Victorian Institute of Forensic Medicine

Clinical Communiqué

VOLUME 4. ISSUE 1.
March 2017 Edition
ISSN 2204 - 0099

CONTENTS

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Next Edition: June 2017

EDITORIAL

Venous thromboembolism (VTE), in the form of pulmonary embolus (PE) or deep vein thrombosis (DVT), is a disease entity that from the time it was first described in the 19th century by Virchow, has generated a substantial amount of deliberation and debate, and is likely to continue to do so well into the future.

In this issue of the Clinical Communiqué, we focus on PE as the single, specific cause of death. As featured in the three cases presented, PE is a diagnosis that can occur in any healthcare setting, from general practice, to the emergency department, to the post-operative ward. It is a diagnosis that every healthcare practitioner needs to be familiar with to adequately detect and treat it in their patients, every time.

So, why do so many of us continue to grapple with the concepts surrounding diagnosis and management? Decision tools have been created, the reliability of clinical gestalt has been explored, and yet clinically significant cases of PE continue to be missed. Is there an acceptable miss rate? What are the risks of over-investigation? There are too many conundrums and differing scholarly views to adequately explore in this editorial, however, one important point that deserves mentioning is that if you do not think of a PE, you are going to miss it.

A striking feature of the cases in this issue is the persistence of the warning signs and symptoms - hypoxia, tachycardia, calf pain, and breathlessness. The signs and symptoms were not the same in each case, and could result from many other conditions, but they persisted. Pulmonary embolus was not considered, and was subsequently missed with fatal consequences.

Associate Professor David Mountain is an Emergency Physician who includes thromboembolic research amongst his prolific clinical and academic endeavours in emergency medicine. He has also provided expert opinions on this issue's topic to assist in coronial investigations. In his commentary, David addresses some of the complexities around diagnosis, and offers a practical outline to approaching the patient with a possible PE.

Finally, the coronial investigations applied in the three cases are worth noting. The first two cases were closed after an initial investigation without proceeding to inquest. The third case differed in that the coroner ran a joint inquest into the deaths of two patients from pulmonary embolus. These serve as a reminder of the prevention role of a coronial inquiry, whereby the aim is to reduce the number of preventable deaths through the findings of an investigation. If a coroner considers that a court hearing will not add to the information already gleaned from a hospital review or expert statements, and will not contribute to a broader understanding of patient safety measures, then an inquest will not be required. Similarly, if a coroner identifies a number of cases where there is commonality in the issues around patient safety that need exploring, then a joint inquest may be held.

CASE #1 NOT GOING TO PLAN

Case Number:
2014/2408 QLD
Case Précis Author:
Carmel Young RN

CLINICAL SUMMARY

Mr SM was a 32 year old obese male with an intellectual disability and a recent ankle injury. A week before his death, Mr SM was admitted to hospital with acute appendicitis and peritonitis. He underwent emergency surgery and returned to the ward before midnight where he remained on low flow oxygen. In the early hours of the next morning he was tachycardic and hypotensive. His calves were checked and noted to be soft.

The following day, Mr SM showered himself sitting on a chair. He appeared to need encouragement to mobilise. He was found to have low oxygen saturations (SaO2) at 85% and required oxygen via a mask to maintain SaO2 of 97%. On post-operative day three, Mr SM developed abdominal pain with distension, and was diagnosed with a paralytic ilieus. A nasogastric tube was inserted which eased his condition. However, he experienced periods of hypoxia with SaO2 levels of 78-80% whenever he removed his oxygen.

Over the next couple of days, Mr SM had ongoing tachycardia and hypoxia, with episodes of abdominal pain. He was seen by the surgeon and diagnosed with aspiration pneumonia. His pain levels gradually improved as did his bowel function, but he remained dependent on low flow oxygen. On post-operative day six, Mr SM complained of dizziness while mobilising to the bathroom. He sat on a chair, became unresponsive, and despite resuscitative efforts, was unable to be revived.

PATHOLOGY

An autopsy revealed a large saddle embolus in the pulmonary trunk, originating from deep vein thrombi in the right calf (which was 2.5cm larger than the left calf). Microscopic examination reported that the thrombi and emboli were a few days old. The pathologist identified Mr SM's post-operative state, previous inflammatory condition, obesity, and immobility as risk factors for the development of deep vein thrombosis.

INVESTIGATION

The coroner had access to the Root Cause Analysis (RCA) completed by the health service to assist in the investigation, and also engaged an independent medical practitioner in Clinical Forensic Medicine to review the case.

The RCA team found that certain post-operative observations were not recorded on every occasion, some were not trended, and some scores were not added up correctly. The RCA concluded that had an early warning observation tool been completed properly, it would have flagged Mr SM for review more frequently, highlighting the persistently low saturations.

Mr SM was a general surgical patient admitted to the orthopaedic ward as an outlier, which meant the treating team reviewed him on an ad-hoc basis. At the time, the surgical handover did not have a formal structured approach, and staff would often leave during the handover to commence theatre lists or clinics.

Another area of concern was the use of oxygen without medical input. In most cases nursing staff reported low saturations to medical staff but there was no evidence that the reports raised significant concerns or were escalated. Mr SM was on oxygen for six days following his surgery. The continual and unrestricted use of oxygen masked the underlying problem that an otherwise healthy 32 year old male with no medical history could not maintain adequate saturations without supplemental oxygen.

The RCA recommended the implementation of oxygen prescribing within the health service and changes to the National Inpatient Medication Chart to facilitate oxygen prescribing.

The independent medical opinion considered that while Mr SM received appropriate prophylaxis for VTE (subcutaneous heparin and compression stockings), the management of post-operative hypoxia was lacking. The medical practitioner did not agree that the chest x-ray showed evidence of aspiration pneumonia. He considered that blood gases should have been obtained and earlier diagnosis may have changed the outcome for Mr SM.

CORONER'S FINDINGS

The coroner found that there was a failure to identify and appropriately investigate the cause Mr SM's persistently low oxygen saturations. This arose from a combination of systemic issues which resulted in a failure to escalate earlier and more frequent reviews of Mr SM. The coroner was satisfied that the health service had taken action to rectify the issues the RCA highlighted, and did not proceed to inquest.

KEYWORDS

Pulmonary embolus, deep vein thrombosis, post-operative, root cause analysis, hypoxia

ACKNOWLEDGEMENTS

This initiative has been made possible by collaboration with the Department of Forensic Medicine (DFM), Monash University, Victorian Institute of Forensic Medicine (VIFM) and Victorian Managed Insurance Authority (VMIA).

FEEDBACK

The editorial team is keen to receive feedback about this communication especially in relation to changes in clinical practice. Please email your comments, questions and suggestions to: cc@vifmcommuniques.org

CASE #2 WHEN IT'S NOT IN THE DIFFERENTIAL

Case Number:
2013/1504 QLD
Case Précis Author:
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FFCFM (RCPA), FACEM

CLINICAL SUMMARY

Mr SB was a 38 year old male who presented to his GP to obtain a referral letter for an appointment with a respiratory physician, scheduled for the following day. Mr SB had a history of bipolar affective disorder, asthma, and idiopathic thrombocytopaenic purpura (ITP) which occurred 20 years earlier. He had a body mass index of 30 (normal range 18.5-25) and had been a smoker. Mr SB told his GP that he had experienced breathlessness for six months and it had worsened over the past week. He also reported left-sided pleuritic chest pain. The GP noted that Mr SB was febrile, tachypnoeic, tachycardic, with an SaO2 of 96% and reduced air entry at the base of his left lung on auscultation. The GP advised Mr SB to attend the emergency department immediately for a CXR and intravenous antibiotics.

Mr SB arrived at the emergency department approximately two hours later, at 16:30 hours. At triage, his vital signs were: BP111/48, HR 132, RR 28, T 37.9, SaO2 96%. He was seen by a medical resident at 16:34 hours, who then notified a senior medical officer about his presentation at 16:50 hours. Mr SB was moved to a resuscitation cubicle at 16:55 hours where he received oxygen via a Hudson mask, intravenous fluids and antibiotics. A CXR demonstrated consolidation and pulmonary congestion. An ECG showed sinus tachycardia and left axis deviation.

Mr SB deteriorated further at 17:30 hours and became confused, diaphoretic and agitated. An emergency staff specialist reviewed Mr SB at this stage. He was febrile with cold mottled skin and readings of: HR 150, RR 30, SaO2 88%. He was placed on non-invasive pressure support ventilation while the treating team prepared to intubate him and contacted the intensive care consultant.

An intensive care registrar attended to assist. Intubation was difficult due to the presence of fluid in the airway, however Mr SB was successfully intubated on the third attempt at 18:30 hours. He went into cardiac arrest with pulseless electrical activity at 18:34 hours. Cardiopulmonary resuscitation was commenced immediately and continued for approximately 70 minutes.

A bedside echocardiogram was performed by a cardiologist which showed no contractility of the heart. The attending team considered the possible diagnoses to be toxins, drugs, atypical infections, and pre-existing cardiomyopathy. Mr SB never regained a perfusing rhythm and was declared deceased at 19:55 hours.

PATHOLOGY

Mr SB's cause of death was unclear so was reported to the coroner. His family were shocked by his sudden and unexpected death and expressed concern about the care he had received. At autopsy, the pathologist found pulmonary thromboemboli and lung infarcts, bilateral calf muscle deep venous thrombi, dilated cardiomyopathy, mild obesity and pulmonary oedema. There was no evidence of any infective process.

INVESTIGATION

As the cause of death was established at autopsy, the coroner focussed the investigation on the medical treatment that was provided to Mr SB. Statements were obtained from the staff involved, and independent expert statements were sought from a forensic medicine clinician, an intensivist, and a professor of emergency medicine. Mr SB's family engaged two further independent experts – an emergency medicine specialist and a vascular surgeon.

The emergency medicine specialist and vascular surgeon engaged by the family both formed the view that PE should have been among the early differential diagnoses and that the ECG gave strong clues to the diagnosis. The emergency medicine specialist opined that CT angiography should have been arranged and that thrombolysis would have prevented death.

The vascular surgeon added that the history of ITP increased Mr SB's risk of thromboembolism, and had heparin been administered when he was moved to the resuscitation cubicle, he may have survived.

The forensic medicine clinician was more circumspect in his opinion, suggesting that the ECG changes were non-specific and did not favour one diagnosis over another, and that thrombolysis may not have altered the outcome.

The clinician also highlighted that under the circumstances, it was reasonable that Mr SB had not been treated for a PE prior to his cardiac arrest when a team of three emergency doctors, two intensive care doctors and a cardiologist all considered pneumonia to be the cause of his presentation.

His view was supported by the intensivist who pointed out that based on a validated clinical prediction rule, the clinical probability of PE when Mr SB presented to hospital was not high.

The professor of emergency medicine was given the opportunity to comment on the assertions raised by each of the other experts. The professor agreed that Mr SB's calculated risk for PE was low and that pneumonia was an appropriate working diagnosis.

Regarding the question of thrombolysis, the professor referred to a number of guidelines to explain that thrombolysis was not indicated in the setting of an undifferentiated cardiac arrest, and that Mr SB did not demonstrate hypotension or shock, which would have been the only indication for thrombolysis (had a PE been identified). The suggestion of ITP being a risk factor was considered unlikely given that it had been inactive for many years. The professor was of the opinion however, that PE should have been considered as a potential differential diagnosis, but that given Mr SB's rapid decline in the emergency department, any pathology or imaging tests to confirm or rule out the diagnosis would not have been available in time to change the course of events. When Mr SB deteriorated, the treating team appropriately prioritised resuscitative measures over diagnostic procedures.

The coroner heard that the hospital had conducted its own investigation into the case and as a result of their review had implemented a number of changes. These included education sessions on PE for junior medical staff, and the development of an algorithm for the diagnosis of PE.

CORONER'S FINDINGS

The coroner acknowledged that the expert opinions, though at times differing in views, all contributed to a greater understanding of the background and sequence of events in this case.

The coroner concluded that the differences in opinion, particularly with relation to whether the correct diagnosis should have been established, and whether thrombolysis was indicated or could have prevented death, were unlikely to bring about any useful recommendations to improve patient safety if explored further at inquest. The hospital had already addressed many of the recommendations made in its own clinical review. The coroner therefore closed the case without inquest.

KEYWORDS

Pulmonary embolus, deep vein thrombosis, emergency department, expert opinion, thrombolysis

CASE #3 EXCLUDING A LIFE-THREATENING DIAGNOSIS

Case Number:
0314/2012 and 1796/2012 SA

Case Précis Author:
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MBBS, GDipForensMed, DRANZCOG, DDU, FACEM

Two active, middle-aged individuals died from pulmonary thromboembolism. Although unrelated to each other, both consulted their own GP on more than one occasion, had unrecognised risk factors for developing thrombosis, were not diagnosed, and died at home. Their deaths were investigated concurrently by the coroner.

Mr PB

CLINICAL SUMMARY

Mr PB was a 52 year old male with a history of hypertension that was managed by his GP. He presented to his GP six days after a right knee arthroscopy with a sore right calf. An ultrasound excluded a DVT as the cause of the pain.

Mr PB was free of symptoms for the following ten weeks before again presenting to his GP with two days of right calf pain; this was to be two weeks before his death. No physical signs of a DVT could be found following a thorough physical examination; furthermore, the Wells score was zero, indicating a low probability of DVT.

A persistent concern for the coroner throughout the inquest was whether or not a DVT could be excluded on clinical symptoms and signs alone.

His GP attributed the pain to muscular strain resulting from a walk four days beforehand and referred him to see a physiotherapist. During this consultation Mr PB informed his GP about a DVT he had suffered a number of years ago.

The physiotherapist consulted Mr PB on six occasions. During the initial consultation, no mechanism was discovered that might have caused a muscular injury; he was therefore referred back to the GP for re-consideration of the diagnosis. A second GP saw Mr PB on this occasion who misread Mr PB's [past history of right] "PH R DVT", as "no DVT".

After examining Mr PB, it was still concluded that a muscular strain was the most likely cause of his calf pain. Over the next week his leg symptoms resolved, other than a swollen sensation over his right foot. Eleven days after seeing the second GP, Mr PB collapsed and died at home.

PATHOLOGY

Post-mortem examination revealed Mr PB had massive acute bilateral PE and early bilateral infarcts. There was also extensive DVT identified within the right leg that had features of recent clot and clot that was one to three weeks of age.

MS JW

CLINICAL SUMMARY

Ms JW was an active 63 year old female whose only medication was hormone replacement therapy (HRT). Ms JW returned from her annual overseas trip one month prior to consulting her GP for symptoms of chest pain, palpitations and left calf pain. Her GP noted that she was stressed. He examined her calf and concluded that there was a muscle strain for which an anti-inflammatory was given. An ECG was normal and blood tests were indicative of diabetes and a follow-up glucose tolerance test was arranged.

The expert suggested that if there was any thought of a DVT, the only way to exclude the diagnosis was with an objective ultrasound examination.

Ms JW consulted her GP a further three times over the next ten days regarding her blood test results. During these visits she complained of shortness of breath and cough for which new asthma medications were prescribed. She did not appear to be troubled by calf pain.

Ms JW's symptoms appeared to resolve according to her family members and as recorded by her GP during the final consultation, which was two months before her sudden death at home.

PATHOLOGY

Post-mortem examination of Ms JW revealed massive acute bilateral pulmonary thromboembolism as well as other emboli in her pulmonary arteries ranging in ages from a couple of months to a few days old. Several right lung infarcts of various ages were also found.

INVESTIGATION

Regarding the coronial investigation into the deaths of Mr PB and Ms JW, the coroner considered it appropriate to *"conduct concurrent inquests due to a number of features of commonality between the causes and circumstances of their respective deaths. There is no other connection between the two deceased persons"*.

The coroner's focus during the investigation was identifying factors that might prevent future similar deaths. A persistent concern for the coroner throughout the inquest was whether or not a DVT could be excluded on clinical symptoms and signs alone.

Oral testimonies were heard from the general practitioners and the physiotherapist involved in the cases. The pathologist who conducted Ms JW's post-mortem provided a detailed overview of the sources of PE and pathophysiology of the disease at inquest.

An intensivist considered to have specific experience in the field of thrombosis and haematosis was called as an independent expert witness. Although he had past limited experience as a general practitioner, the coroner accepted his expertise, stating *"the diagnosis and identification of a DVT is a matter that is intrinsic to the general practice of medicine and requires the same professional rigour as in any other medical setting"*.

It is incumbent on doctors to consider the possibility of a DVT when there is no overt explanation for calf pain

The intensivist gave evidence that there were *"obvious clues"* to the development of VTE in both cases. However, although the possibility of a DVT was raised for Mr PB, the diagnosis was subsequently missed because further investigation was not arranged. The expert suggested that if there was any thought of a DVT, the only way to exclude the diagnosis was with an objective ultrasound examination. He went on to state that the absence of signs was not a reliable means to exclude the diagnosis of a DVT.

CASE #3 EXCLUDING A LIFE-THREATENING DIAGNOSIS (Continued)

The use of the Wells score* was explored at inquest and the expert stated that the score was derived from hospital experience and therefore had greater applicability to hospital patients. The coroner heard that the presence of an alternative diagnosis that is more likely than a DVT reduces the Wells score by 2.

The coroner questioned how an alternative diagnosis could be considered more likely than a DVT when a patient presents with symptoms suggestive of a DVT without a sensible explanation, such as recent trauma to the leg.

That general practitioners accord significant weight to the opinions of other healthcare practitioners such as physiotherapists who are providing treatment to their patients.

The Wells score was used to assess the probability of a DVT being present in Mr PB's case. Although the score was calculated as zero, it did not take into account his past history of DVT. The coroner noted that such a past history is an acknowledged risk factor for future DVTs and therefore the Wells score should not have been afforded much weight in the exclusion of a DVT.

The intensivist pointed out that in Ms JW's case, VTE as a cause for her symptoms was not considered at all, nor were risk factors sought that would have included her recent travel and HRT. The shortness of breath she complained about was an important red flag, and the apparent resolution of her calf pain was not necessarily reassuring, as it may have represented clot migration to the lungs.

CORONER'S FINDINGS

The coroner made a number of findings in relation to the diagnosis of DVT:

- It is incumbent on doctors to consider the possibility of a DVT when there is no overt explanation for calf pain;
- The cause of unexplained calf pain should not be diagnosed on clinical grounds alone; and
- The suspicion of a DVT, as distinct from the probability of it, ought to prompt further diagnostic consideration.

The coroner concluded that a DVT should have been diagnosed in each case and that both deaths were preventable. A number of recommendations were made including:

- The Royal Australian College of General Practitioners develop guidelines regarding the diagnosis of DVTs and PEs, including the prescription of a low threshold for diagnostic imaging and/or D-dimer blood testing; and
- That general practitioners accord significant weight to the opinions of other healthcare practitioners such as physiotherapists who are providing treatment to their patients.

AUTHOR'S COMMENTS

This concurrent investigation into two deaths occurring outside the hospital setting has several important learnings that are transferrable to almost every medical subspecialty. Readers are encouraged to consider this case in the context of their own workplace.

KEYWORDS

Pulmonary embolus, deep venous thrombosis, general practice, Wells score, calf pain

RESOURCES

CASE #2

Kline JA, Kabrhel C. Emergency Evaluation for Pulmonary Embolism, Part 1: Clinical Factors that Increase Risk. *J Emerg Med* 2015; 48(6): 771-780.

Kline JA, Kabrhel C. Emergency Evaluation for Pulmonary Embolism, Part 2: Diagnostic Approach. J *Emerg Med* 2015; 49(1): 104-117.

CASE #3

Pulmonary embolism: assessment and imaging. Skinner S. *Aust Fam Physician* 2013; 42(9): 628–632. Available at: https://www.racgp.org.au/afp/2013/september/pulmonary-embolism/

* A clinical prediction rule for estimating the pre-test probability of DVT. Score ≥ 3 = high (53% prevalence); score 1-2 = moderate (17% prevalence); score ≤ 0 = low (5% prevalence). See https://www.mdcalc.com/wells-criteria-dvt

EXPERT COMMENTARY LESSONS FROM CORONIAL CASES ON VENOUS THROMBOEMBOLISM

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The cases presented in this issue are a timely reminder of the significant challenges in diagnosing venous thromboembolism (DVT and PE), and the potential for fatal outcomes if that opportunity is missed. Almost universally there is agreement that both DVT and PE are difficult to diagnose. The difficulties are mainly due to the variety of possible presenting symptoms and signs, almost all of which can be attributed to a broad range of differential diagnoses.

Research in primary care suggests patients with VTE delay seeking medical attention on average 3-4 days after symptom onset (which may falsely reassure their doctor that it is not a life-threatening issue). There may then be another delay of 2-4 days after initial review before they are diagnosed.

Very few patients present with the full house of clinical features and many have only minor symptoms and signs and minimal risk factors. This can be seen from studies that divide patients with possible PE into three risk groups (low, intermediate and high) using expert clinical judgement or validated scoring systems e.g. Wells Score (https://www.mdcalc.com/wells-criteria-pulmonary-embolism), or Revised Geneva Score (https://www.mdcalc.com/geneva-score-revised-pulmonary-embolism). Low risk groups have only a 3-10% rate of PE, but up to 25% of all VTE are diagnosed from this group, as 50-70% of patients assessed for VTE are considered low risk. Intermediate risk patients have a 15-30% PE rate but constitute up to 50% of all diagnosed PE, as 30-40% of patients are assigned to this group. Patients categorised as high risk have 40-70% PE diagnosis rates, but these "full house" features are seen in only 5-15% of assessed patients, with only 20-30% of PE diagnosed from this group.

There are some very important issues that clinicians need to keep in the forefront of their minds when assessing a patient for VTE. The acceptable rate of missed PE/DVT is <2-3%.

This rate has been set by contrasting the risks of radiation, dye, false positive tests and excessive treatment/bleeding versus the risks of missed VTE. No clinician (even true thrombosis experts) can reliably exclude VTE to <3% on clinical grounds alone, except where a definitive alternative diagnosis can be made. Examples include: an obvious cellulitis or calf tear by history and exam; or a proven pneumothorax or pneumonia on CXR; or ECG findings for AMI or pericarditis; that all clearly fit the clinical picture. If these definitive findings are seen, and documented, it is safe to exclude DVT/PE. This is why all guidelines emphasise the need for good history, clinical examination and routine non-invasive investigations e.g. CXR, bloodwork, ECG etc. The other acceptable pathway to avoiding further testing for PE is where patients are assessed as low risk for PE using an acceptable risk assessment rule (Wells/Geneva), then have the validated 8-point PERC rule applied (https://www.mdcalc.com/perc-rule-pulmonary-embolism). It is vital that when clinicians use these rules they apply them as designed (see 'Tips to Remember').

High risk VTE patients should have immediate imaging and be started on treatment unless they have major bleeding risks. In general practice, patients designated high risk should be immediately transferred by ambulance to hospital.

Most patients with a non-high risk of PE should have a high sensitivity D-dimer first if available that day (preferably within 3 hours). The patient must be told to attend hospital emergently if symptoms worsen. A negative highly sensitive D-dimer (talk to your lab) reliably excludes PE in non-high risk patients and low risk DVT patients.

High risk DVT patients should have same day ultrasound scanning but if unavailable, they can be started on full treatment (heparin/novel oral anticoagulants) until tested, unless they have a high bleeding risk, when they should be managed in hospital.

Another important issue raised by these cases is that patients at significantly increased risks of thrombosis (e.g. previous VTE, recent high risk surgery, leg immobilisation/ trauma, active cancer, late pregnancy/ post-partum etc.) should have lowered thresholds for investigation. Firstly, they are at increased risk, but secondly, it is highly unlikely that missing a VTE diagnosis in these patients will be considered defensible.

The presence of multiple other potential causes for the patient's symptoms e.g. hypoxia in the post-operative/ post-partum period, is not a defence if clear, well-known life threats were not excluded with definitive testing (ultrasound, CT pulmonary angiogram or ventilation-perfusion scans). If a post-operative/ post-partum patient has unexplained respiratory distress or chest pain, it is crucial to find the cause, and make sure that treatment improves the patient's condition (e.g. if symptoms are attributed to atelectasis, then physiotherapy and/or analgesia should rapidly improve oxygenation). Remember, it is complex patients with multiple competing causes where a diagnosis of DVT and PE is frequently delayed or missed.

RESOURCES

Raja A, et al. Evaluation of Patients with Suspected Acute Pulmonary Embolism: Best Practice Advice from the Clinical Guidelines Committee of the American College of Physicians. *Ann Intern Med* 2015; 163(9): 701-711. Available at: http://annals.org/aim/article/2443959/evaluation-patients-suspected-acute-pulmonary-embolism-best-practice-advice-from.

Konstantinides S, at al. 2014 ESC Guidelines on the diagnosis and management of acute pulmonary embolism: The Task Force for the Diagnosis and Management of Acute Pulmonary Embolism of the European Society of Cardiology (ESC) *Eur Heart J* 2014; 35(43): 3033-3073. Available at: http://www.escardio.org/Guidelines/Clinical-Practice-Guidelines/Acute-Pulmonary-Embolism-Diagnosis-and-Management-of.

Walen S, Damoiseaux RA, Uil SM, van den Berg JW. Diagnostic delay of pulmonary embolism in primary and secondary care: a retrospective cohort study. *Br J Gen Pract* 2016; 66(647): e444-50. Available at: http://bjgp.org/content/66/647/e444.

Geersing GJ, et al. Safe exclusion of pulmonary embolism using the Wells rule and qualitative D-dimer testing in primary care: prospective cohort study. *BMJ* 2012; 345: e6564. Available at: https://www.ncbi.nlm.nih.gov/pubmed/23036917.

TIPS TO REMEMBER

Know the exclusions to the rules and remember –

- You cannot add or subtract parts just because you think they are important or are inconvenient. If you cannot find the information or do the test, or are unsure, presume that it is positive and err on the side of caution.
- Whenever VTE is a differential without a definitive alternative diagnosis (or cannot be excluded using PERC), the patient needs further testing. All patients should be risk stratified, using a validated scoring system unless you are clearly an expert. If you have to think about it – you are not!