Published in final edited form as:

Crit Care Med. 2018 March ; 46(3): e229–e234. doi:10.1097/CCM.0000000000002921.

Pediatric In-hospital Cardiac Arrest Secondary to Acute Pulmonary Embolism

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Abstract

Objective—Pulmonary embolism (PE) is a rarely reported and potentially treatable cause of cardiac arrest in children and adolescents. The objective of this case series is to describe the course of five adolescent patients with in-hospital cardiac arrest secondary to PE.

Design—Case series.

Setting—Single, large academic children's hospital.

Patients—All patients under the age of 18 years (n = 5) who experienced an in-hospital cardiac arrest due to apparent PE from August 1, 2013 through July 31, 2017.

Interventions—All five patients received systemic thrombolytic therapy (intravenous tissue plasminogen activator; tPA) during cardiac arrest or peri-arrest during ongoing resuscitation efforts.

Measurements and Main Results—Five adolescent patients, aged 15 to 17 years, were treated for PE-related cardiac arrests during the study period. These accounted for 6.3% of all children and 25% of adolescents (12–17 years of age) receiving at least five minutes of in-hospital cardiopulmonary resuscitation during the study period. All five had venous thromboembolism risk factors. Two patients had known, extensive venous thrombi at the time of cardiac arrest and one was undergoing angiography at the time of arrest. The diagnoses of PE were based on clinical suspicion, bedside echocardiography (n = 4), and low end-tidal carbon dioxide levels relative to arterial carbon dioxide values (n = 5). Intravenous tPA was administered during cardiopulmonary resuscitation in three patients and after the return of spontaneous circulation, in the setting of

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Reprints ordered: No

Financial support for this study: None

Copyright form disclosure: Dr. Topjian received support for article research from the National Institutes of Health. Dr. Sutton's institution received funding from the National Heart, Lung, and Blood Institute; he received funding from Zoll Medical (speaking honoraria); and he disclosed that he is a member of the American Heart Association's Get with the Guidelines Pediatric Research Task Force. The remaining authors have disclosed that they do not have any potential conflicts of interest.

severe hemodynamic instability, in the other two patients. Four of five patients were successfully resuscitated and survived to hospital discharge.

Conclusions—PE was recognized as the etiology of multiple adolescent cardiac arrests in this single center series and may be more common than previously reported. Recognition, high-quality cardiopulmonary resuscitation, and treatment with thrombolytic therapy resulted in survival in four of five patients.

Key words for indexing

Cardiac arrest; Cardiopulmonary resuscitation; Pulmonary embolism; Thrombolytic therapy; Venous thromboembolism; Adolescent

Introduction

Pediatric in-hospital cardiac arrest (IHCA) occurs between 5,000 and 10,000 times annually in the United States alone (1). While outcomes have improved substantially in recent years (2), more than half of children with IHCA do not survive to hospital discharge (3). In addition to the delivery of high-quality cardiopulmonary resuscitation (CPR), recognition and prompt treatment of the underlying etiology of IHCA is imperative to optimize resuscitation success (4).

In adults, massive pulmonary embolism (PE) causes between 5% and 5.7% of IHCAs (5, 6) and between 2.8% and 13.3% of out-of-hospital cardiac arrests (OHCAs) (7–9). When PE is recognized as the etiology of adult cardiac arrest, systemic thrombolytic therapy can be an effective treatment (10–12). Pediatric PE-associated cardiac arrest is seldom reported, but single-institution studies have reported it in multiple patients (13, 14) and autopsy studies have demonstrated high numbers of PE among children with unknown causes of death (15). This suggests that PE may be under-recognized and under-reported as a pediatric cardiac arrest etiology. Notably, there are no published reports of survival with thrombolysis following cardiac arrest due to PE in children or adolescents. In this case series, we describe the clinical courses of five adolescent patients from a single institution who had PE-associated IHCA and received systemic thrombolytic therapy, four of whom survived to hospital discharge.

Methods

This retrospective observational case series was conducted after the Institutional Review Board of the Children's Hospital of Philadelphia waived the need for informed consent. An institutional cardiac arrest research database was queried to identify all children < 18 years of age who received at least five minutes of CPR for an IHCA from August 1, 2013 through July 31, 2017. A minimum CPR duration of five minutes was selected to include a population with sufficient duration of resuscitation efforts to allow for an evaluation for reversible causes, including PE (16, 17). Patients who had CPR initiated in the emergency department, pediatric intensive care unit (PICU), general inpatient care areas, operating room, and radiology were included. Patients with OHCAs and IHCAs occurring in ambulatory settings, neonatal intensive care unit, and cardiac intensive care unit were

excluded. The resuscitation records of eligible patients were reviewed to identify cases in which PE was identified as the possible etiology of IHCA by the resuscitation team. The pertinent aspects of those cases were collected and described. These included the details of the pre-, intra-, and post-IHCA course and the presence of venous thromboembolism (VTE) risk factors, including known history of VTE, obesity, malignancy, prolonged bed rest, recent major surgery or trauma, lower extremity fracture, or other minor risk factors (18, 19). Pediatric Cerebral Performance Category (PCPC) scores, which range from 1 (“normal”) to 6 (“brain death”) (20), were calculated and reported at pre-arrest baseline and at hospital discharge.

In addition to the case descriptions, additional outcomes included the proportion of patients with IHCA in whom PE was recognized as the etiology, the proportion of adolescent IHCA patients in whom PE was recognized as the etiology, and rates of survival to hospital discharge in PE-associated IHCA and in other IHCAs.

Results

Incidence/case identification

Pulmonary embolism was identified and documented as the cause of IHCA in 6.3% (5/79) of eligible patients. All five patients with PE were adolescents, accounting for 25% (5/20) of adolescent patients (12–17 years of age) requiring CPR for at least five minutes.

Case Descriptions (Table 1)

Patient 1—A 16-year-old obese male with a history of a fibular fracture 12 days earlier presented to the Emergency Department with dyspnea and hypoxemia. His symptoms progressed to pulseless electrical activity (PEA) cardiac arrest. CPR was performed for two minutes with a brief return of spontaneous circulation (ROSC). Recurrence of PEA cardiac arrest prompted 12 additional minutes of CPR before obtaining sustained ROSC. One hour after ROSC, computed tomography angiography (CTA) revealed extensive bilateral pulmonary artery (PA) emboli, dilation of the main PA, and enlarged right heart structures with a flattened interventricular septum (Figure 1). Heparin therapy was initiated. Four hours post-ROSC, the patient exhibited profound hemodynamic instability necessitating high-dose inotrope and vasopressor support. A large gradient between arterial carbon dioxide (60–70 mmHg) and end-tidal carbon dioxide (ETCO₂; 20–30 mmHg) was suggestive of compromised pulmonary blood flow. Bedside echocardiography demonstrated right ventricular (RV) dilation and dysfunction. Intravenous tPA (10 mg bolus followed by 90 mg over two hours) was administered with prompt hemodynamic improvement. Angiography, performed two hours after the bolus dose of tPA, demonstrated PE resolution. The patient was transferred out of the PICU on hospital day four and was later discharged home without impairment. He now attends college full-time.

Patient 2—A 16-year-old female with newly diagnosed diffuse large B-cell lymphoma developed altered mental status and hypoxemia while an inpatient on the Oncology ward. She was not receiving pharmacologic or mechanical VTE prophylaxis at the time. She underwent emergent endotracheal intubation, developed bradycardia with poor perfusion,

and CPR was initiated. After eight minutes of CPR, ROSC was briefly attained. During that time, bedside echocardiography demonstrated moderate RV dilation and severe RV systolic dysfunction. PEA recurred and CPR continued for 20 additional minutes. Despite profound arterial hypercarbia and objective evidence of high-quality CPR, including invasively measured diastolic blood pressures greater than 25 mmHg, ETCO₂ values remained between 8 and 12 mmHg. In the setting of underlying VTE risk factors, ongoing PEA, low ETCO₂ values, and evidence of RV dilation, massive PE was considered as the primary etiology of cardiac arrest.

At that time, tPA was administered intravenously (10 mg bolus followed by 90 mg over two hours). During the following two minutes of ongoing CPR, ETCO₂ values rose to the mid-thirties. Sustained ROSC was achieved 40 minutes after the initial start of CPR. Thirty minutes post-ROSC, CTA demonstrated an enlarged main PA and multiple filling defects in the segmental and subsegmental arteries of both lungs with an embolus in a proximal branch of the left PA. The patient required two days of mechanical ventilation and three days of inotropic support. The patient was transferred from the PICU four days post-arrest and was later discharged home without impairment. She now attends college full-time.

Patient 3—A 17-year-old female with 22q13 deletion, developmental delay, and autism was admitted to the PICU with septic shock and acute respiratory failure. Sequential compression devices were used for mechanical VTE prophylaxis while she was bedbound. On hospital day seven, after she had been extubated and weaned from vasopressor support, she developed altered mental status, hypoxemia, and bradycardia. Shortly after endotracheal intubation, she had bradycardia that progressed to PEA and CPR was started. CPR continued for 29 minutes, with intermittent periods of a perfusing rhythm that each lasted for less than one minute, followed by bradycardia, declining ETCO₂ to < 10 mmHg and loss of central pulses, prompting resumption of CPR. During one such period, bedside echocardiography performed by the critical care team demonstrated a moderately to severely dilated RV, interventricular septal flattening with compression of the left ventricle, and moderately diminished RV systolic function. A 7.5 mg bolus of tPA was administered intravenously with improvement in hemodynamics and sustained ROSC. Ninety minutes post-ROSC, CTA did not reveal persistence of PE, though Doppler ultrasonography demonstrated an occlusive upper extremity deep venous thrombosis. The patient developed hypoxic-ischemic brain injury as a result of her cardiac arrest and was discharged home with a PCPC score of 5, worsened from her baseline of 4.

Patient 4—A 17-year-old female with new-onset diabetic ketoacidosis developed an occlusive thrombus of her right common femoral vein associated with a central venous catheter that had been placed at a referring hospital. Despite anticoagulation, the thrombus propagated from the popliteal vein to the distal inferior vena cava (IVC). During mechanical thrombectomy with site-directed tPA in Interventional Radiology, she had a sudden decrease in ETCO₂ to 15 mmHg, which was associated with hypotension and hypoxemia that rapidly progressed to PEA cardiac arrest. After five minutes of CPR and two doses of epinephrine, ROSC was obtained. A large gradient between arterial carbon dioxide (80 mmHg) and ETCO₂ (15–20 mmHg), along with her underlying diagnosis and profound hypotension was

suggestive of persistent massive PE, and intravenous tPA was administered (10 mg bolus followed by 90 mg over two hours) with subsequent improvement in hemodynamics. Pulmonary angiography revealed complete occlusion of the right PA (Figure 2). Serial pulmonary angiograms showed gradual improvement in right lung perfusion, and complete lung perfusion was restored with catheter-directed tPA to the right PA. An echocardiogram showed RV strain, which improved within 24 hours of ROSC. She was extubated two days later, transferred to the Endocrinology service for further management of her diabetes, and was later discharged home without neurologic deficits.

Patient 5—A 15-year-old female was diagnosed with pelvic osteosarcoma with spinal and vascular invasion, complicated by neurogenic bowel with severe constipation and extensive tumor thrombus within the infrarenal IVC. She had a long-acting subcutaneous birth control implant in situ. Three days following chemotherapy initiation and after failing aggressive medical therapy for constipation, she underwent a fecal disimpaction under general anesthesia. Immediately after the procedure, she developed acute hypoxemia and tachycardia in the Operating Room. Her ETCO₂ dropped to <10 mmHg during bag-mask ventilation and she was hypotensive with rapid progression to PEA cardiac arrest. During CPR, she had multiple periods of non-sustained ROSC – during one such period an echocardiogram demonstrated a severely dilated RV and minimal RV contractility. Intravenous tPA (10 mg bolus dose) was administered approximately 30 minutes after the initiation of CPR without discernible effect. Despite institution of venoarterial extracorporeal membrane oxygenation during CPR, she had persistent shock with multisystem organ dysfunction and no evidence of brain function. Life-sustaining technologies were withdrawn and the patient died. Autopsy revealed tumor thrombus in the IVC and pulmonary arteries.

Discussion

This case series describes five adolescent patients with IHCA due to PE. These patients represent 6.3% of all children and 25% of adolescents admitted to our PICU who received at least five minutes of in-hospital CPR. All five of the patients in whom PE was strongly suspected were treated with thrombolytic therapy (tPA), three during CPR and two soon after ROSC in the setting of severe post-arrest hypotension. Four of five patients survived to hospital discharge and three had favorable neurologic outcomes. The frequency of PE-associated cardiac arrests over a four-year period may be related to increased awareness in our PICU of a previously under-recognized problem. Notably, PE may have been unrecognized as the etiology of IHCA in other patients without focused diagnostic testing or post-mortem examinations; it's true prevalence may actually be higher than that reported here. Importantly, these cases establish that prompt recognition and systemic thrombolysis for pediatric patients with PE-associated cardiac arrests can be life-saving.

All of these patients with PE-associated cardiac arrests were adolescents with VTE risk factors, which raised the level of clinical suspicion for PE among the resuscitation teams. Two patients were specifically known to have extensive venous thrombi in the IVC. Cardiac arrest characteristics that further suggested diagnosis of PE included the arrest rhythm, findings of intermittent ROSC, ETCO₂ values, and echocardiography. While none of these

cardiac arrest characteristics independently confirm a diagnosis of PE-associated IHCA, the constellation of findings should raise concern and prompt consideration for targeted thrombolytic therapy. PEA is the initial rhythm in the majority of adult PE-associated cardiac arrests (21) and was present in all five of the patients described here. Although it can be difficult to differentiate PEA from severe hypotension (22), all of these patients were non-responsive without palpable pulses and thus met clinical criteria for providing CPR (23). During CPR, four patients had multiple non-sustained periods of ROSC, followed by recurrence of PEA. We speculate that ROSC may have been related to mechanical thrombus disruption via chest compressions with recurrence of PEA due to recurrent PA obstruction. During CPR and periods of spontaneous circulation, wide gradients were noted between arterial carbon dioxide concentrations and ETCO_2 , suggesting either poor cardiac output or compromised pulmonary blood flow and severe ventilation-perfusion mismatch (24). Lastly, four of these five patients had real-time bedside cardiac ultrasound that demonstrated evidence of right ventricular dilation. Severe right ventricular dilation provides support for the diagnosis of PE (25–28), though it is not necessarily due to PE in the setting of cardiac arrest (29). Though intra-arrest cardiac ultrasound is feasible in children (30), caution should be applied to not compromise CPR quality by excessive interruptions in chest compressions.

All five patients in this study with PE-related IHCA received systemically administered tPA and four patients survived to hospital discharge. The non-surviving patient had actual tumor thrombus on imaging and autopsy, which is likely less amenable to thrombolytic therapy. To the authors' knowledge, these are the first reports of successful resuscitation from PE-associated cardiac arrest using systemic thrombolysis in pediatric or adolescent patients. In adults with cardiac arrest due to confirmed PE, systemic thrombolysis has generally been associated with improved rates of ROSC and short-term survival (10–12, 31–35), though at least one retrospective study has failed to identify a survival benefit (36). According to updated 2015 American Heart Association (AHA) guidelines, “in patients with confirmed PE as the precipitant of cardiac arrest, thrombolysis, surgical embolectomy, and mechanical embolectomy are reasonable emergency treatment options (Class IIa, LOE C-LD) (16).” In children, systemic thrombolysis during PE-related cardiac arrest has been reported, though previously without survival to hospital discharge (13, 37). Pediatric dosing recommendations for thrombolysis during cardiac arrest do not exist. In four of the adolescent patients described here, adult doses of tPA were administered, and the other patient received a weight-adjusted bolus dose extrapolated from adult dosing recommendations. Due to the experience with these patients, our institution has developed a specific order set in the electronic medical record for “tPA for cardiac arrest from suspected massive PE.” This provides tPA dosing guidelines of a 10 mg bolus dose followed by 90 mg over two hours for patients ≥ 60 kg, and a 0.1 mg/kg bolus dose followed by 0.9 mg/kg over two hours for patients < 60 kg. There were no apparent tPA-related complications in the patients reported here, though previous publications suggest that the bleeding risks associated with tPA are potentially increased in cardiac arrest patients compared to other patients treated with thrombolytic therapy (33, 38). Nevertheless, we believe that the potential benefit of tPA outweighs this risk when PE is confirmed or highly suspected as an etiology of cardiac arrest.

Pediatric advanced life support guidelines do not specifically address PE as an etiology for cardiac arrest or discuss the approach to diagnosis or treatment (4). This is not surprising, given the paucity of pediatric literature on the topic and the low incidence reported to date (39). Despite the observational nature of the current study, it suggests that clinicians should be suspicious of PE as an etiology of pediatric in-hospital cardiac arrest, and that systemic thrombolytic therapy can be effective.

Conclusions

PE-associated IHCA was diagnosed and treated in five adolescent patients over a four-year period in our institution. These data suggest that PE may be a more common etiology of pediatric IHCA than previously reported, especially among adolescents with known VTE risk factors. The cardiac arrest rhythm, ETCO₂ monitoring during CPR, and echocardiography findings can aid in the diagnosis of PE. Most importantly, the use of systemic thrombolytic therapy can be a life-saving intervention.

Acknowledgments

We wish to thank our colleagues from Hematology, Oncology, Emergency Medicine, Pharmacy, General Surgery, Radiology, Cardiology, Neurology, Endocrinology, Critical Care Medicine, and the Pharmacy at the Children's Hospital of Philadelphia for their care of these patients, as well as Sitara Kumar and Natalie Atkin for their assistance with data compilation.

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Figure 1. CT angiogram, Patient 1

Computed tomography angiogram of Patient 1, acquired one hour after return of spontaneous circulation and prior to further hemodynamic decompensation and administration of tissue plasminogen activator, demonstrating extensive bilateral pulmonary emboli (thick arrows) and dilated main pulmonary artery (thin arrow).

Figure 2. Pulmonary angiogram, Patient 4

Pulmonary angiogram of Patient 4, acquired after return of spontaneous circulation and approximately ten minutes after administration of tissue plasminogen activator, demonstrating extensive filling defect in the right pulmonary artery (arrow).

Table 1

Case Summaries

Sex, Age (years)	Arrest Location	Underlying Diagnoses	Venous thromboembolism risk factors	Initial Rhythm; Subsequent Rhythm	Cardiac arrest features consistent with PE	Timing of thrombolysis	Outcome; Admission vs. Discharge PCPC score
Male, 16	Emergency Department	Fibular fracture 12 days prior to presentation	Long bone fracture, immobilization, obesity	PEA	Intermittent, non-sustained ROSC; low ETCO ₂ ; RV dilation/dysfunction on echocardiography	Post-arrest (shock with RV dilation/dysfunction, low ETCO ₂)	Discharged home PCPC: 1 → 1
Female, 16	Oncology Ward	Diffuse large B-cell lymphoma	Malignancy, immobilization, no VTE prophylaxis	Bradycardia with poor perfusion; PEA	Intermittent, non-sustained ROSC; low ETCO ₂ ; RV dilation/dysfunction on echocardiography	Intra-arrest (after 38 minutes of CPR)	Discharged home PCPC: 1 → 2
Female, 17	Pediatric Intensive Care Unit	22q13 deletion, autism, septic shock, acute hypoxemic respiratory failure	Immobilization, central venous catheter-associated thrombus (diagnosed post-ROSC)	PEA	Intermittent, non-sustained ROSC; low ETCO ₂ ; RV dilation/dysfunction on echocardiography	Intra-arrest (after 29 minutes of CPR)	Discharged home PCPC: 4 → 5
Female, 17	Interventional Radiology	Diabetic ketoacidosis, DVT extending from popliteal vein to IVC	Diabetic ketoacidosis, known DVT (central venous catheter-associated)	PEA	Low ETCO ₂ ; angiography with main pulmonary artery thrombus	Post-arrest (shock with right pulmonary artery thrombus)	Discharged home PCPC: 2 → 2
Female, 15	Operating Room	Pelvic osteosarcoma with spinal and vascular invasion	Malignancy, immobilization, hormonal contraceptive, extensive IVC tumor thrombus	PEA	Intermittent, non-sustained ROSC; low ETCO ₂ ; RV dilation/dysfunction on echocardiography	Intra-arrest (after 30 minutes of CPR)	Death PCPC: 2 → n/a

Patient and cardiac arrest characteristics of five patients with PE-associated cardiac arrest. *Definition of abbreviations:* PCPC, pediatric cerebral performance category; PEA, pulseless electrical activity; ROSC, return of spontaneous circulation; ETCO₂, end-tidal carbon dioxide; RV, right ventricle; VTE, venous thromboembolism; CPR, cardiopulmonary resuscitation; DVT, deep vein thrombosis; IVC, inferior vena cava.