Editor’s choice

An end to depression in primary care?

I enjoyed Moscrop’s essay.1 As a GP Trainee in the early 70s with an interest in the psychological I was taught to distinguish between endogenous depression (no evident trigger, serious, chronic, more likely to respond to antidepressants) and reactive depression (for example, triggered by bereavement, relationship breakdown, or job loss, and less likely to respond to antidepressants). Inspired by the works of Michael Balint and Colin Murray Parks I tried to offer a listening ear to troubled patients in long appointments at the end of normal surgery times. There were inevitable disappointments, such as the newly-widowed lady who came to see me weekly over several months who plaintively asked on her last visit ‘So am I not getting any pills?’ An early addition to my Patient’s Unmet Needs list.

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REFERENCE
DOI: 10.3399/bjgp12X658142

Predicting risk of bladder cancer in the UK

We were interested to read the paper by Shepherd et al1 on the clinical features of bladder cancer in primary care, as we have previously published a paper2 in the BJGP where we reported on symptoms associated with renal tract cancer (of which around 80% were bladder cancers) in primary care. Both studies evaluated the risk or positive predictive value of symptoms for detecting cases of cancer, and both used UK primary care databases, but the study designs were different with Shepherd et al using a case-control approach while we used a cohort design.

A key finding of both studies is that haematuria is a strong predictor of bladder/renal cancer, with a stronger association at younger ages. Indeed we reported that around three-quarters of renal cancer cases had reported haematuria before their diagnosis. Both studies also found at least a doubling of risk in people consulting with abdominal pain. A strength of our study design was that we were able to combine the risks associated with the different symptoms and clinical features with the established risks associated with smoking, into an algorithm that can predict absolute risk in individuals presenting with symptoms. We also accounted for the steep increases in risk with age and for different risks in men and women, since rates in men are at least three times higher than in women. Crucially we validated this algorithm in a separate cohort of patients, and found that it discriminated extremely well between cancer cases and the remainder of the cohort (ROC values of 0.95 in men and 0.91 in women). The 10% of patients with highest risks contained 87% of all renal cancers diagnosed within 2 years. It is unclear how many cancer cases would be detected using the approach of Shepherd et al, nor is there any validation on an external dataset which is an essential step needed to determine whether the results are valuable.3

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Financial Disclosure Statement

JH-C is professor of clinical epidemiology at the University of Nottingham and co-director of QResearch, a not-for-profit organisation that is a joint partnership between the University of Nottingham and EMIS (leading commercial supplier of information technology for 60% of general practices in the UK). JH-C is also director of ClinRisk, which produces open and closed source software to ensure the reliable and updatable implementation of clinical risk algorithms within clinical computer systems to help improve patient care. CG is associate professor of medical statistics at the University of Nottingham and a consultant statistician for ClinRisk.
Over-reliance of D-dimer in isolation to exclude venous thrombosis should be avoided

The shift to primary care expected in the initial ‘diagnostic’ management of cases of venous thromboembolism is indeed welcome. A recent article in the BJGP highlighted the role of D-dimer in reducing referrals for radiological imaging. However, one of the messages that needs to be stressed in this context is the importance of clinical probability scoring system. It is important that the primary care physicians do not over-rely on the D-dimer, and clinical evaluation should be considered as the first step. Reliability on the D-dimer in isolation can have problems especially since there is evidence in the literature for thromboembolic episodes occurring in the context of normal D-dimer.\(^1\)\(^2\)\(^3\)

There are several possible explanations for a normal D-dimer even in the presence of venous thromboembolism. The levels of D-dimer increase in the circulation due to the breakdown of the fibrin-bound clots. Very often, individuals present with symptoms of lower limb thrombosis many days after the onset of symptoms. The clot breakdown in these cases may have ceased by the time they arrive for medical attention and the result would be a normal D-dimer. Second, in the patients who receive anticoagulation treatment sometimes before the hospital assessment is undertaken (patients who have problems with transport, or from the hospice, started on anticoagulation empirically), inhibition of clot lysis can cause normal D-dimer. This phenomenon has been noted to occur within 24 hours after receiving heparin therapy.\(^4\) It is also important to bear in mind that a normal cut-off of D-dimer is arbitrary and may not be applicable to every individual, since the clot-breakdown capacity varies between individuals. This is exemplified by the report in pregnancy of deep vein thrombosis and normal D-dimer.\(^5\) Last, there is the issue of wide variability between many different D-dimer assays.\(^6\) Each caregiver should take into consideration the appropriate cut-off suited for the assay and setting before they can attribute a level useful in exclusion of thrombosis.

In summary, there is no alternative to good clinical assessment in the exclusion of venous thromboembolism and D-dimer level is only a useful adjunct.

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DOI: 10.3399/bjgp12X658188

Eosinophilic oesophagitis: a clinical update

I would like to thank you for the recent clinical intelligence article on eosinophilic oesophagitis.\(^1\) As a current GPVTS working in ENT I found this clinical update very informative and relevant to my work. Interestingly only a few days after reading this article we admitted a 17-year-old young man complaining of a food bolus sensation following eating chicken earlier in the day. He was normally fit and well, and of note did not suffer with any atopic conditions. He was managed initially with medical therapy, however, after some initial improvement his symptoms deteriorated and the time between consumption and regurgitation of water progressively shortened.

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DOI: 10.3399/bjgp12X658179

Steam inhalation therapy
I think the conclusion of the article *Steam inhalation therapy: severe scalds as an adverse side effect*\(^1\) is excessively restrictive.

I do not know how ‘steam inhalation therapy’ is administered in the Netherlands, but I know practice in Britain has changed in the last four decades. We no longer use Nelson inhalers. Many patients inhale over a washing-up bowl of boiling water, which brings in risks of transporting water from kettle to bowl to accessible table. I recommend either the use of a mug-full of boiling water, or the less-risky ‘hot beverage’, that certainly does not induce ‘steaming’ in a bath or shower of normal bathing temperature, this is substantially less than boiling, and should not induce more scalds than the ordinary weekly ablutions.

I disagree with the article’s conclusion that there is no evidence of therapeutic benefit. There is a huge amount of anecdotal evidence for its therapeutic efficacy, in ENT and chest medicine in hospital as well as in general practice. I have never seen a scald from steaming. I have seen many from hot drinks: should we ban drinking?

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DOI: 10.3399/bjgp12X658160