Generic substitution in allergic rhinitis

To the Editor: We would like to clarify the paragraph on generic substitution (paragraph 9) in the guideline on allergic rhinitis published as a supplement to the *SAMJ* of December 2006.[1]

The authors of the guideline state that generic substitution of drugs should not be done, unless trials showing clinical equivalence have been undertaken. From a regulatory perspective, antihistamines and intranasal corticosteroids are not included in the Medicines Control Council (MCC)'s list of non-substitutable medicines[2] and this recommendation would therefore be incorrect.

We know of no randomised controlled clinical trials comparing innovator and therapeutically equivalent generic products in the treatment of allergic rhinitis. The statement in the guideline is therefore not evidence-based or substantiated. In the absence of evidence the Allergy Society of South Africa is unable to make a recommendation either way, and it will therefore be up to health care professionals, health care funders and patients themselves to decide which product is best suited to the individual. We believe that generic products that are therapeutically equivalent have enabled greater availability of more cost-effective therapy to patients with allergic rhinitis.

We would also like to point out that reference 191 should read 'The Medicines and Related Substances Act, Act 101 of 1965, as amended, Section 22F'.

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Plea for privacy

To the Editor: It is surprising that the South African Medical Association (SAMA) has 'called on the medical profession, media, political parties and the public to respect the privacy of the Minister of Health, and to stop speculating about her health and the cause of her medical condition' (*Medigram* vol 15, No. 4, 16 March 2007).

Compassion is, of course, due to all who have serious illness, and the seriousness of the minister's illness makes this particularly the case. However, requests for privacy from enquiry into the cause and sequelae of her illness raise a different aspect.

Dr Manto Tshabalala-Msimang elected to go into public office and *ipso facto* expose herself to public scrutiny. Further, as a Minister of Government she became a public servant, and the public has a right to know whether their employee is capable of fulfilling the demands of office.

Unfortunately the Minister has shown herself to be less than forthright regarding her illness, and the impression is that she sought to cover up its cause and effects. In these circumstances her employers (the electorate) have valid cause to speculate about her illness. Speculation will be inevitable and legitimate until she clarifies her incapacity.

If Dr Manto Tshabalala-Msimang had wished to preserve her privacy, the direction open to her was to resign early in her illness, and allow an unimpaired replacement to take office. This would have allowed her to control her privacy, as well as enhancing the esteem in which one assumes she would want to be held.

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Collapsing focal segmental glomerulosclerosis as a possible complication of valproic acid

To the Editor: Long-term use of valproic acid (VPA) leads to multiple organ damage, including tubulointerstitial nephritis.[1] Collapsing focal segmental glomerulosclerosis (CFSGS) associated with VPA has never been reported. Here we report on a case in which such an association appears highly probable.

In October 1995, a 35-year-old man was admitted to the nephrology unit (Lapeyronie Hospital, Montpellier, France) for oedema of the lower limbs. His history was of epilepsy since 1989, treated with VPA, and chronic renal failure (glomerular filtration rate (GFR) 34.14 ml/min in April 1995). On admission, clinical examination showed blood pressure 160/110 mmHg, weight 68.4 kg and oedema of the lower limbs. Acute abdominal pain occurred during hospitalisation. Laboratory examination showed the following findings: GFR 20.22 ml/min, albumin 22 g/l, and proteinuria 4.85 g/d. Other tests were normal, including VPA blood level and serology. Kidney biopsy (light microscopy) showed collapse of the glomeruli with severe vacuolisation of the podocytes (Fig. 1). Immunofluorescence study showed no immune deposits. Abdominal computed tomography (CT) scan revealed pancreatitis. As VPA toxicity was suspected, carbamazepine was substituted for VPA. Haemodialysis was started in April 1996.

CFSGS related to VPA toxicity has never been reported. CFSGS has been related to HIV.[2] In the present case chronic renal lesions might have evolved to CFSGS, but such a possibility has never been reported before.[3] The causal role of VPA appears

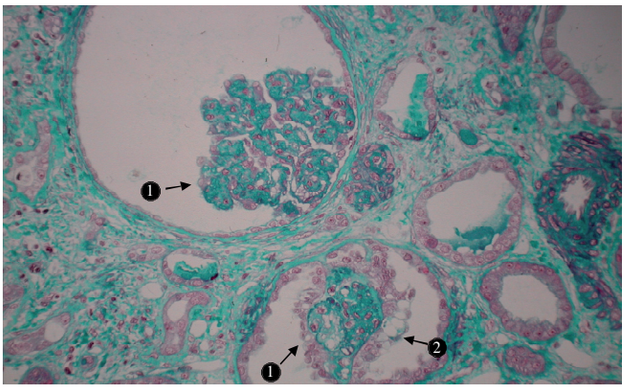

Fig. 1. Renal biopsy Masson trichrome x 400: collapsing focal and segmental glomerulosclerosis. Note the collapse of the glomerular tuft (1) and the intense vacuolisation of the podocytes (2).

to us likely. Indeed vacuoles in tubules, known to be related to VPA toxicity,[1] were also found in the glomeruli. These vacuoles might have originated the collapse of the glomeruli. Furthermore VPA toxicity is related to time exposure but not to blood level and occurs through lipid peroxidation,[4] and our patient had been using VPA for several years. Chronic renal failure could have also magnified VPA toxicity through production of peroxides. Acute pancreatitis occurring at the time of oedema suggests renal damage by VPA as pancreatitis can occur after VPA.[5] In conclusion, long-term use of VPA could lead to CFSGS. Assessment of renal function in this setting must be mandatory. When renal dysfunction is present, preferring another antiseizure drug is highly advisable.

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Mosvold Hospital ARV programme

To the Editor: Mosvold Hospital is a 250-bed government district hospital in northern KwaZulu-Natal. It has a catchment area of 110 000. The HIV-positivity rate for the area, as determined by prevention of mother-to-child transmission (PMTCT) statistics, is 28%.

The antiretroviral (ARV) programme at Mosvold Hospital began on 16 September 2004. By 7 December the first 100 patients had commenced treatment and by 30 October 2006, 1 400 had been started on ARVs.

The programme has gone through many changes. It started as a hospital-based programme, but after 3 months it was changed to a decentralised, clinic-based programme. A new ARV prescription card was developed allowing easy follow-up of patients, results and side-effects. Initially we only allowed a certain number of patients to start each month, but this was changed to a 'no waiting list' policy after a few months. A HAST committee was developed and continues to meet on a monthly basis, a new database was developed, and an ARV policy manual was developed and distributed to clinics.

Of the first 100 patients started on ARVs, 89 are still on the programme and in the district. Of those who left the programme, 8 died, 1 defaulted medication, and 2 transferred out of the district to return to work.

Of the 8 patients who died, 5 were identified as at high risk at the time of commencing medication according to one or more of the following criteria: (*i*) CD4 count < 50 cells/ul; (*ii*) World Health Organization stage 4 disease; (*iii*) new opportunistic infection in the month before starting ARVs; (*iv*) haemoglobin level < 8 g/dl; and (*v*) on intensive-phase tuberculosis treatment.

Of the 3 lower-risk patients, 1 died as a result of suicide, 1 as a result of clinically diagnosed lactic acidosis (at that time we were unable to measure lactate levels), and 1 as a result of immune reconstitution syndrome.

Of the remaining 89 patients, 76/89 (85%) have viral loads < 400 on their 18 - 24-month blood work; 5/89 (5.6%) have viral loads 400 - 5 000 (3 of these are children who are thriving); 5/89 (5.6%) have levels that would allow them to be considered for regimen 2; (one has changed and is doing well; the others have