Pulmonary embolism: assessment and imaging

Dear Editor

The diagnosis of pulmonary embolus (PE) remains difficult in any setting, and particularly so in primary care where the incidence is very low. We were pleased to read some of the content in the recent article by Skinner[1] in regard to choices of advanced imaging modalities; however we have some concerns about applying the PE assessment approach as outlined in this review. It has a three-arm stratification where there is no difference between two of the arms, and relies heavily on D-dimer as a differentiating test. In reality, especially in urban practice, the decision is not 'who to investigate', but 'who to send to the emergency department'. We commonly see and have to deal with the consequences of applying this pathway at face value.

We believe there should be more emphasis on clinical assessment without necessarily D-dimer in patients with low pretest probability (PTP). Indiscriminately testing all patients with low PTP with D-dimer will inevitably lead to unnecessary imaging and its consequences. False positives will greatly outnumber true positives, given the low specificity of this test and the low incidence of PE in the group being tested. Even quantitative D-dimer has specificity reported to be around 50%, meaning 50% of patients without the disease will have a positive result when tested, often leading to further work-up.

The proportion of patients with low PTP being worked up has been rising, and the incidence of diagnosed PE falling in PE studies for several decades. The PIOPED study in 1990 reported an overall incidence of PE of 33%.[2] More recent studies have found an overall incidence of PE as low as 5%, with the majority of patients in a low PTP group by clinical gestalt or Wells score 0–2.[3] One of the common problems we find with use of the Wells score is the variable of 'alternative diagnosis less likely than PE'. An alternative diagnosis must be more likely most of the time, despite being hard to make in hospital practice and even more so in primary care. 'Likely' is a variable influenced by clinical experience, medicolegal concerns and, rarely, by science.

It is important to understand the concept that there must be a group of patients with such low risk that the chances of a D-dimer helping the patient is less than the chances of it causing harm. Clinical decision aids, such as the Pulmonary Embolus Rule Out Criteria (PERC) by Kline, and clinical gestalt, are now recommended to define a group of patients that have PTP of PE below the test threshold for further work-up, including D-dimer testing.[4]

Harm from overzealous use of D-dimer includes having unnecessary advanced imaging that is either negative or false positive: ionising radiation, intravenous injection site complications, contrast nephropathy, complications of anticoagulant therapy given before confirmation of diagnosis and for incidental minor PE. Although not all minor PE needs treatment, as mentioned by Skinner, the diagnosis, once made, can have ongoing effects. There is potential harm from further investigation in attempts to find contributing factors to a diagnosis of PE, such as work-up for coagulation disorders, and malignancy. Harm can be from follow-up imaging studies and work-up of common incidental findings on CT pulmonary angiography such as lung nodules. We find when these potential adverse effects of further work-up for PE are discussed with many low PTP patients, as part of shared decision making, commonly the result is the patient not wanting to pursue further investigation. There is also the harm to the health system and other patients through consumption of financial resources and, in our area of practice, congestion in an already commonly overloaded emergency department.

Risk stratification and work-up of patients for possible PE brings into focus some important elements of clinical medicine. The potential harms of D-dimer as part of a strategy need careful consideration.

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Reply

Dear Editor

I thank Dr Bridgford for the detailed comments regarding my recent article 'Pulmonary embolism: assessment and imaging'. My main aim was to improve general practitioners' understanding of different imaging modalities so that they may be confident ordering appropriate imaging investigations and, in appropriate cases, commence management outside the hospital setting.

Dr Bridgford quite rightly emphasises the need to avoid overdiagnosis in a population where the prevalence of the disease being tested is low. This is particularly pertinent for women, especially those of childbearing age, who are at a relatively greater risk of adverse effects from radiation.

I agree with Dr Bridgford's recommendation that the combination of a simplified clinical decision rule and gestalt impression of clinical likelihood of venous thromboembolism should be the first step in assessment of patients presenting with symptoms of venous thromboembolism (VTE). Our experienced general practitioner colleagues already use this technique in their practice by dismissing VTE as a possible differential diagnosis in many cases. Stratification of low pretest probability (PTP) into a 'very low' and 'low' PTP may still be out of reach for junior and inexperienced practitioners.

Sarah Skinner

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Clubbing

Dear Editor

I refer to the letter from Christopher R Foerster (Clubbing should not be attributed to COPD, *AFP* March 2014), mainly to support his message that established finger clubbing in patients with chronic obstructive pulmonary disease (COPD) should prompt consideration of another pulmonary comorbidity.

The paper to which he refers,[1] showed that in a small subset of patient with COPD there was a significant right-to-left shunt and those patients had changes in the fingernails, which I interpreted as minimal or mild digital clubbing. These changes consisted of a filling out of the nail fold angle and a slight bending forward of the nail, but in no instance was the clubbing more than subtle. The point of the paper was to make this physiological point, which was in keeping with the general observation that conditions associated with significant right-to-left shunting (arteriovenous intrapulmonary malformations, cyanotic congenital heart disease) were associated with obvious clubbing.

Despite the WHO (2013)[2] definition of COPD, in 1964 the term 'chronic obstructive lung disease' referred to patients with irreversible airflow obstruction due to obstructive bronchitis and emphysema and my cohort certainly contained patients with emphysema. I was interested to see that Foerster included emphysema as one of the respiratory conditions to be considered as a cause of clubbing in the context of COPD!

I doubt that my tiny study is responsible for the erroneous reviews to which Foerster refers. Fifty years of thoracic medicine has taught me that COPD should not be on any list of causes of established finger clubbing.

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Osteoarthritis

Dear Editor

Thank you for the update on osteoarthritis ('Osteoarthritis: where are we for pain and therapy in 2013', *AFP* November 2014). Jones illustrates a common case in general practice of a patient with osteoarthritis of the knee. While there is evidence to support the use of corticosteroid injection, this is for short-term, clinically significant pain relief for up to 1 week.[1] We would suggest that repeated injections every 2 months for 2 years would be excessive and that alternative therapies be considered earlier.

The evidence is currently limited but other options of treatment include growth factor injections, platelet-rich plasma injections and high tibial osteotomy.[2] We agree with the author that treatment should be tailored to the individual but general practitioners need to be aware of other emerging non-arthroplasty treatments.

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Reply

Dear Editor

I thank Drs Leung and Sim for their interest in my article on osteoarthritis. The review they quoted concludes that there is only a short-term effect with corticosteroid injections in the knee. However, other evidence suggests there is a moderate effect overall[1] and a durable effect (lasting up to 24 weeks) in an Australian setting.[2] The long-term safety remains uncertain but a small MRI-based paper showed no structural harm of an injection every 3 months for 2 years, suggesting this frequency of injections is not excessive.[3] In my view, the total lack of good quality trials of the other therapies mentioned makes them of research interest only at this point in time and their promoters should provide this proof prior to rolling them out.

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Letters to the Editor

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