

WebM&M

Morbidity and Mortality Rounds on the Web

Spotlight

Delay in Appropriate Diagnosis and Treatment Leading to Death from Pulmonary Embolism



Agency for Healthcare Research and Quality
Advancing Excellence in Health Care



Source and Credits

- This presentation is based on the February 2021 AHRQ WebM&M Spotlight Case
 - See the full article at <https://psnet.ahrq.gov/webmm>
 - CME credit is available
- Commentary by: David Barnes, MD and William Ken McCallum, MD
- AHRQ WebM&M Editors in Chief: Patrick Romano, MD, MPH and Debra Bakerjian, PhD, APRN, RN
 - Spotlight Editors: Jacqueline Stocking, PhD, MSN, and Patricia Poole, PharmD
 - Managing Editor: Meghan Weyrich, MPH

Objectives

At the conclusion of this educational activity, participants should be able to:

- Recognize and interpret historical elements, laboratory and imaging studies, and ECG findings to diagnose pulmonary embolism
- Appreciate that asthma and orthopedic surgery are both associated with increased risk of pulmonary embolism
- Compare and contrast different diagnostic approaches to patients determined to be at low, intermediate, or high risk for pulmonary embolism
- Use and prioritize effective, timely communication and verification of information to maximize patient safety
- Develop an approach to managing patients diagnosed with pulmonary embolism including anticoagulation, use of targeted response teams, and consultation with surgical and interventional specialists

DELAY IN APPROPRIATE DIAGNOSIS AND TREATMENT LEADING TO DEATH FROM PULMONARY EMBOLISM

A case describing how lack of timely communication and verification of information led to missed diagnosis of submassive pulmonary embolism resulting in fatality

Case Details (1)

- 56-year-old woman presented to the ED for shortness of breath
 - Past medical history of mild, persistent asthma and recent Achilles tendon repair
- In the ED, chest tightness was relieved with Albuterol
- Patient had diffuse wheezing on physical examination and mild pulmonary edema (but no focal abnormalities) on chest radiograph
- Patient was admitted to the hospital for acute asthma exacerbation

Case Details (2)

- Patient initially responded to treatment with aggressive pulmonary toilet and bronchodilator therapy
- Eight hours after admission, her serum lactate rose to 4.7 mmol/L (normal <2.0 mmol/L) and her oxygen requirements increased to 3-4 liters/minute
- Based on her limited mobility secondary to recent orthopedic surgery, a computed tomography (CT) angiogram of the chest was obtained to rule out a pulmonary embolism (PE)

Case Details (3)

- The radiologist's summarized impression – communicated by telephone to the care team 12 hours after admission – noted bilateral filling defects to the subsegmental level in all five lung lobes without right heart strain or saddle embolus
 - However, the written radiology impression was not reviewed, nor did the care team independently review the CT images
 - The radiologist's full note mentioned “profound evidence of right heart strain” but this critical finding was not conveyed to the primary team
- The patient was started on a direct oral anticoagulant (DOAC)

Case Details (4)

- On hospital day 2, her serum lactate continued to rise and the rapid response team (RRT) responded for increasing tachycardia
 - She remained hemodynamically stable and not in acute distress, so neither the RRT nor the primary team made changes to the care plan
- That evening, she was transferred to the ICU after becoming hemodynamically unstable with hypotension and increasing tachycardia
- Bedside point-of-care ultrasound demonstrated marked right heart strain

Case Details (5)

- Arterial and central venous catheters were placed and she was given tissue plasminogen activator (tPA)
 - Approximately five minutes later, she developed acute signs of stroke, seizure-like movements, and required closed chest compressions and cardiopulmonary resuscitation
 - After return of spontaneous circulation, the patient was cannulated for extracorporeal membrane oxygenation (ECMO)
 - Ongoing resuscitation, including administration of vasopressor therapy, continued overnight
- She ultimately transitioned to comfort care and died on hospital day three

DELAY IN APPROPRIATE DIAGNOSIS AND TREATMENT LEADING TO DEATH FROM PULMONARY EMBOLISM

THE COMMENTARY

By David Barnes, MD and William Ken McCallum, MD

Background (1)

- Studies have found that discriminating between uncomplicated asthma exacerbation and other causes of dyspnea, including PE, is challenging.
- In this case, the emergency physician may have prematurely closed on the diagnosis of asthma exacerbation and failed to explore alternative diagnoses, particularly in the setting of recent Achilles tendon surgery
 - Achilles tendon rupture has been associated with a higher risk for venous thromboembolism (VTE), including deep vein thrombosis (DVT) and PE.
 - Anchoring on the diagnosis of asthma led to a delay in the diagnosis of PE and alternative diagnoses were considered only after clinical decompensation became obvious.

Background (2)

- The radiologist did not convey critical findings from the chest CT angiogram to the inpatient team, which was only made aware of the incomplete initial impression
 - Without full appreciation of disease severity, they considered her to be low-risk and initiated therapy with a direct oral anticoagulant (DOAC)
 - The presence of right heart strain satisfied criteria for submassive PE and activation of the pulmonary embolism response team (PERT), which includes a member of pulmonary/critical care, should have followed

Background (3)

- This “perfect storm” of events--an error pattern commonly referred to as the “Swiss cheese model”--eventually led to the correct diagnosis of PE but only after significant delay.
 - Clinical decompensation (rising serum lactate and increased supplemental oxygen needs) was the primary mechanism of error recognition.
 - The communication lapse between radiology and the primary team could have been avoided had the primary team independently reviewed the CT images
 - With an incomplete appreciation of severity, the PERT was not activated. Only after the patient developed hemodynamic instability, and right heart strain was identified on bedside ultrasound, was the severity of the patient’s condition truly appreciated.

Emergency Department Diagnosis of Pulmonary Embolism

ED Diagnosis of Pulmonary Embolism (1)

- PE evaluation combines clinical gestalt, clinical decision rules, D-Dimer measurement, and/or diagnostic imaging such as ultrasound (US), CT angiography, or ventilation-perfusion (VQ) scanning.

ED Diagnosis of Pulmonary Embolism (2)

- Clinical decision aids assist clinicians in determining the likelihood of PE and inform whether or not further testing for PE is indicated
 - The PE Rule-out Criteria (PERC), developed to decrease unnecessary testing for PE, includes abnormal vital signs, physical exam findings concerning for DVT and historical risk factors such as age and exogenous estrogen use
 - Unfortunately, PERC could not rule out PE in this case because of the patient's age and recent surgery

ED Diagnosis of Pulmonary Embolism (3)

- Patients are generally evaluated further using a D-Dimer assay
 - D-dimer, derived from the breakdown of thrombus, is elevated in patients with VTE and should be normal in the absence of a thrombotic process (i.e., high sensitivity).
 - Unfortunately, D-dimer may be elevated in non-thrombotic conditions including pregnancy, malignancy, hemodialysis, and inflammatory diseases (i.e., low specificity).
 - Because D-dimer levels normally rise with advancing age, even in the absence of disease, age-adjusted D-dimer should be used when pretest probability is considered low or intermediate

ED Diagnosis of Pulmonary Embolism (4)

- D-dimer is not a diagnostic study – further diagnostic testing is indicated if D-dimer is elevated, which may lead to CT overutilization if applied haphazardly
 - Physicians must consider the potential harmful effects of CT overutilization, including contrast hypersensitivity reactions and nephropathy, and risk of malignancy secondary to ionizing radiation
- Because D-dimer is highly sensitive but not specific, a value in the normal range essentially rules out the disease.
 - Conversely, any elevation in D-dimer should be followed by an imaging study.
 - Based upon the data available at her presentation, the patient in this case had a low pretest probability for PE and D-dimer measurement may have been an appropriate next step.

ED Diagnosis of Pulmonary Embolism (5)

- Surgery and immobilization are known risk factors for VTE
 - Orthopedic procedures are associated with significant risk of VTE, especially in the first seven days after surgery
 - In an observational study of nearly 46,000 consecutive orthopedic surgeries, repair of Achilles tendon rupture was associated with the highest incidence of VTE, although there were no cases of PE
 - Data specific to Achilles tendon repair suggest symptomatic DVT occurs in up to 7% of patients, with 1.3% developing PE
 - Despite this increased risk, routine VTE prophylaxis after Achilles tendon repair is not currently recommended.
- Although it is not clear if the patient in this case was anticoagulated, the nature of her surgery clearly elevated her risk for developing VTE.

ED Diagnosis of Pulmonary Embolism (6)

- For hemodynamically unstable patients, a combination of clinical features, ultrasonography, and ECG may suggest PE even in the absence of diagnostic imaging.
- Patients with high pretest probability should be evaluated with CT angiography or VQ scanning if hemodynamically stable and able to safely undergo a diagnostic radiological study. Otherwise, empiric treatment is strongly recommended.

To Test or Not to Test... That is the Question

To Test or Not to Test (1)

- Anchoring, a type of cognitive bias, occurs when a clinician fixates on features of a specific diagnosis but fails to adjust their thinking when new information becomes available.
 - Anchoring leads to premature closure on diagnoses that may seem obvious or most likely at first but are ultimately determined to be inaccurate.
- In this case, the reasonable diagnosis of asthma exacerbation was initially pursued based on symptomatic improvement with typical interventions.
 - This approach isn't necessarily wrong; clinicians must balance missing a diagnosis like PE against the likelihood of the favored diagnosis and risks associated with diagnostic imaging and medical waste.

To Test or Not to Test (2)

- While not necessarily common, PE can mimic bronchial asthma
- The risk of PE with asthma is also related to severity of disease and corticosteroid use, possibly due to chronic inflammation or physiologic changes caused by glucocorticoids
 - The patient in this case had a history of asthma and improved with standard treatment
- Testing every asthmatic for PE is unreasonable. Instead, clinicians should carefully consider the risk for PE in every asthmatic who presents with acute dyspnea and determine the need for further testing
- In the current case, additional testing was most likely indicated given the combination of asthma and recent orthopedic surgery

To Test or Not to Test (3)

- Studies traditionally ordered during the workup of dyspnea and chest pain, though lacking specificity, may identify right heart strain and suggest the diagnosis of PE.
 - Assays for brain-type natriuretic peptide (BNP) and Troponin-T are sensitive but not specific for PE.
 - ECG findings associated with right heart strain are associated with increased risk of circulatory shock and death from PE
 - Chest radiography may demonstrate other causes of chest pain and dyspnea
 - POCUS or formal transthoracic echocardiogram are rapid and can be performed at bedside, which is useful if the patient is unstable.
- While isolated abnormalities may not be helpful, a constellation of abnormalities might be

Management of Suspected or Confirmed Pulmonary Embolism

Management of Suspected or Confirmed PE (1)

- PE is classified into one of three risk severity categories: low, intermediate (i.e., submassive), and high (i.e., massive)
- Massive PE causes hemodynamic instability and requires aggressive interventions such as systemic thrombolysis, vasopressor therapy, or cardiopulmonary resuscitation
- Submassive PE does not cause hemodynamic instability but does produce right heart strain
 - Anticoagulation is the mainstay of treatment after diagnosis of PE, but hemodynamically stable patients with high pretest probability for PE should be empirically anticoagulated pending diagnostic studies because mortality increases with delays in anticoagulation administration
 - Patients anticoagulated in the ED have lower mortality and reduced hospital and intensive care unit lengths-of-stay

Management of Suspected or Confirmed PE (2)

- First-line treatment for massive PE is systemic thrombolysis (e.g., tissue plasminogen activator, tPA), though a multidisciplinary approach with advanced interventions (e.g., thrombectomy) may be considered
- Submassive PE is treated with anticoagulation, either a DOAC, low molecular weight heparin (LMWH), or unfractionated heparin (UFH).
 - DOAC therapy is associated with lower all-cause mortality and is recommended unless contraindicated, while patients requiring systemic thrombolysis or procedural intervention should be treated with UFH
 - The patient in this case was appropriately started on a DOAC for submassive PE. After hemodynamic decompensation, low-dose tPA was appropriate given active DOAC treatment, although alternative interventions may have also been considered

Management of Suspected or Confirmed PE (3)

- Several tools exist to predict the severity and prognosis of PE, but none is precise.
 - Clot burden and location of PE on CT do not help with risk stratification.
 - Lactic acid greater than 2 mmol/L in PE is associated with higher all-cause mortality, likely indicating impaired cardiac output and microvascular perfusion.
 - In the current case, the patient had an elevated lactic acid along with other signs of cardiogenic shock, which should have alerted the inpatient team that her disease was more severe than first suspected.

Radiology and Communication

Radiology and Communication (1)

- The American College of Radiology (ACR) defines three time frame categories for actionable findings that require communication: within minutes (category 1), within hours (category 2), or with days (category 3)
 - Category 1 findings include those that require immediate action and direct communication with providers to avoid death or significant morbidity.
 - ACR recommends inclusion of PE as a Category 1 diagnosis with expedited communication of diagnostic reporting in emergent or non-routine clinical situations.

Radiology and Communication (2)

- Treatment delays can be caused by miscommunication of findings, failure of timely reporting, as well as failure of clinicians to read radiology reports.
 - Miscommunication may occur verbally, in written reports, or both.
 - While inpatient physicians are not radiologists, many abnormal radiology findings are apparent even to non-radiologists. However, in a survey of providers who ordered imaging studies, only 38% read the entire radiology report and 18% read it only if the conclusion was unclear, suggesting that clear report conclusions are key to effective communication.

Radiology and Communication (3)

- To improve patient safety, ACR places shared responsibility on ordering physicians for timely follow-up of diagnostic studies.
- In this case, the diagnosis of submassive PE was listed in the complete report. Yet, the final impression did not accurately relay critical information found in the detailed interpretation. The failure of the radiologist to communicate these findings to the treatment team in a timely manner contributed to patient harm.

Pulmonary Embolism Response Team (PERT)

Pulmonary Embolism Response Team (1)

- Purpose is to treat a heterogeneous patient population with massive or submassive PE, streamline patient assessment, and facilitate and expedite treatment options.
 - Studies evaluating mortality and other benefits of PERT are mixed. Advantages include shortened ICU and overall hospital length-of-stay as well as improved median time from diagnosis to anticoagulation.

Pulmonary Embolism Response Team (2)

- PERT was not activated in this case, and escalation of care was delayed despite obvious clinical deterioration.
- Earlier identification of submassive PE and PERT consultation may have changed the patient's course with timely and appropriate anticoagulation, earlier escalation of care, and consideration of alternate treatment options.

Approach to Improving Safety

Approach to Improving Safety (1)

- This case represents a classic Swiss cheese model in which several, small, independent variables align to cause patient harm.
 - The Swiss cheese model is a conceptual framework used to explain systems errors that lead to poor outcomes. Each layer of cheese represents an independent layer of defense against error as long as the holes do not align.
 - In complex systems, holes are constantly opening, closing, and shifting; holes arise due to active failures by actors and latent conditions within the system that were not previously identified and mitigated.

Approach to Improving Safety (2)

- In this case, multiple errors occurred across the spectrum of providers and were exacerbated by latent systems issues, including:
 - Anchoring bias with premature closure on an asthma diagnosis
 - Failure to recognize significant risk factors for PE (recent Achilles tendon surgery)
 - Not providing empiric anticoagulation once PE was suspected
 - Delayed notification of critical radiologic findings
 - Failure of the primary team to review imaging studies
 - Failure to recognize multiple indicators of right heart strain
 - Delay in escalation of care after signs of decompensation
 - Failure to consult PERT and mobilize additional resources

Approach to Improving Safety (3)

- Clinicians should be alert to anchoring bias when making initial admission diagnoses.
- On an individual level, physicians should be aware of, and take action to address, their cognitive biases such as considering alternative diagnoses, developing reflective approaches to problem-solving, and relying less on memory.
- Hospitals should develop physician training programs, formalize accountability and feedback processes, and incorporate system-level changes to streamline processes and information flow to reduce diagnostic error.
- Clinicians should independently verify information provided by consultants to ensure that all available clinical data are considered.

Approach to Improving Safety (4)

- Communication gaps and miscommunication in documentation can lead to diagnostic delays and errors.
- Healthcare systems should continually evaluate for and address communication barriers that lead to these latent errors.
- Information technology may improve communication by identifying and locating ordering providers as well as prompting radiologists to follow recommended procedures.

TAKE HOME POINTS

Take-Home Points (1)

- Asthma exacerbations and PE may present similarly. Patients with asthma and those with recent orthopedic procedures have increased risk of PE. It is important to maintain a broad differential diagnosis in order to avoid anchoring on other diagnoses.
- Laboratory, ECG, and imaging findings suggestive of PE are nonspecific, but a constellation of abnormal findings should raise suspicion in patients at risk for PE.

Take-Home Points (2)

- In the absence of contraindications, empiric anticoagulation should be administered while awaiting confirmatory testing when PE is likely (i.e, high risk patients).
- Clinicians are responsible for appropriate communication and review of all available data to maximize patient safety and minimize diagnostic error.
- PERT activation broadens treatment options for PE, especially those with massive or submassive PE.

REFERENCES

References (1)

1. Morrone D, Morrone V. Acute Pulmonary Embolism: Focus on the Clinical Picture. *Korean Circ J*. 2018;48(5):365–17. doi:10.4070/kcj.2017.0314.
2. Rivera-Lebron B, McDaniel M, Ahrar K, et al. Diagnosis, Treatment and Follow Up of Acute Pulmonary Embolism: Consensus Practice from the PERT Consortium. *Clin Appl Thromb Hemost*. 2019;25:1076029619853037. doi:10.1177/1076029619853037.
3. Stavenuiter XJR, Lubberts B, Prince RM III, Johnson AH, DiGiovanni CW, Guss D. Postoperative Complications Following Repair of Acute Achilles Tendon Rupture. *Foot Ankle Int*. 2019;40(6):679-686. doi:10.1177/1071100719831371.
4. Makhdom AM, Garceau S, Dimentberg R. Fatal Pulmonary Embolism following Achilles Tendon Repair: A Case Report and a Review of the Literature. *Case Reports in Orthopedics*. 2013;2013(3):1-3. doi:10.1155/2013/401968.
5. Nilsson-Helander K, Thurin A, Karlsson J, Eriksson BI. High incidence of deep venous thrombosis after Achilles tendon rupture: a prospective study. *Knee Surg Sports Traumatol Arthrosc*. 2009;17(10):1234-1238. doi:10.1007/s00167-009-0727-y.
6. Bernal AG, Fanola C, Bartos JA. Management of PE. American College of Cardiology. <https://www.acc.org/latest-in-cardiology/articles/2020/01/27/07/42/management-of-pe>. Published January 27, 2020. Accessed December 4, 2020.
7. Carroll BJ, Beyer SE, Mehegan T, et al. Changes in Care for Acute Pulmonary Embolism Through A Multidisciplinary Pulmonary Embolism Response Team. *AJM*. 2020;133(11):1313-1321.e1316.
8. Duffett L, Castellucci LA, Forgie MA. Pulmonary embolism: update on management and controversies. *BMJ*. 2020;18:m2177–23. doi:10.1136/bmj.m2177.
9. Furfaro D, Stephens RS, Streiff MB, Brower R. Catheter-directed Thrombolysis for Intermediate-Risk Pulmonary Embolism. *Annals ATS*. 2018;15(2):134-144. doi:10.1513/AnnalsATS.201706-467FR.
10. Leentjens J, Peters M, Esselink AC, Smulders Y, Kramers C. Initial anticoagulation in patients with pulmonary embolism: thrombolysis, unfractionated heparin, LMWH, fondaparinux, or DOACs? *Br J Clin Pharmacol*. 2017;83(11):2356-2366. doi:10.1111/bcp.13340.
11. Ortel TL, Neumann I, Ageno W, et al. American Society of Hematology 2020 guidelines for management of venous thromboembolism: treatment of deep vein thrombosis and pulmonary embolism. *Blood Adv*. 2020;4(19):4693-4738. doi:10.1182/bloodadvances.2020001830.
12. Pollack C, Schreiber D, Goldhaber SZ, et al. Clinical Characteristics, Management, and Outcomes of Patients Diagnosed With Acute Pulmonary Embolism in the Emergency Department. *Journal of the American College of Cardiology*. 2011;57(6):700-706. doi:10.1016/j.jacc.2010.05.071.
13. Rali PM, Criner GJ. Submassive Pulmonary Embolism. *Am J Respir Crit Care Med*. 2018;198(5):588-598. doi:10.1164/rccm.201711-2302CI.
14. Smith SB, Geske JB, Maguire JM, Zane NA, Carter RE, Morgenthaler TI. Early Anticoagulation Is Associated With Reduced Mortality for Acute Pulmonary Embolism. *Chest*. 2010;137(6):1382-1390. doi:10.1378/chest.09-0959.
15. Kline JA, Kabrhel C. Emergency Evaluation for Pulmonary Embolism, Part 2: Diagnostic Approach. *JEM*. 2015;49(1):104-117. doi:10.1016/j.jemermed.2014.12.041

References (2)

16. Kline JA, Hernandez-Nino J, Jones AE, Rose GA, Norton HJ, Camargo CA. Prospective Study of the Clinical Features and Outcomes of Emergency Department Patients with Delayed Diagnosis of Pulmonary Embolism. *Academic Emergency Medicine*. 2007;14(7):592-598. doi:10.1197/j.aem.2007.03.1356.
17. Glober N, Tainter CR, Brennan J, et al. Use of the d-dimer for Detecting Pulmonary Embolism in the Emergency Department. *JEM*. 2018;54(5):585-592. doi:10.1016/j.jemermed.2018.01.032.
18. Kline JA, Courtney DM, Kabrhel C, et al. Prospective multicenter evaluation of the pulmonary embolism rule-out criteria. *Journal of Thrombosis and Haemostasis*. 2008;6(5):772-780. doi:10.1111/j.1538-7836.2008.02944.x.
19. Wells PS, Anderson DR, Rodger M, et al. Excluding pulmonary embolism at the bedside without diagnostic imaging: management of patients with suspected pulmonary embolism presenting to the emergency department by using a simple clinical model and d-dimer. *Ann Intern Med*. 2001;135(2):98-107. doi:10.7326/0003-4819-135-2-200107170-00010.
20. Kabrhel C, Mark Courtney D, Camargo CA Jr., et al. Factors Associated With Positive D-dimer Results in Patients Evaluated for Pulmonary Embolism. *Academic Emergency Medicine*. 2010;17(6):589-597. doi:10.1111/j.1553-2712.2010.00765.x.
21. American College of Emergency Physicians Clinical Policies Subcommittee (Writing Committee) on Thromboembolic Disease., Wolf SJ, Hahn SA, et al. Clinical Policy: Critical Issues in the Evaluation and Management of Adult Patients Presenting to the Emergency Department With Suspected Acute Venous Thromboembolic Disease. *Annals of Emergency Medicine*. 2018;71(5):e59-e109. doi:10.1016/j.annemergmed.2018.03.006.
22. Keller K, Beule J, Balzer JO, Dippold W. D-Dimer and thrombus burden in acute pulmonary embolism. *American Journal of Emergency Medicine*. 2018;36(9):1613-1618. doi:10.1016/j.ajem.2018.01.048.
23. Geissenberger F, Schwarz F, Probst M, et al. D-Dimer Predicts Disease Severity but Not Long-Term Prognosis in Acute Pulmonary Embolism. *Clin Appl Thromb Hemost*. 2019;25(4):107602961986349-7. doi:10.1177/1076029619863495.
24. Saleh J, El-Othmani MM, Saleh KJ. Deep Vein Thrombosis and Pulmonary Embolism Considerations in Orthopedic Surgery. *Orthopedic Clinics of NA*. 2017;48(2):127-135. doi:10.1016/j.ocl.2016.12.003.
25. Lapidus LJ, Ponzer S, Pettersson H, de Bri E. Symptomatic venous thromboembolism and mortality in orthopaedic surgery - an observational study of 45 968 consecutive procedures. *BMC Musculoskeletal Disorders*. 2013;14(1):1-1. doi:10.1186/1471-2474-14-177.
26. Croskerry P. The importance of cognitive errors in diagnosis and strategies to minimize them. *Academic Medicine*. 2003;78(8):775-780. doi:10.1097/00001888-200308000-00003.
27. Zöller B, Pirouzifard M, Memon AA, Sundquist J, Sundquist K. Risk of pulmonary embolism and deep venous thrombosis in patients with asthma: a nationwide case-control study from Sweden. *Eur Respir J*. 2017;49(2):1601014. doi:10.1183/13993003.01014-2016.
28. Lee P-H, Fu P-K. Pulmonary Embolism and Severe Asthma: Case Report and Literature Review. *Medicina*. 2019;55(10):647-6. doi:10.3390/medicina55100647.
29. Majoor CJ, Kamphuisen PW, Zwinderman AH, et al. Risk of deep vein thrombosis and pulmonary embolism in asthma. *Eur Respir J*. 2013;42(3):655-661. doi:10.1183/09031936.00150312.
30. Suzuki T, Lyon A, Saggari R, et al. Editor's Choice-Biomarkers of acute cardiovascular and pulmonary diseases. *European Heart Journal: Acute Cardiovascular Care*. 2015;5(5):416-433. doi:10.1177/2048872616652309.

References (3)

31. Shopp JD, Stewart LK, Emmett TW, Kline JA. Findings From 12-lead Electrocardiography That Predict Circulatory Shock From Pulmonary Embolism: Systematic Review and Meta-analysis. Jones AE, ed. *Acad Emerg Med*. 2015;22(10):1127-1137. doi:10.1111/acem.12769.
32. Moore AJE, Wachsmann J, Chamarthy MR, Panjikaran L, Tanabe Y, Rajiah P. Imaging of acute pulmonary embolism: an update. *Cardiovasc Diagn Ther*. 2018;8(3):225-243. doi:10.21037/cdt.2017.12.01.
33. Carroll BJ, Heidinger BH, Dabreo DC, et al. Multimodality Assessment of Right Ventricular Strain in Patients With Acute Pulmonary Embolism. *The American Journal of Cardiology*. 2018;122(1):175-181. doi:10.1016/j.amjcard.2018.03.013.
34. Tapson VF, Weinberg AS. Treatment, prognosis, and follow-up of acute pulmonary embolism in adults. In: Mandel J, Hockberger R, Finlay G, eds. *UpToDate*. Waltham, MA; 2020.
35. Aggarwal V, Nicolais CD, Lee A, Bashir R. *Acute Management of Pulmonary Embolism*. 2017. <https://www.acc.org/latest-in-cardiology/articles/2017/10/23/12/12/acute-management-of-pulmonary-embolism>.
36. Vanni S, Viviani G, Baioni M, et al. Prognostic Value of Plasma Lactate Levels Among Patients With Acute Pulmonary Embolism: The Thrombo-Embolism Lactate Outcome Study. *Ann Emerg Med*. 2013;61(3):330-338. doi:10.1016/j.annemergmed.2012.10.022.
37. Larson PA, Berland LL, Griffith B, Kahn CE, Liebscher LA. Actionable Findings and the Role of IT Support: Report of the ACR Actionable Reporting Work Group. *Journal of the American College of Radiology*. 2014;11(6):552-558. doi:10.1016/j.jacr.2013.12.016.
38. American College of Radiology. *ACR Practice Parameter for Communication of Diagnostic Imaging Findings*. 2020:1-8.
39. Viertel VG, Trotter SA, Babiarz LS, et al. Reporting of Critical Findings in Neuroradiology. *American Journal of Roentgenology*. 2013;200(5):1132-1137. doi:10.2214/AJR.12.9041.
40. Wallis A, McCoubrie P. The radiology report - Are we getting the message across? *Clinical Radiology*. 2011;66(11):1015-1022. doi:10.1016/j.crad.2011.05.013.
41. Rosovsky R. Changes in treatment and outcomes after creation of a pulmonary embolism response team (PERT), a 10-year analysis. *Journal of Thrombosis and Thrombolysis*. 2018;47(1):31-40. doi:10.1007/s11239-018-1737-8.
42. Xenos ES, Davis GA, He Q, Green A, Smyth SS. The implementation of a pulmonary embolism response team in the management of intermediate- or high-risk pulmonary embolism. *J Vasc Surg Venous Lymphat Disord*. 2019;7(4):493-500. doi:10.1016/j.jvsv.2018.11.014.
43. Wright C, Elbadawi A, Chen YL, et al. The impact of a pulmonary embolism response team on the efficiency of patient care in the emergency department. *Journal of Thrombosis and Thrombolysis*. 2019;48(2):331-335. doi:10.1007/s11239-019-01875-0.
44. Collins SJ, Newhouse R, Porter J, Talsma A. Effectiveness of the surgical safety checklist in correcting errors: a literature review applying Reason's Swiss cheese model. *AORN J*. 2014;100(1):65-79.e65. doi:10.1016/j.aorn.2013.07.024.
45. Reason J. Education and debate. Human error: models and management. *BMJ*. 2000;320:768-770.