Installing the NZF icon on the Medtech toolbar

Dear Editor,
How do we go about linking with the New Zealand Formulary in Medtech 32 PMS? Another surgery I worked at had an icon in Medtech that we clicked on and it came through to the website.

Practice Manager
Tauranga

Response from bpac^nz editorial team:

The New Zealand Formulary (NZF) provides health professionals in New Zealand with free, clinically validated information about medicines which enables them to select safe and effective treatments for individual patients. A number of clinicians prefer to be able to access the NZF directly from their Patient Management System. Instructions on how to install the NZF icon on the Medtech toolbar are available from:

www.nzformulary.org/wp-content/uploads/2012/10/NZFIcon.pdf

This process needs to be performed for each work station in the practice that requires an NZF icon installed.

Can urine dipstick be used to "rule-out" kidney disease in patients with mildly reduced renal function?

Greetings,
I am under a tree in Tapawera pursuing my usual holiday habit of catching up on bpac journals [Best Practice Journal] (which incidentally is why I prefer hard copy) and have reached the CKD article in Issue 66 [BPJ Feb, 2015]. My question is about dipsticks for patients with an eGFR between 60 and 89 mL/min/1.73m^2. Currently, if the lab reports a mildly reduced eGFR when performing cardiovascular risk assessments or testing renal function prior to medicine initiation, I arrange a urine dipstick and if negative for protein I just put them on annual recall. The article would suggest I should be sending the urines to the lab for ACR. There are quite a lot of these. Is a dipstick sufficient?
Thanks

Dr Emma Dunning, General Practitioner
Wellington

Response from bpac^nz editorial team:

Proteinuria is a sign of chronic kidney disease (CKD) as well as being an indicator for progressive CKD and future cardiovascular events.[1] Many clinicians will routinely use urine dipstick to test for proteinuria. However, New Zealand guidelines now recommend assessing patients with risk factors for CKD with urinary albumin:creatinine ratio (ACR), serum creatinine and blood pressure testing;[2] the addition of these tests to all diabetes screening and cardiovascular risk assessments is also recommended.[3] The frequency of CKD testing for patients with risk factors is:[2]

- At least every one to two years for patients without CKD
- At least every 12 months for patients with diabetes

ACR is the preferred method for quantifying proteinuria because:[1]

- Urinary dipstick is not sensitive enough to reliably detect proteinuria (see below)
- Albumin is the main protein excreted in the vast majority of proteinuric kidney disease
- ACR provides greater sensitivity in the detection of lower, but clinically significant, proteinuria compared with measures of total protein, i.e. protein:creatinine ratio (PCR)

Despite it being a rapid and simple point-of-care test the ability of urine dipstick to detect anything other than overt proteinuria is limited.[4] In a sample of urinalysis results for more than 10,000 Australian adults aged 25 years and older, a dipstick test result ≥ 1+ protein identified ACR ≥ 3.4 mg/mmol, i.e. microalbuminuria, with 57.8% sensitivity and 95.4% specificity, meaning four out of ten patients with microalbuminuria would be expected to return a false-negative result on urine dipstick testing.[5] When the threshold for detection was raised to ACR ≥ 33.9 mg/mmol, i.e. macroalbuminuria, the sensitivity of dipstick was increased to 98.9% with a specificity of 92.6%.[5]

The concern in using a negative result on urine dipstick to effectively "rule-out" clinically significant proteinuria in patients with reduced renal function is that while patients with

macroalbuminuria, who are admittedly at the highest risk, are likely to be identified, some patients with microalbuminuria may be missed.

Urine dipstick can, however, provide useful information in some situations in patients with reduced renal function. For example, dipstick testing for haematuria can provide useful diagnostic information.

It is acknowledged that routine ACR testing in all patients with a mildly reduced eGFR, i.e. 60–89 mL/min/1.73m^2, will include a substantial number of patients and an associated cost. It is important to remember that in New Zealand 7–10% of the adult population is estimated to have CKD;[3] with future rates of CKD expected to rise secondary to the increasing prevalence of obesity and diabetes, early detection in primary care is a priority. The overarching aim of CKD surveillance is to reduce the number of people reaching end-stage kidney disease and the resultant need for patient dialysis and kidney transplants.

For further information, see: "Interpreting urine dipstick tests in adults: a reference guide for primary care", BT (Jun, 2013) and "The detection and management of patients with chronic kidney disease in primary care", BPJ 66 (Feb, 2015).

References

1. National Health Service (NHS). Proteinuria: detection and quantification in adults using ACR - information for GPs. NHS: UK,. 2009. Available from: www.birminghamquality.org.uk/DLopen/proteinuria_GPs.pdf (Accessed Jun, 2015).
2. Kidney Health New Zealand. Chronic kidney disease (CKD): management in General Practice. 2013. Available from: www.kidneys.co.nz/resources/file/ckd_management_in_general_practice._2014_version.pdf (Accessed Dec, 2015).
3. Ministry of Health (MOH). Managing chronic kidney disease in primary care: national consensus statement. Wellington: Ministry of Health. 2015. Available from: www.health.govt.nz/publication/managing-chronic-kidney-disease-primary-care (Accessed Jun, 2015).
4. BMJ Best Practice. Assessment of proteinuria. 2015. Available from: http://bestpractice.bmj.com/best-practice/monograph/875.html
5. White SL, Yu R, Craig JC, et al. Diagnostic accuracy of urine dipsticks for detection of albuminuria in the general community. Am J Kidney Dis 2011;58:19–28. doi:10.1053/j.ajkd.2010.12.026

LABA without ICS in patients with COPD

Dear Editor

I have a question regarding COPD from issue 66 [BPJ Feb, 2015]. I was clearly in need of this article because I must be well out of date – in asthma we were told no LABA without ICS due to risk of sudden death – is this not the case for COPD?

Thanks

Emma Dunning, General Practitioner
Wellington

Response from bpac^{nz} editorial team:

Concerns about the use of long-acting beta$_2$-agonist (LABA) monotherapy for patients with asthma were first raised in the 1990s and confirmed by the Salmeterol Multicenter Asthma Research Trial (SMART), published in 2006. This was a 28-week, randomised, double-blind, placebo-controlled study comparing the safety of adding salmeterol or placebo to usual care in more than 26 000 patients with asthma aged over 12 years.1 The investigators found small, but statistically significant increases in respiratory-related deaths or life-threatening events in patients with asthma who were prescribed a salmeterol inhaler in addition to their normal treatment, which for some patients included inhaled corticosteroids (ICS);[1] the study was not designed to assess the effect of ICS treatment on patient outcomes. The finding from the SMART study was replicated and it was subsequently found that the increased risk of death in patients with asthma taking salmeterol was reduced with concomitant ICS treatment.[2] This resulted in the United States Food and Drug Administration (FDA) recommending that LABA monotherapy be contraindicated in the treatment of asthma.[3] The FDA also advised that LABAs should only be used as additional treatment for patients with asthma who were taking, but not receiving adequate control from, a long-term asthma control medicine such as ICS.[3]

In contrast to patients with asthma, LABA monotherapy has not been found to increase the risk of serious adverse events for patients with COPD.[3] Monotherapy with a LABA or long-acting muscarinic receptor antagonist (LAMA) is recommended in the stepwise management of COPD for patients with persistent dyspnoea.[4] Specifically, guidelines state that treatment with formoterol or salmeterol significantly improves FEV$_1$, lung volumes, dyspnoea, quality of life and exacerbation rate in patients with COPD, with no effect on mortality.[5] However, it is important to note that patients with asthma-COPD overlap syndrome (ACOS), i.e. patients with features of both asthma and COPD, should not be treated with LABA monotherapy.[5]

The reason why monotherapy with a LABA increases the risk of adverse events in patients with asthma but not in patients with COPD is uncertain. Eosinophilic airway inflammation due to allergic sensitisation and a T helper 2 lymphocyte-mediated immune response is a characteristic of asthma.[6] In the airways of patients with COPD a neutrophil response is typically present involving T helper 1 lymphocytes, often in association with bacterial colonisation.[6] However, some patients with COPD and ACOS also display eosinophilic airway inflammation.[6] Given the heterogeneity of asthma and COPD it is perhaps not surprising that different groups of patients with chronic airway diseases may not receive the same benefit from the same medicine.

For further information, see: "Are blood eosinophil counts helpful in predicting patient responses to inhaled corticosteroids in COPD?", Page 3 and "Newly-subsidised medicines for the treatment of patients with COPD", Page 7.

References

1. Nelson HS, Weiss ST, Bleecker ER, et al. The Salmeterol Multicenter Asthma Research Trial: a comparison of usual pharmacotherapy for asthma or usual pharmacotherapy plus salmeterol. Chest 2006;129:15–26. doi:10.1378/chest.129.1.15
2. Weatherall M, Wijesinghe M, Perrin K, et al. Meta-analysis of the risk of mortality with salmeterol and the effect of concomitant inhaled corticosteroid therapy. Thorax 2010;65:39–43. doi:10.1136/thx.2009.116608
3. United States Food and Drug Administration (FDA). FDA Drug Safety Communication: Drug labels now contain updated recommendations on the appropriate use of long-acting inhaled asthma medications called Long-Acting Beta-Agonists (LABAs). 2010. Available from: www.fda.gov/Drugs/DrugSafety/PostmarketDrugSafetyInformationforPatientsandProviders/ucm213836.htm
4. Abramson M, Frith P, Yang I, et al. COPD-X concise guide for primary care. 2014. Available from: www.copdx.org.au (Accessed Mar, 2016).
5. Global Initiative for Chronic Obstructive Lung Disease. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease (updated 2016). 2016. Available from: www.goldcopd.org/uploads/users/files/WatermarkedGlobal%20Strategy%202016%281%29.pdf (Accessed Mar, 2016).
6. George L, Brightling CE. Eosinophilic airway inflammation: role in asthma and chronic obstructive pulmonary disease. Ther Adv Chronic Dis 2016;7:34–51. doi:10.1177/2040622315609251

We value your feedback. Write to us at: Correspondence, PO Box 6032, Dunedin or email: editor@bpac.org.nz