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Uremic Myoclonus presenting as Acute Meningoencephalopathy with Refractory Generalized Myoclonus : A Case Report

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Abstract

Uremic encephalopathy is a well recognized complication of advanced renal dysfunction;however,generalized refractory myoclonus as the predominant presentation is uncommon and mimic infectious or autoimmune encephalitis.Uremic myoclonus is a movement disorder characterized by sudden,brief,involuntary muscle jerks seen in uremia.Here,we report a case of uremic myoclonus in a patient.

Keywords: Uremic encephalopathy,Uremic myoclonus,Acute kidney Injury,Hemodialysis,Generalized myoclonus

Introduction

Uremic encephalopathy is a reversible neurological syndrome occuring in patients with severe renal dysfunction due to accumulation of neurotoxic metabolites.Clinical manifestations range from mild cognitive impairment to coma and movement disorders.Myoclonus is a recognized but relatively

Uncommon manifestation and may resemble epileptic seizures or autoimmune encephalitis, leading to diagnostic uncertainty.Prompt recognition and initiation of renal replacement therapy are essential because neurological deficits are potentially reversible.

We report an elderly patient with progressive acute kidney injury who presented with severe stimulus sensitive generalized myoclonus and encephalopathy, initially mimicking meningoencephalitis and who demonstrated dramatic neurological recovery following hemodialysis.

Case Report

A 73 year old male patient with a history of type 2 diabetes mellitus and hypertension , on tab telmisartan and metformin ,presented with complaints of four day history of fever,altered sensorium and 2-3 episodes of vomiting.Patient also had urinary incontinence .On admission,his vitals were stable and GCS was E4V3M6.He was irritable and exhibited generalized stimulus sensitive abnormal jerky movements triggered by tactile stimulation.CNS examination revealed neck stiffness and normal power and tone in all limbs.

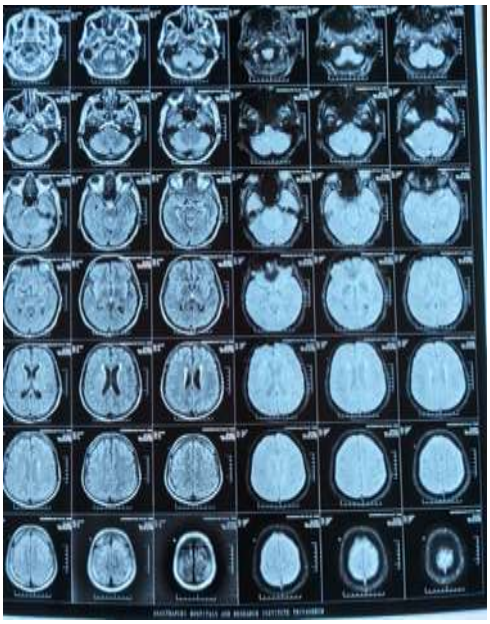
Initial laboratory investigations revealed severe anemia,thrombocytopenia,normal WBC counts,deranged renal function tests and mild elevation of inflammatory markers,normal LFT and mild prolongation of PT,INR and APTT.Urine output was preserved initially.Arterial blood gas analysis revealed metabolic acidosis with negative serum

ketones.EEG was done and was not showing any epileptiform discharges.2 D Echo was done and was normal.Glycemic control was done with injection insulin.Antibiotics Injection meropenem, Doxycycline and Azithromycin),Neuro and Renal protective measures were started.

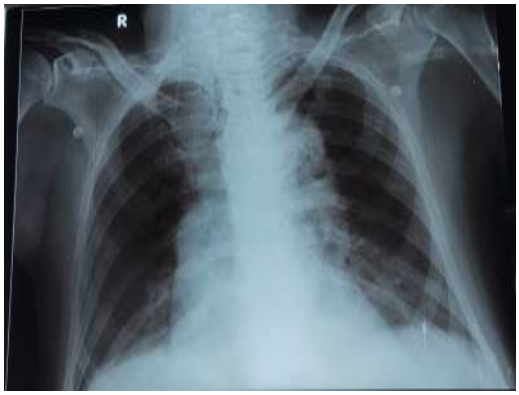
Repeat investigations revealed worsening of azotemia despite improvement of serum creatinine and further decline in platelet counts on second day. GCS was same. Urine output was preserved.Serum ammonia levels,stool occult blood and tumor markers were negative.CT chest revealed bilateral mild pleural effusion with passive atelectasis of underlying lung segments and mild pericardial effusion.CT Abdomen and pelvis revealed mild ascites,bilateral renal parenchymal changes and colonic diverticulosis.

Because of persistent fever,neck stiffness and encephalopathy despite giving antibiotics and neuro and renal protective measures,an extensive diagnostic evaluation was done.LP study was done a n d CSF analysis including routine, microscopy, culture and meningitis profile,biofire PCR panel was done and was all negative.

MRI brain showing chronic microbleeds in right anterior temporal subcortical w h i t e m a t t e r a n d l e f t o c c i p i t a l periventricular white matter in this patient



CXR showing ventilator associated pneumonia in this patient



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Autoimmune encephalitis panel,paraneoplastic antibody profile,tropical fever panel ,Weil felix test and microbiological cultures of blood,urine and sputum were all negative.CT brain was taken and was normal.

The patient's neurological status progressively deteriorated with worsening consciousness and abnormal jerky movements necessitating endotracheal intubation and mechanical ventilation.There was further worsening of azotemia and myoclonus which was not settling with antiepileptics and benzodiazepines.

EEG was done and was having no epileptiform discharges.MRI brain was done and demonstrated only microbleeds in right anterior temporal subcortical white matter and left occipital peri ventricular white matter.There was no evidence of acute infarction or encephalitis.

With progressive deterioration of renal function, uremic encephalopathy with uremic myoclonus was suspected and hemodialysis was initiated,The initial dialysis session was complicated by intradialytic hypotension requiring vasopressor support.Subsequently,alternate day hemodialysis was continued.

During intensive care unit stay,inflammatory markers was increased,and the patient developed ventilator associated pneumonia.Endotracheal aspirate culture grew multi-drug resistant Klebsiella pneumoniae.Antibiotics were escalated to injection Ceftazidime-Avibactam and Aztreonam, along with chest physiotherapy and regular nebulizations.

Following serial hemodialysis sessions ,blood urea nitrogen and serum creatinine progressively declined.Simultaneously myoclonus markedly decreased,neurological status improved and patient became progressively more responsive.Inflammatory markers were reduced and patient was successfully weaned from mechanical ventilation and extubated.Renal function continued to improve,and he was subsequently transferred from intensive care unit to ward.

Discussion

Movement disorders are well recognized manifestations of uremic encephalopathy but are less frequently encountered than altered consciousness.Generalized myoclonus occurs because of accumulation of uremic toxins,which disrupts cortical and subcortical neuronal function,producing increased neuronal excitability.

Our patient initially fulfilled several clinical features suggestive of meningoencephalitis,including fever,neck stiffness,altered sensorium and abnormal movements.However,comprehensive investigations excluded infectious,autoimmune,paraneoplastic and metabolic causes.

The generalized stimulus sensitive myoclonus remained refractory to conventional antimyoclonic and antiseizure medications.EEG excluded epileptic myoclonus,suggesting a metabolic etiology.Progressive azotemia with metabolic acidosis and subsequent dramatic improvement following serial hemodialysis

Strongly supported the diagnosis of uremic myoclonus.

MRI findings of chronic cerebral microbleeds were unlikely to explain the severity of neurological manifestations ,emphasizing that metabolic encephalopathy should remain a leading differential diagnosis even in patients with incidental neuroimaging abnormalities.

This case also highlights the importance of recognizing intensive care complications ,particularly ventilator associated pneumonia caused by multidrug resistant organisms,which required escalation of antibiotic therapy without altering the underlying neurological diagnosis.

The close temporal relationship between reduction in blood urea nitrogen levels and improvement in myoclonus further supports the reversible nature of uremic neurological dysfunction. Conclusion

This case demonstrates that uremic myoclonus may present as severe encephalopathy with stimulus sensitive generalized myoclonus,closely resembling infectious or autoimmune encephalitis.Asystematicexclusionofalternative etiologies,absence of epileptiform activity on EEG,and rapid neurological improvement following hemodialysis confirmed the diagnosis.Early initiation of renal replacement therapy remains the cornerstone of management and can lead to complete neurological recovery despite severe initial presentation.

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