LETTER TO THE EDITOR

Compulsive urination as a presenting symptom of frontotemporal dementia

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Sir,

Frontotemporal dementia (FTD), one variant of frontotemporal lobar degeneration, is characterized by personality changes, behavioral dyscontrol and executive dysfunction [1]. Compulsive and repetitive behavior, such as repetitive checking, hyperorality, and pacing, are well documented presenting signs and symptoms of FTD [2]. Urinary symptoms including urgency, frequency, and incontinence also occur, but are typically considered a late feature. We describe two male patients whose initial complaints were excessive, frequent urination who went on to a diagnosis of FTD.

Patient 1 is a 36-year-old right-handed man seeking evaluation for personality changes, urinary frequency, and mild episodic memory loss over the previous 1.5 years. He began urinating almost constantly as often as 5 min apart with voided volumes of 5–50 cc of urine. The patient has never complained of pain nor did he have any symptoms of urinary tract infection or incontinence. His past medical history is otherwise unremarkable. Over the following months the patient developed other compulsions including showering up to 16 times daily, checking the locks repeatedly, and eating every 5 min.

Patient 2 is a 34-year-old right-handed man presented for evaluation of change in behavior, urinary frequency, and complaint of memory loss over the past 2 years. He gradually became more withdrawn from family members and apathetic. At home, he would urinate every 10 min for several hours at a time. He then developed several other compulsions, including repeatedly checking doors of his car and his home to the point of interfering with his ability to travel and breaking the locks.

There was no family history of neurodegenerative disease in either patient. On neurologic examination, both patients demonstrated 'frontal' mannerisms, with dysinhibition in patient 1 and apathy in patient 2. Both patients demonstrated moderate cognitive dysfunction on bedside testing and had frontal release signs. The remaining neurologic exam including visuospatial, motor, gait, and station testing was normal.

Patient 1 underwent neuropsychologic testing and his full-scale I.Q. was 108, verbal I.Q. 110, and performance I.Q. 103. Abnormalities were demonstrated in test of motor and speed operations. Arithmetic abilities were scored at the 75th percentile, whilst reading recognition was scored at the 91st percentile and comprehension at the 50th percentile. Patient 2's neuropsychologic testing revealed a full-scale I.Q. of 81, verbal I.Q. of 85, performance I.Q. of 80, vocabulary at the 9th percentile, with arithmetic ability at 63rd percentile. Following this, a diagnosis dementia was made, although apparently the evaluation was felt to be inadequate to determine its cause.

Both patients underwent extensive workup with normal renal, hepatic, and thyroid function. Urinalysis and urologic evaluation was unrevealing. Screening for a mutation in the microtubule association protein tau gene was performed in patient 1 and was negative. Brain MRI in patient 1 revealed quite profound frontal and temporal lobe atrophy and there was frontotemporal hypometabolism on FDG PET scan in keeping with a clinical suspicion of FTD. In patient 2, MRI demonstrated mild frontal and temporal lobe atrophy and FDG PET scan revealed hypometabolism most prominent in anterior frontal lobe and anterior cingulate gyrus. The FDG-PET scans from both patients were analyzed by three dimensional stereotactic surface projection and were compared to a normal control group (Fig. 1). The region most severely affected overlapped in both patients and was localized to the superior medial frontal cortex extending into the anterior cingulated gyrus [3].

Based on revised Neary criteria [4], both patients were ultimately diagnosed with FTLD. Both patients demonstrated insidious personality and behavioral changes. Both patients developed

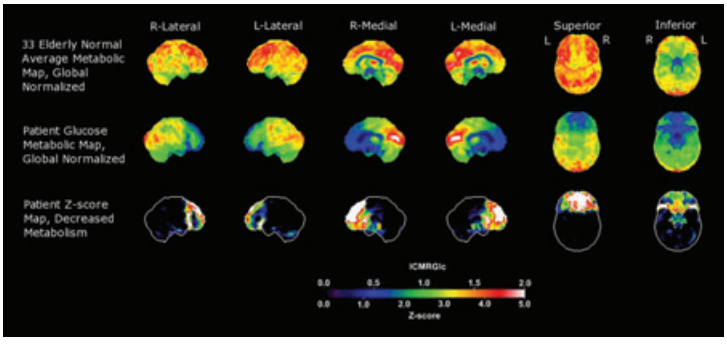

Figure 1 Statistical stereotactic surface projection maps showing pixels significantly hypometabolic compared with control in patient 1 (first row) and patient 2 (second row). *Z*-score values are color-coded as indicated in the color scale. Both patients have hypometabolism affecting the frontal lobes, particularly medial frontal and anterior cingulate cortex. Lateral frontal regions are more affected in patient 1 and anterior temporal cortex is more affected in patient 2.

perseverative behaviors, the most impressive being compulsive urinations which were treated with quetiapine. Resolution of urinary compulsions occurred with a total dosage of 100 mg daily in Patient 1 and 150 mg daily in Patient 2.

The two cases presented highlight urinary symptoms as a prominent feature of early FTD. We suspect that urinary symptoms may be under-recognized as a presenting feature of FTD, and offer an opportunity for early diagnosis if appropriately addressed, as well as avoiding cost and morbidity of unnecessary urologic testing.

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