is an urgent need for first-aid measures and to impart relevant skills to the parents but the pathways to these are not described clearly. Could we have role-play models, documentaries, pamphlet distribution, and healthcare information in the media to highlight the issue? There is also an urgent need to discard the old traditional, social and religious views. The problem must be tackled effectively and lessons may be learnt from developed countries. Overall, this is an interesting topic, which generates much debate. The authors and the Editor should try to highlight such articles and I would sincerely applaud their sincere efforts.

SRIJIT Das
Department of Anatomy, Universiti Kebangsaan Malaysia, Jalan Raja Muda Abdul Aziz, 50300 Kuala Lumpur, Malaysia

Correspondence:
Dr. Srijit Das, Jalan Raja Muda Abdul Aziz, 50300 Kuala Lumpur, Malaysia.
E-mail: das_srijit23@rediffmail.com

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BENIGN PROSTATIC HYPERPLASIA: IS IT A GROWING PUBLIC HEALTH CONCERN FOR INDIA?

Sir,

BPH is the most common urological problem of ageing men, manifested as severe obstruction in urinary flow with discomfort and pain. BPH is a complex disease from the etiological and pathogenesis point of view.[1] A recent AUA guideline (2003) suggests an increase in the incidence of BPH worldwide and predicts by the age of 60 years, more than 50% of men will have microscopic evidence of the disease and by the age of 85 years, as many as 90% of men will be affected.[2] Worldwide investigations for incidence of BPH are scanty and at times difficult to compare due to uneven definition of BPH based on different clinical parameters. There is also great geographical disparity in prevalence and degree of severity of symptoms of BPH. Benign enlargement of the prostate gland is reported to be most common in blacks, Caucasians, and Jews, but less frequent in males from the Far East.

Asian, particularly vegetarian, men consume low-fat, high-fiber diets rich in weak dietary phytoestrogens, which have been proposed as chemopreventive agents. But the very few studies conducted on BPH patients from India suggest BPH as the most common pathological condition with an incidence of 92.97% (n = 185) and (93.3% (n = 200).[4] Though not well performed epidemiological investigations, these data along with clinicians’ practical evidence indicate substantial need for a survey of the incidence of BPH in India. Interestingly, the

BIPOLAR AFFECTIVE DISORDER IN PARKINSON’S DISEASE: CLINICAL DILEMMAS

Sir,

Psychiatric disorders are a common co-occurrence in people with Parkinson’s disease (PD).[1] Psychiatric symptoms may be the direct result of PD, its co-morbid pathologies, or occur as a side effect of its pharmacotherapy. About 10% of patients on treatment for PD will experience euphoria and 1% will develop mania.[2,3] We report a case of bipolar affective illness complicated by idiopathic PD and its treatment with special reference to its response to Clozapine.

The patient is a 56-year-old married lady with premorbid paranoid personality traits without family history of any neuropsychiatric illness. Her index visit was in August 2005 when she was diagnosed to have severe depression with suicidal intent, and a detailed neurological evaluation at that time did not reveal any symptoms of PD. She was treated with a course of seven bilateral brief pulse modified electro convulsive therapy and Sertraline 100 mg, and became euthymic within one month. One year later
she developed unilateral tremors for which neurology consultation was sought. She was diagnosed with left hemi Parkinsonism and started on Ropinarole 0.125 mg thrice daily and Trihexyphenidyl 1 mg thrice daily. Following poor response of PD symptoms, in Sep 2006 the neurologist started her on Selegiline 5 mg in the morning. Subsequently the patient was reported to be irritable, dysorphic and over-religious; these symptoms remitted on stopping Selegiline but there was inevitable worsening of motor symptoms. On restarting Selegiline she developed a full manic syndrome characterized by over-activity, over-talkativeness, irritability, hyper-religiosity and decreased sleep with grandiose delusions. Selegiline was stopped considering the possible interaction with Sertraline and as a doubtful precipitating factor for mania but other drugs were continued according to the neurologist’s advice. Treatment options for mania were discussed with the patient and caregivers, who were willing to opt for Clozapine only after a conventional trial in view of serious side effects. The patient was started on Sodium Valproate, which was increased to 1200 mg daily (serum Valproate level of 55 microgram per ml) and Olanzapine 20 mg daily. With this treatment her manic symptoms improved but she did not become euthymic. After one month there was a worsening of manic symptoms for which the patient had a history of depression prior to the onset of PD and the mania responded well to a low dose of Clozapine without worsening of motor symptoms. No guidelines or strategies are available for the management of mania or bipolar illness in PD. However, guidelines are available for the prevention and management of psychosis in PD. Further, the possibility of Selegiline precipitating mania in a patient who has vulnerability for a bipolar illness should not be discounted.

The authors would like to stress the need for good liaison between the various specialities and for a specialist who can manage both mood and movement problems together.

**NAVEEN THOMAS, PRASANNA JEBARAJ, KISHOR KUMAR S.**
Department of Psychiatry, Christian Medical College, Vellore-632 002, Tamil Nadu, India

**Correspondence:** Dr. Naveen Thomas, Department of Psychiatry, Christian Medical College, Vellore-632 002, Tamil Nadu, India. E-mail: naveenlinda2002@yahoo.co.in

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