

Letters

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Calcium

Editor, – I refer to the article 'Calcium supplementation: the bare bones' by J.D. Wark and C. Nowson (Aust Prescr 2003;26:126–7). I would like to ask on what information they base their assertion that calcium citrate is more expensive than calcium carbonate.

Calcium carbonate (Caltrate) and calcium citrate (Citracal) are both on the Pharmaceutical Benefits Scheme and their regulated price is identical.

These two products are largely prescribed on concession scripts for an identical cost, and are also regularly bought by consumers at an equal retail price of about \$12.

How then, can calcium citrate be more expensive?

David Haworth
Pharmacist
Kirrawee, NSW

Professor J. Wark, one of the authors of the article, comments:

It is true that the price of a 120-tablet pack of Citracal is the same as a 120-tablet pack of Caltrate. However, the former contains 250 mg elemental calcium while the latter contains 600 mg. This makes Citracal a substantially more expensive source of calcium, even if one accepts that it has somewhat better oral bioavailability than Caltrate (which is not a consistent finding in the literature). It is worth emphasising that consumers and prescribing doctors alike should check the elemental calcium content of supplements.

Off-label prescribing

Editor, – Craig Patterson and Brian Foster make some strong statements in Letters to the Editor (Aust Prescr 2003;26:51–2). Will pharmacists also be 'hung out to dry' and 'subjected to a compensation claim' for off-label dispensing?

I think it would be timely for *Australian Prescriber* to help clarify the situation with regard to off-label prescribing. The Australian Medicines Handbook uses the terms 'marketed indications' and 'accepted indications'. Do the professional indemnity organisations have an opinion here? Has the Therapeutic Goods Administration had any more recent thoughts than the (1992) reference quoted by Craig Patterson?

If I prescribe sodium valproate for prevention of migraine when other treatment has failed, use pethidine in the epidural space for obstetric analgesia or give ketorolac intravenously for post-operative pain control, where do I stand?

A survey in Sydney showed 26% of prescription medicines were used for off-label indications.¹ Other studies have shown that in the USA 9.2% of 500 medicines were for off-label use², in one UK specialist palliative care unit 25% of prescriptions affecting 66% of their patients were for off-label use, and in European audits between 39 and 55% of prescriptions were for off-label use.³

It would seem that Craig Patterson's washing line will need many clothes pegs!

The issue of using the Pharmaceutical Benefits Scheme to supply a drug outside the restrictions for authority prescribing is much clearer: it is a breach of the National Health Act. It would however be salutary for health professionals to know what penalties the Act provides for even when the prescription is written in good faith.

Roger Goucke
Head, Department of Pain Management
Sir Charles Gairdner Hospital
Perth

References

1. Bicknell ME, Weekes LM. Off-label drug use in an outpatient setting – a pilot study. *Aust J Hosp Pharm* 1995;25:527-9.
2. Erickson SH, Bergman JJ, Schneeweiss R, Cherkin DC. The use of drugs for unlabeled indications. *JAMA* 1980;243:1543-6.
3. The use of drugs beyond licence in palliative care and pain management. A position statement prepared on behalf of the Association for Palliative Medicine and the Pain Society. 2002. http://www.painsociety.org/pdf/drugs_doc.pdf [cited 2004 July 1]

Mr C. Patterson, one of the correspondents, comments:

Dr Goucke is right to highlight that off-label prescribing occurs extensively and, in certain populations such as children, this is through necessity. I am uncertain, however, that the potential increase in professional liability is widely recognised. Off-label prescribing would often be defended by the body of published evidence of an effect. My main point is that, in the gabapentin example, the pharmaceutical company was the voice goading this off-label prescribing, and doctors displayed good faith that what they were being told was true and accurate. Should the doctor find themselves in a legal dispute, that same voice would be strangely silent when it comes to supporting off-label use.

Management of acute gout

Editor, – In his excellent article ‘Management of acute gout’ (Aust Prescr 2004;27:10–3) Dr McGill mentioned that ‘the acute attack is also an opportunity to assess and manage associated disorders such as obesity, excessive alcohol consumption, hypertension, hyperlipidaemia and renal insufficiency’. He went on to say that ‘controlling these problems may prove to be of greater long-term benefit to the patient than controlling their hyperuricaemia’, but he does not mention what part a diet low in purines plays, if any, in the long-term management of gout.

Charles Dickens’ Mr Pickwick suffered from gout, which was portrayed as being related to his alcohol intake, and this remains the perception of many of our patients.

John A. Comerford
General practitioner
Newstead, Qld

Dr Neil W. McGill, the author of the article, comments:

Although patients may attribute acute attacks to dietary indiscretions, I am not aware of any study that has shown that a particular dietary event increases the likelihood of a gouty attack. With respect to the influence of diet on the chronic management of gout, hyperuricaemia is clearly associated with alcohol intake and obesity (3.4% of people below the 20th percentile and 11.4% of people above the 80th percentile for body weight are hyperuricaemic).

The effect of purines in the diet is complex and poorly understood. A prospective study of 47 150 men showed an increased risk of gout in association with the intake of meat and seafood, and a reduced risk with low-fat dairy foods. Total protein, animal protein and purine-rich vegetable intake were not associated with the risk of gout.¹ It would therefore appear sensible to recommend correction of obesity, a low alcohol intake, avoidance of high intakes of meat and seafood, and plenty of low-fat dairy products. However, it should be remembered that dietary intervention usually reduces the uric acid by a maximum of 15%, is often difficult to maintain and has never been prospectively shown to reduce the incidence of gout.

For patients with proven recurrent gout, especially those with tophi, erosions, persistent symptoms between attacks and renal impairment, encouraging lifelong compliance with hypouricaemic drug therapy is the most effective means of maintaining a healthy uric acid concentration and preventing disease progression.

Reference

1. Choi HK, Atkinson K, Karlson EW, Willett W, Curhan G. Purine-rich foods, dairy and protein intake, and the risk of gout in men. *N Engl J Med* 2004;350:1093-103.

Nitrofurantoin

Editor, – There has been some adverse publicity regarding the long-term use of nitrofurantoin. Some of my patients who require long-term prophylactic antibiotics, usually for urinary tract infection, are asking to come off this medication.

I find nitrofurantoin is a very useful antibiotic which is readily available (30 tablets with one repeat helps to ensure that patients do actually stay on it!). Nitrofurantoin is rapidly absorbed and rapidly excreted with high urinary concentrations and has good activity against Gram negative bacteria. It has a very low incidence of fungal problems especially vaginal candidiasis and a low incidence of gastrointestinal adverse effects.

It would be useful to know how these benefits can be weighed up against the risk of harm.

Tim Skyring
Urological surgeon
Figtree, NSW

Professor J. Turnidge, Infectious disease physician, comments:

Dr Skyring’s letter highlights the dilemma faced by many practising clinicians: do I change my practice because of increasing reports of adverse reactions when the drug has a number of advantages?

He points out the significant benefits of nitrofurantoin and is rightly concerned that patients have been put off by recent publicity. For nitrofurantoin, the rates of adverse reactions are low, but some of these reactions are troublesome.

The reaction of most recent concern is peripheral neuropathy, although this problem has been known for many years. It is most likely in the elderly and others with reduced renal function. Of equal concern is immune-mediated hepatotoxicity, which most often resolves after cessation, but which can be fulminant. A third problem is pulmonary toxicity that can mimic pulmonary fibrosis.¹

There are other serious reactions to nitrofurantoin, but the question remains as to whether they are more frequent than with other drugs used for prophylaxis against urinary tract infections, such as trimethoprim with or without sulfamethoxazole. Without a clear picture of the comparative toxicities of drugs taken over the longer term, it is not possible to make sensible recommendations about which drugs are favoured. The best way of dealing with the dilemma is to discuss the benefits and harms of **all** options with the patient. Dr Skyring should note that nitrofurantoin is still recommended in the current version of Therapeutic Guidelines: Antibiotic.

Reference

1. Pulmonary toxicity with long term nitrofurantoin. *Aust Adv Drug React Bull* 2004;23:15.