Patients participating in the study were provided with treatment packs of acyclovir 800 mg tablets at a reduced price. Compliance was excellent, with 84% of the patients maintaining the 5-times-daily regimen and only 14% forgetting a tablet or two. About two-thirds of the patients had commenced therapy within the recommended 72 hours of onset of rash (56% within 48 hours). However, there were unnecessary delays, mainly due to difficulties in diagnosis in the early prodromal period and a lack of awareness by patients of the need for early treatment. The majority of patients (57%) advocated greater public awareness of shingles so that early signs and symptoms could be recognised. The majority of patients (61%) had also never previously heard of shingles and fewer than half (46%) of those who had were unaware of the correct cause or the need for early therapy. The source of all health care information for the great majority of patients (76%) was their doctors.

With the advent of prodrugs with greatly enhanced oral bio-availability, such as valaciclovir and famciclovir, the early treatment of herpes zoster has been greatly facilitated and is considerably cheaper. In a recent clinical evaluation valaciclovir was demonstrated to reduce symptomatic markers of herpes zoster disease significantly, compared with acyclovir.[3] Greater doctor and patient awareness should significantly enhance the management of this distressing illness.

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Post-menopausal panic disorder

To the Editor: Two letters to the *SAMJ* recently discussed the differential diagnosis and management of hot flushes, night sweats and palpitations. Maartens *et al.*[1] asserted that the most common non-hormonal benign causes are 'stress and medications'. Davey[2] agreed that such symptoms certainly need to be taken seriously, and commented that anxiety states may precipitate hot flushes.

We would like to focus the attention of clinicians on the importance of panic disorder as a possible diagnosis in patients presenting with episodes of intense fear or discomfort, with hot flushes or chills, palpitations or accelerated heartbeat, sweating, sensations of shortness of breath, and other symptoms of apparent autonomic discharge.[3] Nocturnal panic attacks are frequently seen in this disorder.[4]

Panic disorder is one of the most common psychiatric disorders.[5] Although it is still mistakenly viewed by some as benign or 'in the mind', panic disorder is now known to be associated with significant morbidity and even mortality, and appears to be mediated by a specific neurobiological circuitry.[6] In view of the excellent pharmacological and psychotherapeutic treatments currently available for panic disorder,[7] it is essential that this diagnosis not be missed.

From a theoretical viewpoint, it is interesting to speculate about the specific neurobiology of post-menopausal panic disorder. It has been convincingly argued that decrease of panic attacks during pregnancy and increase of panic attacks premenstrually reflect alterations in PCO_2 at these times.[8] Certainly, neurotransmitters involved in panic disorder can be modulated by hormonal factors.[9] From a clinical perspective, there is very limited anecdotal evidence of response of panic disorder to hormonal treatment,[10] while standard anti-panic treatments have convincingly been shown to be effective.

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An unusual cause of rhabdomyolysis

To the Editor: In our centre rhabdomyolysis is usually caused by crush injury sustained in mining accidents. We present a case of rhabdomyolysis caused by 8 hours of motionless squatting in a minibus taxi.

A 42-year-old man was admitted to Ernest Oppenheimer Hospital complaining of severe generalised joint and muscle pain. He emphatically denied toxin ingestion. Examination revealed signs of dehydration, peripheral vasoconstriction and severe muscle tenderness.

Laboratory findings were as follows: potassium 8.4 mmol/l, urea 17.9 mmol/l, creatinine 263 mmol/l, corrected calcium 1.24 mmol/l, aspartate aminotransferase (AST) 7 040 U/l, alanine aminotransferase 700 U/l, uric acid 0.573 mmol/l, leucocytes 26.1 x 10^9/l. Arterial blood gas measurement showed compensated metabolic acidosis with respiratory compensation.

A diagnosis of acute renal failure and myopathy of unknown causation was made. Haemodialysis was instituted and cefotaxime commenced. The next day the patient was found to have developed a compartment syndrome of both legs, necessitating bilateral fasciotomies. Laboratory investigations at this stage revealed positive urine myoglobin, creatine phosphokinase 424 320 U/l, lactate dehydrogenase 22 220 U/l, creatine kinase muscle-brain 7 200 U/l, AST 2 590 U/l.