Ambulatory management of pulmonary embolism

ABSTRACT

The diagnosis of pulmonary embolism can be very difficult and elusive. It depends greatly on the use of diagnostic tests, which are in turn interpreted according to a pre-test clinical probability. These include non-specific tests such as the chest X-ray and electrocardiograph, which help exclude other conditions such as pneumonia or myocardial infarction. On the other hand, more specific tests such as computed tomography or ventilation/perfusion scanning are used to confirm or exclude the diagnosis of pulmonary embolism. The condition is potentially fatal, and in the past patients with suspected pulmonary embolism constituted a significant number of hospital admissions. Despite this, the majority were found not to have pulmonary embolism. More recently, studies have suggested that most patients with suspected pulmonary embolism who are haemodynamically stable can be safely managed on an ambulatory pathway. Therefore, there is a paradigm shift towards investigating and treating pulmonary embolism in the outpatient setting. This article discusses the ambulatory pathway of the diagnosis and treatment of pulmonary embolism.

Pulmonary embolism is a common, potentially life-threatening condition. Presentation is variable, with many non-specific findings, making diagnosis a challenge (Laack and Goyal, 2004). Furthermore, no single blood test conferring high specificity and sensitivity exists to aid diagnosis, compared to relatively highly sensitive troponin I in acute coronary syndromes or amylase in acute pancreatitis (Douketis, 2001). This article covers the wider scope of pulmonary embolism diagnosis and management in the medical assessment unit, with special emphasis on ambulatory care.

Dr Houssam Abusibah, CT2 in Acute Medicine, Department of Acute Medicine, James Cook University Hospital, Middlesbrough

Dr Muntasir M Abdelaziz, Consultant Respiratory Physician and Clinical Lead for General Internal Medicine, Department of Respiratory Medicine, Tameside Hospital, Ashton-Under-Lyne

Dr Peter Standen, Registrar in Acute Medicine, Department of Acute Medicine, James Cook University Hospital, Middlesbrough

Dr Praveen Bhatia, Consultant Respiratory Physician, Department of Respiratory Medicine, Tameside Hospital, Ashton-Under-Lyne

Dr Mahir MA Hamad, Consultant Physician and Clinical Director for Acute Medicine, Department of Acute Medicine, James Cook University Hospital, Middlesbrough TS4 3BW

Correspondence to: Dr MMA Hamad (m.hamad@nhs.net)

Clinical probability

Calculating the clinical probability of pulmonary embolism is essential in the diagnostic process, influencing both yield and predictive value of subsequent investigations including ventilation/perfusion scanning and computed tomography pulmonary angiogram (Stein et al, 2007). Studies suggest that objective assessment of clinical probability can predict the likelihood of pulmonary embolism with great accuracy (Wells et al, 1998). There are different systems available, the two-level Wells score being most widely accepted in the UK. Most rely on the presence or absence of thromboembolism risk factors and whether an alternative diagnosis explains the clinical presentation. The National Institute for Health and Care Excellence (2015) guidelines recommend the use of the Wells score. Kline et al (2004) developed initial assessment parameters to help exclude pulmonary embolism (pulmonary embolism rule-out criteria). These criteria include eight variables:

1. Age <50 years
2. Pulse <100 beats per minute
3. Oxygen saturation ≥95%
4. No haemoptysis
5. No oestrogen use
6. No surgery or trauma requiring hospitalization within 4 weeks
7. No previous venous thromboembolism
8. No unilateral leg swelling.

Pulmonary embolism rule-out criteria have been suggested for use in low-probability patients to avoid the need to measure D-dimer or perform further imaging. A meta-analysis found that the pooled sensitivity of pulmonary embolism rule-out criteria is 97.2%, with pulmonary emboli potentially being missed in 0.32% (44 of 13 855 cases) (Singh et al, 2012).

Subsequently, Kline et al (2013) suggested that the presence of chest pain, postpartum status and pregnancy are factors whereby pulmonary embolism rule-out criteria may be more likely to miss pulmonary embolism. Although pulmonary embolism rule-out criteria are not included in National Institute for Health and Care Excellence guidance, these criteria are recommended by the American College of Physicians before requesting D-dimer to avoid unnecessary imaging (which may lead to false-positive results) when suspicion of pulmonary embolism is low (Raja et al, 2015).

Investigations

The investigations used in diagnosis of pulmonary embolism are divided into two categories: non-specific and specific tests. Non-specific tests help by raising or lowering the clinical suspicion of pulmonary embolism. Specific tests differ in sensitivity, and as such are used to exclude the diagnosis of pulmonary embolism.

Non-specific tests

Chest radiograph

Several radiographic abnormalities are described in association with pulmonary embolism (*Figure 1*). However, these are non-specific and cannot diagnose pulmonary embolism (Stein et al, 2007). In most cases the chest X-ray is normal. Nevertheless, chest X-ray is useful in excluding other aetiologies, e.g. pneumothorax or pneumonia. Furthermore, it may be useful in later ventilation/perfusion interpretation (Konstantinides et al, 2014).

Electrocardiograph

The electrocardiograph is non-specific and non-diagnostic of pulmonary embolism, but helps exclude other conditions, e.g. myocardial infarction and pericarditis (Davies et al, 2003). The commonest pulmonary embolism-associated electrocardiograph finding is sinus tachycardia.

Arterial blood gases

Typically, pulmonary embolism results in hypoxaemia and hypocapnia. However, several studies show that pulmonary embolism can present with normal gases, especially in young people with good respiratory reserve (Rodger et al, 2000). It is not common practice to measure arterial blood gases in stable patients with suspected pulmonary embolism.

Specific tests for pulmonary embolism

D-dimer

Unlike myocardial infarction or acute coronary syndrome, there is no blood test to diagnose pulmonary embolism. Levels of D-dimer, a derivative of degraded fibrin, are raised in thromboembolism and other conditions, including trauma, infections and neoplasia (Davies et al, 2003). Consequently, a positive D-dimer test cannot diagnose pulmonary embolism. Despite this, D-dimer has a high negative predictive value, especially if combined with pre-test clinical probability discussed earlier (Konstantinides et al, 2014; National Institute for Health and Care Excellence, 2015). Therefore, negative testing with low clinical probability can essentially exclude pulmonary embolism and obviate further investigation. However, with high pre-test clinical probability, further investigation is required regardless of the D-dimer level. It is suggested that negative D-dimer fails to exclude pulmonary embolism in over 15% of patients with high clinical probability (Konstantinides et al, 2014).

The negative predictive value of D-dimer depends on the assay used. Latex agglutination has the least negative predictive value, while ELISA (enzyme-linked immunosorbant assay) has the best. Red blood cell agglutination has an intermediate negative predictive value compared with the other methods (Davies et al, 2003).

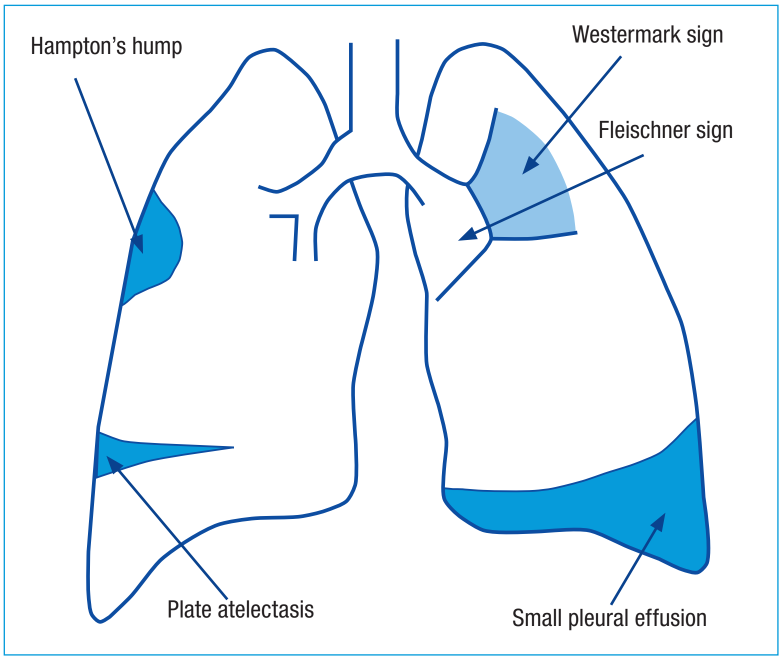

Figure 1. The chest radiography in pulmonary embolism is usually normal. This figure shows the abnormalities that may be encountered in patients with pulmonary embolism. Hampton's hump represents pleural-based pulmonary infarction. Fleischner sign is a prominent and amputated pulmonary artery, while Westermark sign indicates peripheral oligaemia distal to Fleischner sign. Other chest X-ray findings in pulmonary embolism include plate atelectasis and small pleural effusion. From Abdelaziz et al (2012).

Ultrasound of the lower limb

Compression ultrasound of the legs was suggested to be positive in one-third of patients with pulmonary embolism (Stein et al, 1991), but may aid diagnosis of pulmonary embolism where computed tomography pulmonary angiogram or ventilation/perfusion scan are contraindicated (computed tomography in patients with severe renal impairment or contrast allergy; ventilation/perfusion in patients with abnormal chest X-ray). Nevertheless, a negative ultrasound scan result does not exclude pulmonary embolism.

Ventilation and perfusion nuclear scan

The Prospective Investigation of Pulmonary Embolism Diagnosis (PIOPED) study demonstrated that the positive predictive value of a high-probability ventilation/perfusion scan interpretation is very high; likelihood of pulmonary embolism in this group reached 87%. Additionally, the negative predictive value of a low-probability ventilation/perfusion is very high, so this safely excludes pulmonary embolism (PIOPED Investigators, 1990).

Interestingly, incorporating pre-test clinical probability improves the diagnostic value of the ventilation/perfusion scan significantly (combining concordant clinical and ventilation/perfusion probabilities increases or decreases the positive predictive value from 87% to 96% (for high probability) and 14% to 4% (for low probability) respectively (PIOPED Investigators, 1990)). Unfortunately, 57% of patients with suspected pulmonary embolism have intermediate ventilation/perfusion probability, which has a low diagnostic value necessitating further investigations.

Although computed tomography pulmonary angiogram is the investigation of choice for pulmonary embolism, ventilation/perfusion plays a role in diagnosis of pulmonary embolism in patients with contrast allergy or renal impairment.

Computed tomography pulmonary angiogram

Compared with ventilation/perfusion scanning, computed tomography pulmonary angiogram has many advantages. It is now the gold-standard investigation in diagnosing or excluding pulmonary embolism in most centres (Rathbun et al, 2000), benefitting from speed and reduced interpreter variation. It can provide additional imaging of iliac vessels and inferior vena cava without requiring additional contrast, albeit with higher radiation exposure to the reproductive organs (Srivastava et al, 2004) and is not influenced by chest X-ray abnormalities, often providing alternative diagnoses (e.g. pneumonia or neoplasia).

Computed tomography pulmonary angiogram is also helpful in assessing pulmonary embolism severity (see below); hence, it is the investigation of choice in suspected massive and sub-massive pulmonary embolism (British Thoracic Society Standards of Care Committee Pulmonary Embolism Guideline Development Group, 2003).

The main disadvantage of computed tomography pulmonary angiogram is reduced sensitivity in detecting sub-segmental emboli. The clinical implication of missing sub-segmental emboli is as yet unclear. It has been shown that multi-detector computed tomography scanners are more sensitive in detecting sub-segmental emboli than older scanners (Stein et al, 2006). Other disadvantages include risk of contrast nephropathy and allergy.

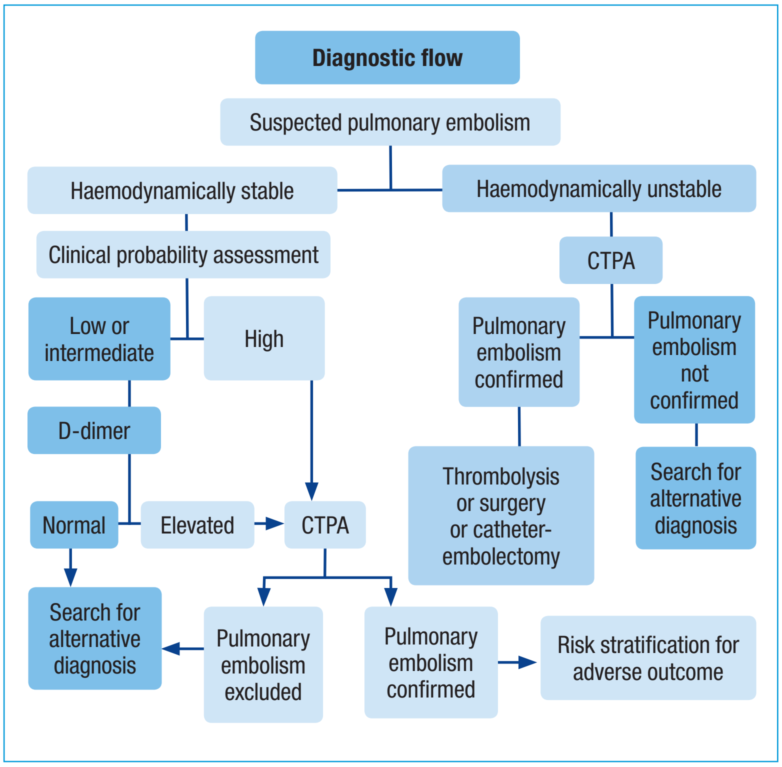

Figure 2. Diagnostic workup of suspected pulmonary embolism. CTPA = computed tomographic pulmonary angiography.

As with ventilation/perfusion, studies have shown the diagnostic value of computed tomography pulmonary angiogram improved when combined with clinical probability. These studies suggest that with high clinical probability, the negative predictive value of computed tomography pulmonary angiogram in excluding pulmonary embolism is relatively less when compared with its negative predictive value in patients with low or intermediate probability (Musset et al, 2002). The British Thoracic Society Standards of Care Committee Pulmonary Embolism Guideline Development Group (2003) guideline states that it is safe to withhold anticoagulation in patients with good quality negative computed tomography pulmonary angiogram.

Significantly, PIOPED II examined the role of multi-detector computed tomography pulmonary angiogram in diagnosis of pulmonary embolism in 773 patients, using a composite reference standard to exclude pulmonary embolism, including ventilation/perfusion showing a high probability of pulmonary embolism, a positive digital subtraction angiogram or positive lower limb compression venous ultrasonography with non-diagnostic ventilation/perfusion scan (Gottschalk et al, 2002). In this study, the sensitivity and specificity of computed tomography pulmonary angiogram were 83% and 93% respectively. It also demonstrated that the sensitivity and specificity of computed tomography pulmonary angiogram is increased to 90% and 95% respectively when combined with venous imaging. Similar to ventilation/perfusion scanning, the study also showed that computed tomography pulmonary angiogram sensitivity improved when combined with clinical assessment and pre-test probability (Gottschalk et al, 2002; Stein et al, 2006).

Diagnostic approach

The authors encourage direct GP referral of patients with suspected pulmonary embolism to ambulatory care provided they are normotensive with oxygen saturations above 94% (*Figure 2*). However, initial GP assessment is required to avoid sending unstable patients to ambulatory care. Pre-test clinical probability of pulmonary embolism should be determined before requesting D-dimer. D-dimer should only be used in those with low pre-test clinical probability (two-level Wells score). A negative D-dimer effectively excludes pulmonary embolism and no further tests are required. Excessive non-judicial D-dimer testing can lead to unnecessary imaging without improving diagnostic outcome. The use of pulmonary embolism rule-out criteria may help reduce D-dimer requests among patients with a low index of suspicion for pulmonary embolism. It is also important to think laterally and consider alternative diagnoses. In reality most patients referred with suspected pulmonary embolism do not have pulmonary embolism, so an alternative diagnosis should be made.

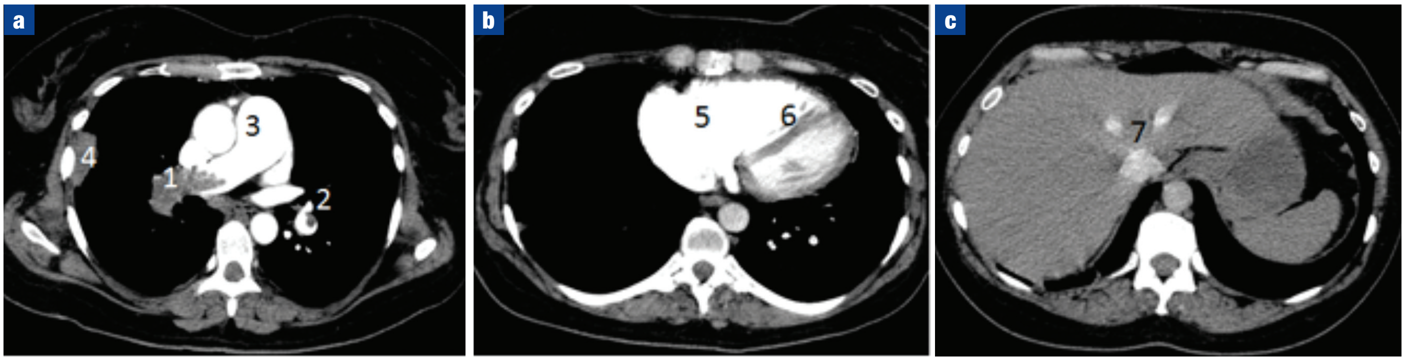

Figure 3. Computed tomographic pulmonary angiography scan of a patient showing **(a)** a large massive pulmonary embolism in the right pulmonary artery (1), an embolus in a left segmental artery (2), a large pulmonary artery trunk diameter greater than the aorta (3), a pleural-based pulmonary infarction (Hampton's hump) in the right side (4), **(b)** dilated right ventricle which is larger than the left ventricle size (5), bowing of the inter-ventricular septum to the left (6) and **(c)** reflux of contrast in the inferior vena cava and hepatic veins (7).

Further tests include computed tomography pulmonary angiogram or ventilation/perfusion scanning. The latter could be considered in patients with normal chest X-ray and no cardiopulmonary disease to reduce cost. Low probability ventilation/perfusion interpretation can safely exclude pulmonary embolism with low pre-test clinical probability. A high probability ventilation/perfusion scan is very suggestive of pulmonary embolism especially when combined with high pre-test clinical probability. Intermediate probability ventilation/perfusion scan interpretation is regarded as equivocal, requiring further investigation (Gottschalk et al, 2002).

Conversely, good quality negative computed tomography pulmonary angiogram virtually excludes pulmonary embolism when the clinical probability is low. It must be emphasized that no diagnostic approach replaces good clinical acumen and expertise.

Risk stratification and ambulatory care

Mortality and morbidity outcomes following acute pulmonary embolism vary according to multiple factors. Post-pulmonary embolism mortality ranges from 1–30%, therefore a robust risk stratification system is required to help guide management and treatment options, such as appropriate patient selection for ambulatory *vs* in-hospital management, or anticoagulation *vs* thrombolysis.

Comorbidities and clinical presentation

Registry data (Goldhaber et al, 1999) regarding pulmonary embolism outcomes show that age and associated comorbidities (e.g. chronic obstructive pulmonary disease, cancer) have higher incidences of complications and mortality. Following this, pulmonary embolism severity assessment tools were developed such as the pulmonary embolism severity index (Choi et al, 2009), simplified pulmonary embolism severity index (Uresandi et al, 2007) and criteria from the Hestia study (Zondag et al, 2011).

Right ventricular status

Right ventricular dysfunction is associated with higher mortality and risk of complications such as chronic pulmonary hypertension (McConnell et al, 1996). The right ventricle can be assessed in several ways:

Echocardiography

Echocardiography is used to evaluate pulmonary embolism severity rather than diagnostically. Abnormalities observed in pulmonary embolism include right ventricular dysfunction, tricuspid regurgitation and regional wall motion abnormalities (Stein et al, 2006). McConnell's sign (regional wall motion abnormalities sparing the apex) is suggestive of pulmonary embolism with 77% sensitivity and 95% specificity, although this is not used to diagnose pulmonary embolism in clinical practice (McConnell et al, 1996). Transoesophageal echocardiography can detect pulmonary embolism in the right heart and main pulmonary artery, but again is not commonly used diagnostically (Torbicki et al, 1997). The authors request echocardiography if troponin or brain natriuretic peptide levels are raised, electrocardiograph is abnormal or to help risk stratify to avoid missing sub-massive pulmonary embolism. However, the importance of clinical acumen and discretion must be emphasized.

Computed tomography pulmonary angiogram

Signs of right ventricular dysfunction on computed tomography pulmonary angiogram are shown in *Figure 3b* and include:

- Increased right ventricle:left ventricle diameter ratio on axial sections or reconstructed four-chamber views. Studies showed increased in-hospital, 30-day and 3-month mortality if ratio >0.9 on computed tomography (Kang et al, 2011). Although right ventricular dimension on computed tomography pulmonary angiogram has more prognostic value than thrombus size or central location (Meinel et al, 2015), radiologists invariably tend to comment more on the latter points.
- Flattening or left-sided bulging of the interventricular septum
- Contrast reflux into inferior vena cava and hepatic vein (Abdelaziz et al, 2012).

Cardiac biomarkers

Troponin: Troponin rise is indicative of right ventricular myocardial necrosis, correlating with right ventricular dysfunction and increased incidence of cardiogenic shock and in-hospital mortality (Konstantinides et al, 2014).

Other novel biomarkers, including heart-type fatty acid–binding protein, cardiac expression of growth differentiation factor-15 and D-dimer, also have prognostic value but are not currently used clinically.

The authors' practice is to request biomarkers in every patient referred to ambulatory care. Computed tomography pulmonary angiogram may take 1–2 days, so it is important to differentiate patients with potential for sub-massive pulmonary embolism requiring inpatient care. Generally biomarker results are available before computed tomography pulmonary angiogram (and therefore definitive pulmonary embolism diagnosis); this may help guide the potential appropriateness of ambulatory management.

Natriuretic peptides: Both brain natriuretic peptide and N-terminal pro-brain natriuretic peptide levels rise in response to stretch and/or ventricular pressure, indicating right ventricular dysfunction or failure. Studies suggest elevated levels predict short-term mortality and morbidity in patients with pulmonary embolism (Konstantinides et al, 2014).

Treatment of pulmonary embolism in ambulatory care

Based on clinical presentation and risk stratification, two major groups are identified:

Haemodynamically unstable patients (massive pulmonary embolism)

Haemodynamic instability is defined as sustained hypotension (systolic blood pressure <90 mmHg or >40 mmHg drop for >15 minutes) or peri-cardiopulmonary arrested patients (Piazza and Goldhaber, 2010). All current evidence shows that patients with massive pulmonary embolism benefit from thrombolysis (Goldhaber, 2002; Konstantinides et al, 2014; Meinel et al, 2015; National Institute for Health and Care Excellence, 2015).

Haemodynamically stable patients

These are patients who are normotensive on presentation. This is further subdivided into:

1. Submassive pulmonary embolism with evidence of right ventricular dysfunction and/or evidence of myocardial necrosis
2. Low-risk pulmonary embolism.

Submassive pulmonary embolism

Although haemodynamically stable, patients with submassive pulmonary embolism tend to be more symptomatic; clinical examination may reveal evidence of right ventricular dysfunction or pulmonary hypertension (e.g. loud S2, parasternal heave). There may be electrocardiograph changes e.g. S1-Q3-T3, raised biomarkers, and echocardiography will reveal right ventricular dysfunction. Subsequent computed tomography pulmonary angiogram would evidence large central thrombus with right ventricular dysfunction.

Patients with sub-massive pulmonary embolism are usually haemodynamically stable and may be difficult to differentiate from those with low-risk pulmonary embolism who are suitable for ambulatory care. The literature outlines several different approaches. Investigations which demonstrate right ventricular dysfunction (echocardiogram or computed tomography pulmonary angiogram) or myocardial damage (troponin and brain natriuretic peptide) can be useful. Unfortunately, out-of-hours echocardiogram is not routinely available acutely, and computed tomography pulmonary angiogram may take 1–2 days to perform. In such circumstances, clinicians may rely on myocardial damage markers such as troponin or brain natriuretic peptide – often more sensitive than electrocardiograph. However, a meta-analysis demonstrated that the prognostic value of troponin in a normotensive patient is low (Jiménez et al, 2009). Conversely, a more recent study suggested that high sensitivity troponin T has better prognostic value in pulmonary embolism, and may help identify those who likely to develop more adverse complications secondary to pulmonary embolism (Lankeit et al, 2010).

As these tests are more readily available than imaging, the authors use high sensitivity troponin as a screening test to help identify patients more likely to develop adverse complications and therefore unsuitable for ambulatory care. Nevertheless, a meta-analysis demonstrated that cardiac biomarkers (troponin, brain natriuretic peptide, N-terminal probrain natriuretic peptide or heart-type fatty acid-binding protein) cannot differentiate with certainty between patients who may benefit from thrombolysis or inpatient management *vs* those suitable for ambulatory care (Bajaj et al, 2015). Therefore it must be emphasized that high sensitivity troponin testing should be used in clinical context with the risk stratification score.

Unfortunately, there is no clear consensus on management of sub-massive pulmonary embolism (Konstantinides et al, 2014). Trials show that thrombolysing these patients is associated with rapid haemodynamic stability, clot resolution and subsequent symptom relief. However, no significant improvement in mortality is demonstrated.

Two important studies have attempted to further investigate this. PEITHO is a multicentre, double-blinded, placebo-controlled randomized trial (*n*=1006). The trial compared tenecteplase and heparin therapy (*n*=506) *vs* placebo and heparin therapy (*n*=499) in sub-massive pulmonary embolism. Thrombolysis resulted in a 3% absolute risk-reduction of death or haemodynamic collapse, but with higher major haemorrhage rates (Meyer and PEITHO Investigators, 2014).

The second study was the MOPETT trial (Sharifi et al, 2013); recruited patients had diagnosed 'moderate' pulmonary embolism based on clot burden, perfusion defects, symptoms and signs. Right ventricular dysfunction and cardiac biomarkers were excluded in risk stratification. All 101 patients received heparin. The study groups also received thrombolysis at half-therapeutic dose, as the lungs receive 100% of cardiac output, compared to the 5% and 15% that the heart and brain receive respectively. Pulmonary hypertension at 28 months was reduced in the thrombolysis group (16% *vs* 57%). Recurrent pulmonary embolism at 28 days (16% *vs* 32%) and hospital stay (2.2% *vs* 4.9%) were also significantly reduced. This requires further evaluation in larger trials.

In light of this, the recommendation stands that patients in this group should not routinely receive thrombolytic therapy, but clinical judgment be used to identify individuals who might benefit from thrombolysis. Regardless, sub-massive pulmonary embolism should always be treated as an inpatient. If thrombolysis is not given, the patient should be monitored for signs of increasing right ventricular dysfunction or pulmonary hypertension, considering thrombolysis if the patient deteriorates.

Patients with sub-massive pulmonary embolism are normally haemodynamically stable and may be difficult to differentiate from those with low-risk pulmonary embolism who are suitable for ambulatory care. In this setting the authors rely on investigations, particularly echocardiogram and myocardial damage markers such as troponin and brain natriuretic peptide. Unfortunately, out-of-hours echocardiogram is not routinely available acutely in most hospitals; the authors feel this investigation should be available for such emergencies.

Low-risk pulmonary embolism

- No right ventricular dysfunction or evidence of myocardial necrosis
- Haemodynamically stable.

Studies and guidelines suggest that patients with low-risk pulmonary embolism can be managed safely in outpatients (British Thoracic Society Standards of Care Committee Pulmonary Embolism Guideline Development Group, 2003; Konstantinides et al, 2014; National Institute for Health and Care Excellence, 2015). The introduction of non-vitamin K-antagonist oral anticoagulants has encouraged more centres to manage low-risk patients with pulmonary embolism in the ambulatory setting (Akin et al, 2015).

The James Cook University Hospital experience

Since 1998, James Cook University Hospital, a large tertiary hospital in Middlesbrough, has been treating low-risk patients with suspected pulmonary embolism in the ambulatory care setting (*Figure 4*). The outcome to date has remained successful (Hamad et al, 2011). Patients must fulfil certain criteria to be eligible for outpatient treatment and have negative troponin and brain natriuretic peptide tests (*Table 1*).

Pre-test clinical probability and risk stratification of pulmonary embolism should be determined (*Figure 4*). D-dimer is performed in those with low pre-test clinical

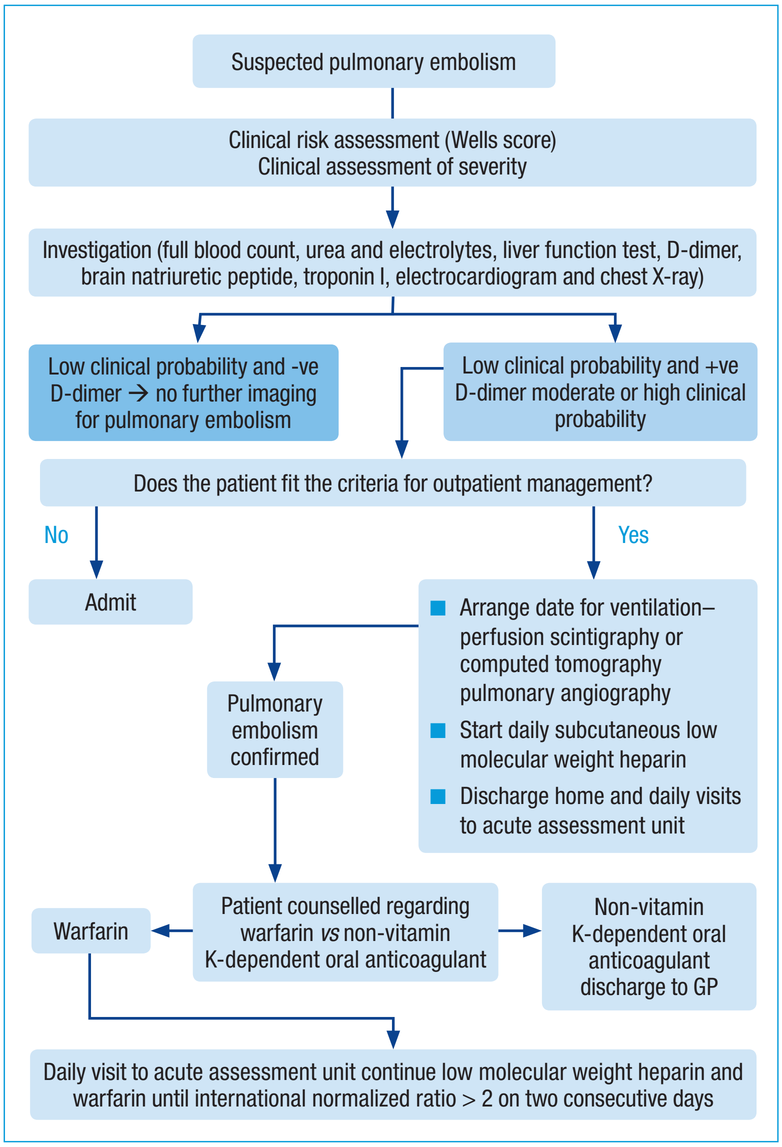

Figure 4. The diagnostic pathway used in James Cook University Hospital for patients with suspected pulmonary embolism.

probabilities. Patients with negative D-dimer are unlikely to have pulmonary embolism and no further tests are required.

Diagnostic tests such as computed tomography pulmonary angiogram and ventilation/perfusion scans are usually done within 2 days; during this time the patient will continue on therapeutic dose low molecular weight heparin, via either ambulatory care or in the community (administered by the patient him-/herself or by district nurses). The best way to obtain these investigations (computed tomography pulmonary angiogram and ventilation/perfusion) quickly is an agreement with radiology departments to have protected slots every day for ambulatory care.

Once pulmonary embolism is confirmed, the patient is counselled regarding anticoagulation and benefits and risks of warfarin *vs* non-vitamin K-antagonist oral anticoagulants are discussed. Patients on non-vitamin K-antagonist oral anticoagulants are discharged for GP monitoring. Patients

Table 1. Criteria for outpatient therapy at James Cook University Hospital

Eligible for outpatient therapy	■ >18 years of age ■ Outpatient follow-up feasible ■ No allergy to warfarin or heparin ■ No history of heparin-induced thrombocytopenia ■ Patient in stable condition ■ No acute syncopal episode ■ Oxygen saturation > 92% on room air ■ No prior cardiopulmonary disease ■ Haemodynamically stable (blood pressure >110/60 mmHg, pulse < 90 beats per minute, respiratory rate < 24 breaths/minute) ■ Chest pain managed with analgesics ■ Low bleeding risk ■ Absence of severe renal disease ■ Negative troponin
Exclude patients with	■ Coagulopathy ■ Active bleeding ■ Intracranial haemorrhage (anytime) ■ Gastrointestinal bleed, trauma or surgery within the last month ■ Platelets <50x10^9/litre

Services required for ambulatory management of pulmonary embolism	■ Anticoagulation clinic
	■ Administration of low molecular weight heparin via:
	  ■ Self injection education
	  ■ Community team administration of injection
	■ Ambulatory emergency care clinic follow-up

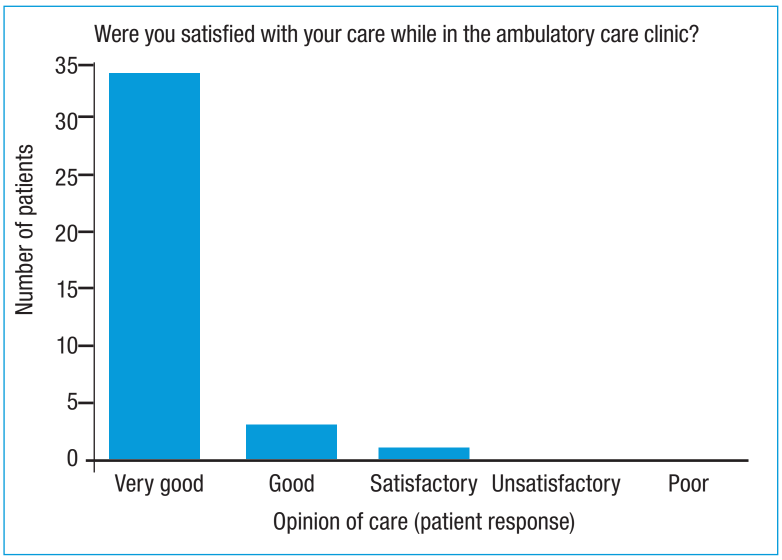

Figure 5. Results of patient satisfaction survey at the ambulatory emergency care clinic (James Cook University Hospital, Middlesbrough), July 2017. 50 patients surveyed – 38 responses.

on warfarin are referred to anticoagulation clinic or ambulatory care for rapid warfarin induction. This involves protocolised warfarin loading and daily international normalized ratio with therapeutic-dose low molecular weight heparin cover. Once the international normalized ratio is therapeutic (2.0–3.0) for 2 consecutive days, low molecular weight heparin is stopped and the patient is discharged (*Figure 4*).

Patient feedback regarding ambulatory management of pulmonary embolism has been universally positive (*Figure 5*), which is encouraging given the potential benefits to the hospital and patients involved.

Conclusions

Diagnosis of pulmonary embolism has been revolutionized with the introduction of multi-detector computed tomography pulmonary angiography, making ventilation/perfusion scanning virtually redundant. The exception to this as per PIOPED II is in high clinical probability patients with negative computed tomography pulmonary angiography where ventilation/perfusion scanning may play a role in exclusion. Another caveat is in poor pickup rates for sub-segmental pulmonary emboli, but a benefit from treatment for these is in itself unclear at present.

Ambulatory and home management of pulmonary embolism is becoming increasingly common, particularly with the widespread adoption of non-vitamin K-antagonist oral anticoagulants, but relies on effective risk stratification to exclude stable patients with sub-massive pulmonary embolism. This is complicated by the need for both myocardial damage markers (brain natriuretic protein, troponins) and echocardiography – the latter of which is generally not available out-of-hours in the acute setting. Provision of this needs to be addressed nationally in future. **BJHM**

Figure 1 *is reproduced from Abdelaziz et al (2012) with kind permission from the* Saudi Journal of Internal Medicine.
Conflict of interest: none.

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KEY POINTS

- Diagnostic tests for pulmonary embolism have improved markedly over the past 25 years, but all must be viewed in the overall clinical context.
- The introduction of new tools for risk stratification is as important as the actual diagnostics, and these are crucial in terms of selecting patients for potential outpatient treatment.
- Judiciously selected patients with non-massive pulmonary embolism can be safely treated as outpatients.
- This approach is popular with patients able to undergo ambulatory or home management for pulmonary embolism and feedback is generally positive.
- Pulmonary embolism outpatient management programmes can help provide significant savings of resources and beds.

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