PE outcomes, similar to what is described in other acute medical crises (ie, STEMI, stroke, trauma).[19-22] In acute PE, the comparative survival of patients transferred for advanced care vs those directly admitted to hospitals with PERT teams rarely has been studied.[23] The PERT Consortium consensus practice document discusses the treatment algorithms for acute PE in detail.[24] The focus of this review is to provide step-by-step granularity in assessing critically ill patients with PE and achieving successful IHT. Figure 1 provides a road map for the IHT process of acute PE.

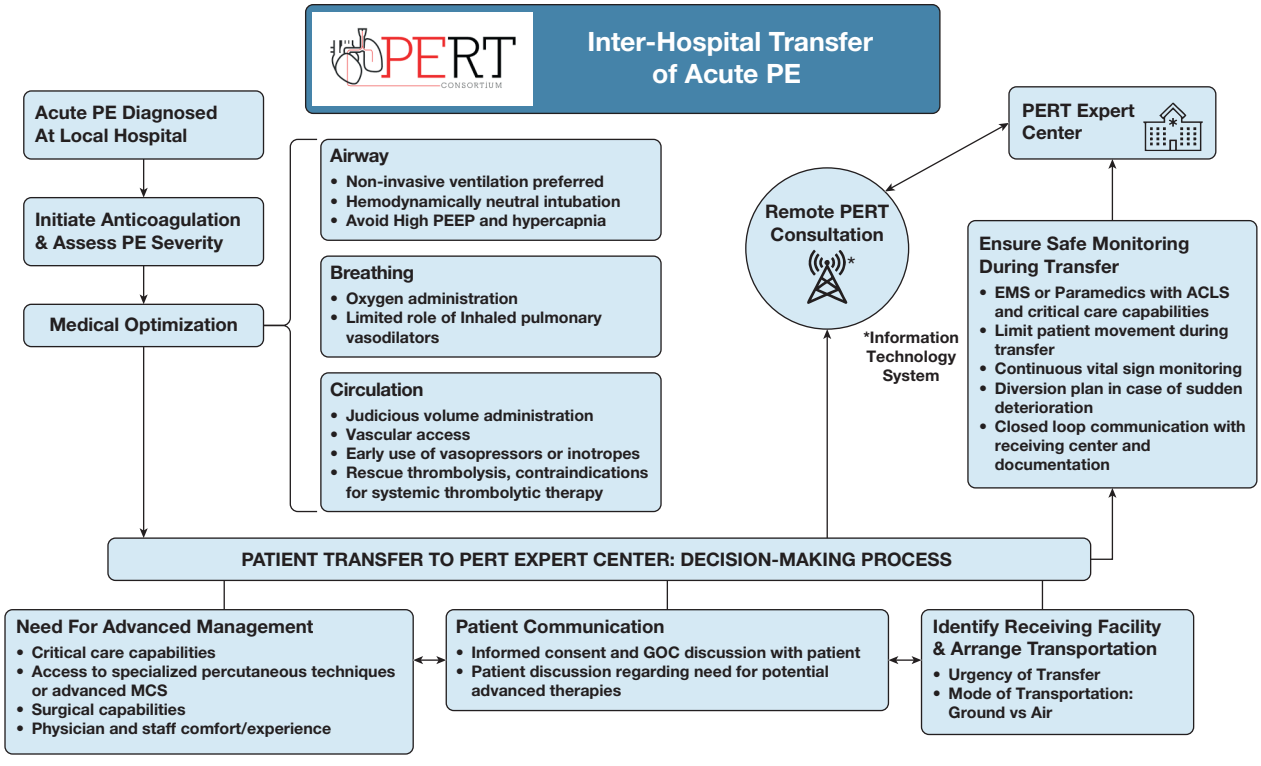

Figure 1 – Interhospital transfer of acute PE. GOC = goals of care; MCS = mechanical circulatory support; PE = pulmonary embolism; PEEP = positive end-expiratory pressure; PERT = pulmonary embolism response team.

Methods

A writing group was established by the PERT consortium from members of Clinical Protocols and Education committees in collaboration with the CHEST Pulmonary Vascular Disease network. A conceptual outline was created by P. R., D. S., and C. R. The writing group was divided into topics based on each member's expertise or interest. Each group reviewed and summarized the relevant published literature and incorporated such information into a manuscript. The final draft of the manuscript was circulated among all authors, and revisions were discussed in monthly meetings of protocols, educational, and the CHEST Pulmonary Vascular Disease network committee.

Initiation of Anticoagulation and Risk Stratification

Timely, effective therapeutic anticoagulation is the mainstay of treatment in acute PE.[25] Anticoagulation, barring absolute contraindications, should be started as soon as PE is diagnosed regardless of transfer status.[15,24,26] Many PERTs favor low-molecular-weight heparin (LMWH) as the anticoagulant of choice, based on achieving predictable therapeutic levels within 3 to 4 hours of administration.[27] Additionally, for patients being considered for IHT, LMWH simplifies the anticoagulation plan with a single-dose administration every 12 hours instead of a continuous parenteral drip of unfractionated heparin (UFH) that requires drug monitoring and titration. However, the decision on anticoagulation (UFH vs LMWH) can be individualized after considering the patient's renal function, weight, risk of bleeding, and the anticipated need for interventional procedures. When surgical or interventional procedure or systemic lysis is planned after the transfer, LMWH regimens may be less titratable. In such cases, UFH may offer the most flexibility for interventional procedures. Overall, best practice mandates the establishment of individualized protocols to achieve rapid therapeutic anticoagulation. This includes appropriate bolus and maintenance infusion doses, as well as time metrics and specific goals after anticoagulation.

Risk stratification, most commonly following the ESC guidelines, is crucial in guiding the initial approach to patients with acute PE and can be assessed through clinical, right ventricular (RV) imaging by computed tomographic pulmonary angiography (CTPA) or echocardiography, biomarkers (troponin), and the PE severity scores (Pulmonary Embolism Severity Index [PESI] score or simplified PE Severity Index [sPESI] score) for choosing the appropriate therapy.[15] The ESC classification risk stratifies patients into low-risk, intermediate-risk (low and high) and high-risk PE. Low-risk PE patients are hemodynamically stable, without any evidence of RV dysfunction and an sPESI score of 0. Intermediate-risk PE patients have an sPESI score $\geq$ 1 or have higher PESI class (Groups 3, 4, or 5). ESC classification further risk stratifies intermediate PE into intermediate-low-risk and intermediate-high-risk group. In intermediate-low-risk PE, there is evidence for RV dysfunction on imaging (echocardiography/CTPA) or elevated cardiac troponin. These patients have either one but not both criteria. If the patients with intermediate-risk PE have evidence of RV dysfunction on imaging (echocardiography/CTPA) and elevated cardiac troponins, they are then considered intermediate-high-risk PE. High-risk PE is defined as patients who are hemodynamically unstable.

The PESI score accounts for patient characteristics (ie, age, male sex, history of heart failure, cancer, chronic lung disease) and clinical parameters (ie, heart rate, systolic BP, respiratory rate, temperature, mental status, oxygen saturation) to risk stratify patients with acute PE. The PESI score risk stratifies the patient into low-risk group (groups 1, 2) and high-risk groups (groups 3, 4, and 5). Mortality increases from 1% in the low-risk group to 24% in high-risk groups.[28,29] The PESI score is rarely calculated in clinical practice, because it is generally considered cumbersome. In contrast, the simplified PESI (sPESI) is brief, contains a limited number of easily accessible clinical parameters (ie, age, history of cancer, chronic lung disease, heart rate, systolic BP, oxygen saturation) and is therefore much more practical. The sPESI score being very sensitive identifies extremely low-risk patients who are not likely to suffer any adverse outcomes.

Assessment of RV size and function by CTPA, echocardiography, or both are essential components of risk stratification of patients with acute PE, with increased RV size and dysfunction associated with poor outcomes.[15] Troponin elevation is caused by myocardial ischemia, whereas B-type natriuretic peptide rise is attributable to RV pressure overload in the setting of acute PE. Elevation of biomarkers carries an independent risk of short-term mortality and RV dysfunction.[15,30] New emerging biomarkers, such as increased serum creatinine, lactic acid, or hyponatremia, can further aid in risk stratification.[15]

Bleeding risk is necessarily assessed in all acute PE patients when considering their candidacy for advanced interventions. BACS score (ie, recent major bleeding, age >75, active cancer, syncope) and PE-CH scores (ie, peripheral arterial disease, age > 65 years, prior stroke with residual effect, history of heart attack) have been recently developed to predict the risk of major bleeding and intracranial hemorrhage in patients considered for systemic thrombolysis.[31,32] Anticoagulation is nearly always adequate for patients with low- and intermediate-low-risk PE. Patients with intermediate-high-risk PE (RV dysfunction, sPESI $\geq$ 1, and elevated troponin or B-type natriuretic peptide) may need escalation of care, and those with high-risk PE (hemodynamic instability) usually do need it.[15]

Medical Optimization Before Transfer: The Airway, Breathing, Circulation Approach

Comprehensive PERT consensus-based PE-specific management approaches have been described previously.[24] This section focuses on the specific A (airway), B (breathing), and C (circulation) issues that may arise before or during the transfer of critically ill PE patients.

Airway (A): Severe hypoxemia may be seen in critically ill PE patients. When supplemental oxygen via nasal cannula is not sufficient to maintain adequate oxygenation, patients can be supported with noninvasive ventilation via high-flow nasal cannula (HFNC) oxygen or bilevel positive-pressure airway pressure, leaving endotracheal intubation as a last resort.[33] Urgent endotracheal intubation in critically ill patients carries a significant risk compared with elective intubation.[33] In critically ill acute PE patients, intubation can potentiate RV failure by causing a reduction in preload and an increase in afterload, which may result in cardiopulmonary arrest. In addition, some induction agents used for endotracheal intubation can reduce systemic vascular resistance and RV preload, which may further precipitate shock.

If endotracheal intubation is unavoidable, it is best performed by an experienced provider. One retrospective case series describes awake bronchoscopic-guided intubation while patients receive HFNC oxygen in patients with pulmonary hypertension.[34] In this series of nine patients, ketamine, fentanyl, and midazolam were the most commonly used induction agents, and all were used at lower doses to avoid hypotension.[34] In a recently performed, systemic review of 892 critically ill adult patients, ketamine use was associated with a reduction in propofol infusion rate (mean difference in dose, −699 μg/min; 95%CI, −1169 to −230; P = .003). Ketamine was not associated with increased mortality, vasopressor dependence, or hospital length of stay.[35] Using a minimal or reduced dose of

TABLE 1] Vasopressors/Inotropes Effects on Right Heart[36,40,41]

Drug	Receptor	Hemodynamic Effects	Comments
Vasopressor			
Norepinephrine	$\alpha_1 > \beta_1$	↑CI, PVR ↑↑SVR ↑HR	The β_1 effect has shown to improve PA/RV coupling in animal models; improves RV myocardial oxygen delivery
Epinephrine	$\alpha_1 / \beta_1 > \beta_2$	↑↑CI, SVR, HR; PVR neutral	Improves CO without increasing PVR in animal study; improves RV contractility in septic shock study
Phenylephrine	α_1	↑↑PVR, ↑SVR	Increases PVR, increases right coronary artery perfusion, no effect on RV contractility; can cause reflex bradycardia
Vasopressin	V1	Low-dose: ↓PVR, ↑↑SVR High-dose: ↑PVR, SVR	At low dose causes pulmonary vasodilation; at high dose increases coronary and pulmonary arterial vasoconstriction
Inotropes			
Dobutamine	β1 > β2	↑↑CI, ↑HR	At low dose improves PA/RV coupling in animal studies; increases myocardial contractility, decreases PVR and SVR; causes vasodilation and hypotension because of β2 effects, increased PVR, and tachycardia at high doses
Dopamine	Low dose: D > β1 Medium dose: D = β1 > α1 High dose: α1, β1, D	↑CI, SVR, no increase in PVR (at low to medium doses), ↑↑HR	Increases CO and CI without increasing PVR at lower dose; increases risk of arrhythmogenic events and death in cardiogenic shock
Milrinone	PDE-3 inhibitor (increase cAMP)	↓PVR, ↑↑CI, ↑HR	Improves inotropy and causes pulmonary vasodilation, augments RV function; tachyphylaxis is common with long-term use; causes mild tachycardia

α1 = alpha-1 adrenergic; β1 = beta-1 adrenergic; β2 = beta-2 adrenergic; cAMP = cyclic adenosine monophosphate; CI = cardiac index; CO = cardiac output; D = dopamine; HR = heart rate; PA = pulmonary artery; PVR = peripheral vascular resistance; PDE-3 = phosphodiesterase-3; RV = right ventricle; SVR = systemic vascular resistance; V_1 = vasopressin 1.

propofol is very helpful because propofol along with endotracheal intubation will lead to a decrease in preload, which could result in cardiac arrest in the setting of acute PE.

Etomidate, opioids, and benzodiazepines are rapid-acting sedatives commonly used during induction with less profound hemodynamic effects compared with general anesthetics (ie, propofol). However, opioids can act synergistically with other sedatives, such as propofol, and exacerbate hypotension.[36]

In critically ill patients, rapid sequence intubation has been shown to increase the success rate of intubation.[37] During the peri-intubation period, avoiding hypoxemia and hypercapnia is important, because the pulmonary vasculature is particularly susceptible to reactive vasoconstriction. Moreover, minimizing time in the supine position has the theoretical advantage of avoiding atelectasis, which can increase pulmonary vascular resistance (PVR) and worsen RV afterload.

Once intubated and mechanically ventilated, a reasonable goal in managing the ventilatory strategy of PE patients is to prevent an increase in RV afterload that can ultimately lead to RV failure.[38] This can be achieved by maintaining a low mean airway pressure, preventing hypoxemia and hypercapnia, and avoiding alveolar de-recruitment and collapse.[39-43] At either low or high lung volumes, there can be a reduction in forward pulmonary blood flow and an increase in RV afterload.[42] Excessive positive end-expiratory pressure (PEEP) also can increase lung volumes and transpulmonary pressures, reduce venous return and RV preload, and increase PVR and RV afterload.[44] For these reasons, a lung-protective ventilatory strategy that limits the plateau pressure with a low PEEP and high F_{IO_2} approach can be used whenever possible. Permissive hypercapnia or high P_{CO_2} may be avoided.

Breathing (B): Administration of supplemental oxygen has been shown to reduce pulmonary artery pressure and PVR and to improve cardiac output in pulmonary arterial hypertension regardless of the inciting cause.[43,45] In acute PE, hypoxic pulmonary vasoconstriction can contribute to an elevated pulmonary artery pressure and an increased RV afterload. A variety of advanced respiratory support techniques, such as HFNC oxygen and bilevel positive pressure airway pressure, can be used to decrease the work of breathing and reduce air hunger if conventional nasal cannula oxygen fails to maintain adequate oxygenation.

Inhaled pulmonary vasodilators, such as nitric oxide or epoprostenol, may counteract the effects of vasoconstrictive cytokines (ie, histamine, serotonin, thromboxane A2) released in acute PE[46] and may improve oxygenation and hemodynamics. However, these agents may not be readily available during transfer, and level 1 evidence is not available supporting their use.

Circulation (C): Optimal hemodynamic resuscitation before the transfer of critically ill patients is of utmost importance.[47] In patients with a high risk of hemodynamic deterioration, central line and arterial line placement can be considered before the transfer. The use

of ultrasound-guided punctures and avoidance of noncompressible sites can reduce bleeding risk, an important caveat because thrombolytics, surgery, or extracorporeal membrane oxygenation may become necessary after the transfer. The inherent value of central and arterial line placement includes the ability to administer vasoactive drugs, real-time hemodynamic monitoring, avoidance of further arterial and venous site punctures after thrombolysis, and conversion into extracorporeal membrane oxygenation lines if needed.

Volume resuscitation is the initial step in treating hypotension in most clinical scenarios.[42,43] The use of small fluid boluses, 250 to 500 mL, may be considered in acutely ill PE patients.[42,43] After volume resuscitation, clinicians may have a low threshold for the use of vasopressors or inotropes to improve contractility and cardiac output in acute RV failure,[48] preserve RV perfusion, and prevent ischemia and right heart collapse.[42] Because inotropic agents carry the risk of increasing systemic hypotension caused by vasodilation, if they are used, then co-administration of vasopressors may be necessary to avoid hypotension.[41-43,45] The characteristics, mechanism of action, and hemodynamic effects of each vasopressor and inotrope on RV overload and PVR are discussed in Table 1. No level 1 evidence is available to clearly guide the clinician regarding a specific vasoactive medication regimen. In terms of PE-specific therapies, therapeutic anticoagulation is the backbone of treating patients with acute PE, regardless of the IHT decision. After risk stratification and identification of the patient as a potential transfer, early assessment of absolute and relative contraindications (Table 2) for thrombolysis can facilitate referring and accepting physicians to make a clear plan for the use of rescue thrombolysis. Several bleeding risk calculators are available, specifically the BACS score[31] or the PE-CH score,[32] to help with this assessment. However, these scores have been shown to have only a modest ability in predicting bleeding risk with thrombolysis. Table 2 lists the absolute and relative contraindications for thrombolytic therapy based on ESC guidelines.

TABLE 2] Absolute and Relative Contraindications for Thrombolytics

Contraindications to Fibrinolysis
Absolute
History of hemorrhagic stroke or stroke of unknown origin
Ischemic stroke in previous 6 months
CNS neoplasm
Major trauma, surgery, or head injury in previous 3 weeks
Bleeding diathesis
Active bleeding
Relative
Transient ischemic attack in previous 6 months
Oral anticoagulation
Pregnancy or first post-partum week
Non-compressible puncture sites
Traumatic resuscitation
Refractory hypertension (systolic BP >180 mmHg)
Advanced liver disease
Infective endocarditis
Active peptic ulcer

If a systemic thrombolytic infusion has been initiated at the transferring facility, extreme caution should be undertaken to avoid injury during transport. Close attention to vital signs and mental status is necessary en route to the accepting hospital. A contingency plan or team pre-brief should be established for any changes in the patient's status (ie, mental status, severe headache, seizure, change in BP, and observed bleeding). The use of ambulance-based thrombolytics is derived mostly from the stroke literature, the PHANTOM-S study from Germany, which demonstrated that its use in acute stroke resulted in decreased time to thrombolysis without any significant increase in adverse events, including intracranial hemorrhage.[49]

Needs for Advanced Management for Transfer

Assessing patients for potential transfer can take into consideration the following factors: the medical necessity, the stability of the patient, and risks and benefits of the transfer. Additionally, transferring physicians can consider the urgency of transfer (immediate vs elective), expected wait time before the transfer, mode of transportation (ground vs air), actual transfer transportation time, and the receiving center's preferences if any.

Published experience with other critical illnesses suggests a benefit in transferring patients to high-volume centers.[17,18,21] However, such data in PE is only recently emerging.[14] The decision to transfer may broadly depend on either PE-specific scenarios (ie, clot-in-transit, high bleeding risk, PE in pregnancy, comorbid medical illnesses) or

TABLE 3] Potential Triggers for IHT[a]

A. Patient-specific factors that may require a higher level of care than can be provided at the transferring facility. This includes but is not limited to:
• Contraindication to anticoagulation or thrombolysis and patient is a candidate for advanced therapies not available at referring center.
• High risk of bleeding (ie, older age, prior stroke, recent major surgeries, acute renal failure)
• PE with severe comorbid medical conditions (ie, end-stage lung or heart disease, peripheral vascular disease, chronic right ventricular dysfunction, pregnancy)
• Syncopal event and fall due to PE.
• Clinical worsening (ie, worsening hypoxemia, tachycardia, need for ICU level of care) or failure to improve on standard anticoagulation.
• Complex PE case scenarios: clot in transit, patent foramen ovale with risk of paradoxical embolism, tumor thromboembolism, illocaval thrombosis.
• Undifferentiated shock with PE in differential.
B. Transfer is part of a regional plan to provide optimal care at a specialized medical facility
C. Patient preference
D. Inability of transferring hospital to have diagnostic (ie, $\dot{V}/\dot{Q}$ scan) and risk stratification (duplex ultrasound, echocardiogram) tools at nights, weekends and holidays.
E. Inability of transferring hospital to deal with potential post-thrombolysis bleeding complications.

IHT = interhospital transfer; PE = pulmonary embolism; $\dot{V}/\dot{Q}$ = ventilation perfusion.

[a]Triggers for transfer may ideally be discussed on individual case-by-case basis.

transferring facility capabilities (ie, ability to offer advanced reperfusion therapies, capability to manage post-thrombolysis bleeding complications, and availability of cardiac surgery and critically trained support staff). Table 3 lists the factors to review when IHT is being considered.

Patient Communication

Informed consent may be obtained from the patient or legal guardian or authorized representative before the initiation of IHT.[50] Furthermore, understanding the patient's goals of care and addressing questions and concerns is essential before initiating the process. However, this may represent a challenge when caring for a life-threatening condition, such as PE. An honest discussion facilitates understanding of the patient's goals and expectations and alignment with the physicians' care plan.[51-54] The physician may want to disclose the urgency of the transfer and the risks and benefits involved. It is helpful to establish realistic expectations about the transfer early in the IHT process. Additionally, it may be important to communicate with the patient that IHT does not mandate advanced PE therapies and that a preliminary plan may change on arrival at the receiving hospital. This decision-making process may be important to document in the patient's chart. If the transferring physician is unable to obtain informed consent, the reason for such inability can be documented as well.[55]

Identifying the Receiving Facility and Transport

Clear communication between referring and receiving centers is of paramount importance to coordinate a safe and expeditious handoff of a critically ill patient.[56,57] Once the decision to transfer the patient is made, the transferring physician identifies the receiving PERT facility that can provide an appropriate higher level of care. Most regional referral centers have a transfer call center that allows direct physician-to-physician consultation, using a single contact number. The receiving facility or transfer call center usually has a pre-identified provider who can triage and discuss the patient's clinical condition with the transferring provider. Depending on protocols, some centers may have a PERT triage physician on call for PE transfers. Similar to the emerging mobile practices for acute ischemic stroke, information technology system may facilitate the remote assessment of acute PE patients. With wireless or cellular-based internet platforms, rapid sharing of pertinent imaging, laboratory values, vital signs, imaging, and medical history can be achieved through various web-based applications or electronic medical records.[58,59] As shown in acute stroke, these information technology system modalities (ie, artificial intelligence software identifying, for example, clot burden and RV function[60,61]) may be able to expedite transfer from remote or rural areas to larger tertiary expert centers.[62]

Alternatively, the accepting physician can involve the PERT, depending on the complexity of the case. If the patient is considered for a specific intervention, it is also appropriate to notify or involve relevant clinical services (ie, cardiothoracic surgeon or interventionalist) before acceptance. This approach allows for proper planning and resource mobilization before the patient's arrival. Table 4 summarizes the key components for IHT.

Deterioration in clinical status (ie, worsening oxygenation, need for noninvasive or invasive ventilation, hypotension, increase in or initiation of new vasopressors, neurological changes) or changes in the patient's goals of care, such as do-not-resuscitate or do-not-intubate, should be communicated, because such development might impact the need for and urgency of transfer.

The referring institution is responsible for preparing a transfer packet that includes progress notes, vital signs, medication administration documentation, and relevant imaging, including discs of CTPA, echocardiography, and venous duplex scans. Delivery of PE imaging studies, that is, CTPA or V/Q̇ scanning, is perhaps the most critical of all because it facilitates advanced care and may prevent unnecessary exposure to additional contrast and delays in care.

TABLE 4] Key Components That Can Be Reviewed During PE IHT

○ Presentation and indication for transfer
○ Relevant medical history (ie, prior bleeding, terminal cancer)
○ Code status
○ Vital signs
○ Available laboratory (troponin, BNP, platelets, aPTT, renal function), and imaging data (chest CTA, ECHO, venous duplex)[a]
○ Bleeding risk assessment, contraindications for thrombolysis
○ Initial therapies: respiratory support (supplemental oxygen, noninvasive ventilation, invasive ventilation), hemodynamic support (intravenous fluids, vasopressors), anticoagulation
○ Stability of the patient for transfer
○ Urgency of transfer, modes of transportation (ground vs air ambulance)
○ Preliminary PE plan, including specific therapies
○ Contingency plan if the patient's condition deteriorates while waiting for transfer

aPTT = activated partial thromboplastin time; BNP = B-type natriuretic peptide; CTA = CT angiogram; ECHO = echocardiogram; PE = pulmonary embolism.
[a]Absence of a specific test result should not delay transfer inquiry in the case of a hemodynamically unstable, critically ill patient in whom PE is suspected or already diagnosed.

Usually, the transfer call center will assist in the management of the logistics of patient transport. The mode of transportation (ground ambulance vs helicopter) will depend on the urgency, availability, and weather travel conditions.[63] Generally, a stable PE patient undergoing IHT can be transferred by an emergency medical service certified in advanced cardiovascular life support. The main benefits of ground ambulance include the ability to operate in most weather conditions and sufficient space in the passenger compartment for critical interventions, and the main benefit of

TABLE 5] Acute PE Patients Monitoring During Transfer

■ Continuous observation of the patient
■ ECG/HR
■ BP (noninvasive or invasive)
■ Oxygen saturation (Sa_{O_2})
■ End-tidal CO_2 ($ETCO_2$) monitoring
■ Signs and symptoms of bleeding: new-onset headache, back pain, blood in the Foley catheters, hematuria, melena
■ Functional 2 large-bore IV line (18 gauge)
Written or electronic record of observations or interventions should be maintained during transport

HR = heart rate

transfer by helicopter is a shorter duration of transport. Depending on the patient's risk for deterioration or current use of hemodynamic or respiratory support, the patient may require an in-transport critical care team.

Unexpected adverse events can happen during the IHT of critically ill patients.[47] Adverse events can be divided into medical and technical incidents during IHT. Cardiovascular events such as hypertension or hypotension, bradyarrhythmia, or tachyarrhythmia (reported incidence, 6%-24%) are the most common adverse events.[47] Other common adverse events include respiratory events such as inadequate ventilation or oxygen desaturation (reported incidence, 0%-15%).[47] During travel, patient movement will need to be minimized. Any changes in posture or movement from one surface to another can have significant hemodynamic, neurological, and respiratory consequences.[64] Therefore, patient transfers (eg, from ambulance to stretcher) must be carefully planned and coordinated between staff. Technical adverse events may include equipment failure (eg, battery/power supply, transport ventilator and monitor function, access to patient elevators, and intubation equipment), and this accounts for 46% of all incidents.[47,65] Known risk factors for developing these adverse events include the use of more than four infusion pumps, sedation before transport, PEEP $>$ 6 cm H_2O, and extended duration of transport.[66] This concern is especially relevant in patients with evolving or deteriorating clinical status, such as worsening respiratory failure or shock in a PE patient. Table 5 provides a list of monitoring parameters for acute PE patients during transfer.

The transfer plan may include predetermined clinical parameters that will trigger diversion to a closer hospital if the patient decompensates during transfer. The transport team is responsible for notifying the accepting team of any changes in the patient's status during transport. Therapies given, adverse events, and procedures undertaken can be clearly documented within the patient's medical chart to ensure that accurate information is obtained by the receiving hospital and compliance with the federal regulations regarding interhospital patient transfers.

Conclusion

The number of advanced therapies for acute PE and critical care delivery in acute PE has evolved over the past decade. Developing IHT protocols are an effective strategy to allocate limited hospital resources and improve the efficiency and safety of transfer systems. Unlike stroke, STEMI, and other acute critical illnesses, the process of interhospital care for acute PE has not received significant focus. This comprehensive document details the current processes involved in IHT of acute, critically ill PE patients. We hope that future research can focus on outcomes of critically ill PE patients undergoing IHT.

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