Abstract:

Clonidine, a centrally acting alpha-2 adrenergic agonist, is commonly used in the management of hypertension and withdrawal symptoms. However, its use in patients with renal impairment, requires careful consideration due to the potential for drug accumulation and adverse neurological effects. We present a case of clonidine-induced encephalopathy in a patient undergoing peritoneal dialysis, highlighting the challenges in managing hypertension in this population and the importance of cautious medication management.

Case Presentation:

A 55-year-old female with end-stage renal disease , undergoing peritoneal dialysis for the past few years, presented with a history of seizure. On admission, she was unconscious and required mechanical ventilation. Initial laboratory investigations showed raised serum creatinine and bun. CT brain showed age related atrophic changes. Hemodialysis was done for the patient after discussion with nephrologist. Patient showed signs of neurological improvement after HD but did not regain full consciousness. She was started on multiple antihypertensives but her blood pressure was uncontrolled. As such clonidine 0.1 mg tds was started after two days of admission.

On 4th day of admission patient's consciousness was found to be further decreased. At this stage her blood pressure was under control. Further evaluation ruled out other causes of encephalopathy such as metabolic disturbances or structural brain lesions. MRI brain revealed no significant abnormality. EEG was done but showed no epileptic activity. Given the temporal association with clonidine initiation and absence of other identifiable causes, clonidine-induced encephalopathy was suspected.

Management and Outcome:

Clonidine was promptly discontinued upon suspicion of drug-induced encephalopathy. Supportive measures were initiated. Over the next two days, the patient gradually regained consciousness, with improvement in neurological status noted on serial examinations. She was extubated from mechanical ventilator on the 7th day from admission.

Discussion:

Clonidine is known to cross the blood-brain barrier and exert its effects on central alpha-2 adrenergic receptors, leading to central nervous system depression. It is predominantly cleared by renal excretion, and its pharmacokinetics can be significantly altered in patients

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with renal impairment, leading to drug accumulation and toxicity. The central nervous system manifestations of clonidine toxicity include sedation, confusion, and rarely, encephalopathy. Patients with impaired renal function, are particularly vulnerable to these effects.

In this case, the patient's pre-existing renal impairment likely contributed to clonidine accumulation, precipitating encephalopathy. The prompt recognition of drug-induced toxicity and discontinuation of clonidine were crucial in achieving clinical improvement.

Conclusion:

Clonidine-induced encephalopathy should be considered in patients with chronic kidney disease, who present with unexplained neurological symptoms. Clinicians should exercise caution when prescribing clonidine in this population, considering dose adjustments and close monitoring for adverse effects. Timely recognition and management are essential to mitigate potential complications associated with drug-induced encephalopathy. This case underscores the importance of individualized medication management and heightened vigilance for adverse drug reactions in patients with chronic kidney disease.

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