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Case Report

Surreptitious Baclofen Causing Delirium in Chronic Kidney Disease

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Abstract

Case report of a patient taking Baclofen regularly. When he developed kidney failure, Baclofen toxicity, as manifested by a gradual onset of delirium, developed. Only after dialysis was started, and improvement of his delirium occurred following dialysis led to a search of possible dialyzable medicines and Baclofin was found in his home medicines he regularly took. Baclofen neurotoxicity in dialysis patients is regularly recognized, but this seems to be the first report of a patient chronically on Baclofen developing neurotoxicity when renal failure developed.

Introduction

Baclofin is an oral derivative of gamma-aminobutyric acid used to treat muscular spasticity from disorders of the central nervous system. Although the precise mechanism of action of Baclofen is not fully known, it is capable of inhibiting polysynaptic and monosynaptic reflexes at the spinal level by hyperpolarization of afferent terminals [1]. Baclofen is rapidly and almost completely absorbed. In healthy subjects, approximately 80% is excreted by the kidneys and about 15% is metabolized by the liver to an inactive form [1]. Recently, there were a few articles, in 'Kidney International' [2,3] and 'Seminars in Dialysis' [4], highlighting the mental status alterations in patients with Chronic Kidney Disease (CKD) receiving Baclofen. These articles report the results of Baclofen administration to patients with CKD. None dealt with patients taking Baclofen before they develop kidney disease.

Case report

We recently witnessed dramatic mental status changes in an individual who had been taking Baclofen for years before CKD, and who developed Baclofen neurotoxicity at the age of 81 when CKD developed. The patient had been taking Baclofen, 10 mg/day, for years for multiple sclerosis. His mild kidney impairment progressed and when his serum creatinine was between 3.5 to 4 mg/dl confusion and gradual delirium occurred. Because of the presumptive diagnosis of uremic encephalopathy, hemodialysis was initiated. With dialysis, the confusion cleared. Dialysis was stopped since the kidney function stabilized, yet confusion returned. Dialysis was restarted and for some months he was stable with variable levels of sensorium. Then, one weekend he missed a dialysis treatment and on Saturday afternoon became unarousable. A search of his medicine cabinet revealed Baclofen which neither the hospital nor the dialysis unit had prescribed or tabulated on his medication list. The Baclofen was stopped and following the next dialysis treatment, his sensorium was dramatically improved. Since his sensorium returned to near normal levels, even when he missed one or two dialysis treatments, it seemed clear in retrospect that his decreased sensorium was a consequence of Baclofen toxicity (accumulation) and not his kidney failure per se.

Discussion and conclusion

In retrospect, since Baclofen is readily dialiazable his improvement following dialysis was due to the removal of this drug rather than treatment of uremic encephalitis. This experience emphasizes the need to aggressively determine all

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medications a patient is taking. In addition, it highlights the possibility for a drug to be well tolerated in an individual with relatively normal kidney function, but as CKD supervenes, the same drug, like Baclofen, can cause serious neurotoxicity.

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