Baclofen toxicity as a reversible cause of decreased consciousness in haemodialysis patients

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Introduction
Baclofen is a γ-aminobutyric acid (GABA) agonist frequently used as a muscle relaxant in the management of spasticity. Renal excretion is the main route of elimination as 70-80% of the drug is excreted unchanged in the urine. In patients with CKD, Baclofen dose should be reduced even with mild CKD. Starting baclofen at a low dose and careful monitoring for toxicity are two crucial points if it is used in haemodialysis patients. In this case report, we describe a haemodialysis patient presented with altered consciousness due to baclofen toxicity.

Method
A 75-year-old lady was admitted from the haemodialysis unit with increased drowsiness and kinetic muscle twitches. She had no apparent motor deficit or focal neurological abnormality. She had had a cerebrovascular stroke a few years ago. CT showed only the old infarct and no acute abnormalities. She was treated with vancomycin and gentamicin for a suspected line infection. However, inflammatory markers were never raised and there was no significant growth from repeated cultures. Despite antibiotics, she continued to be drowsy and hypotensive with intermittent pyrexia.

On day three of her admission, it was noted that her Baclofen (Oral 10mg TDS) had not been ordered from pharmacy. She had become more responsive and even conversational. Once her Baclofen was restarted, her consciousness level deteriorated. The drug was stopped as the suspected cause of the impaired consciousness.

On day four, she was unable to tolerate haemodialysis due to ongoing hypotension. Withdrawal of dialysis and a focus on palliative care was considered. However, it was noted that there had not been an opportunity for the Baclofen to be cleared. After a 4 hours dialysis session, she became more alert. With further dialysis sessions, she continued to become more responsive and the twitching resolved. Once back to her cognitive baseline she was discharged.

Discussion
Altered consciousness in haemodialysis patients encompasses a vast list of differential diagnoses. A systematic approach and review of medications can provide important diagnostic pointers. In this case, Baclofen accumulation was the cause. Adverse reactions as a result of Baclofen include hypotension, CNS depression and less frequently hypertonia.

It is excreted mainly by the kidney. Four hours haemodialysis session removes 79% of the drug. However, patients on dialysis are liable to baclofen toxicity. If Baclofen is required it should be used with a dose reduction and increased time intervals between doses. Alternative drugs should be considered, increasingly Tizanidine and Dantrolene.

It is possible to measure Baclofen levels and there would be merit to checking levels in haemodialysis patients when there is diagnostic uncertainty surrounding altered consciousness.

In cases of toxicity, haemodialysis can be used to aid removal. Multiple sessions may be required to sustain the improvement in consciousness.

Conclusion
Although Baclofen is readily removed by dialysis, dose accumulations can occur. Therefore, dose adjustments should be considered in haemodialysis patients and CKD patients. If Baclofen toxicity is suspected, haemodialysis should be used to aid clearance.