



## HYPERAMMONEMIA SECONDARY TO NON UREASE PRODUCING BACTERIAL URINARY TRACT INFECTION- A RARE CASE

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<p><b>Article Info</b> <i>Received 09/12/2015</i> <i>Revised 14/12/2015</i> <i>Accepted 20/12/2015</i></p> <p><b>Key words:</b> Escherichia coli, Hyperammonemia, Urease, Neurogenic bladder, Urinary tract infection.</p>	<p><b>ABSTRACT</b> Hyperammonemia is a known metabolic disturbance commonly seen in liver failure, porto-systemic shunts and gastrointestinal bleeding. Urease producing organisms are known to produce hyperammonemia in patient with neurogenic bladder with normal liver functions. We report a rare case of hyperammonemia secondary to non urease producing bacteria (E-coli) in patient with neurogenic bladder.</p>
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### INTRODUCTION

A urinary tract infection (UTI) also known as cystitis or urinary bladder infection is caused by urinary retention, chronic prostatitis, catheterization of bladder and through blood borne infection. E-coli is the most common cause of community-acquired urinary tract infections (80–85%) [1].

### CASE REPORT

A 61 year old male patient, known case of seizure disorder, hypertension and neurogenic bladder came to our emergency room (ER) with history of fever and altered sensorium since two days. Patient was admitted in medical ICU for further management. On further examination patient was drowsy, confused with heart rate of 112 beats per minute, blood pressure – 130 / 70 mm Hg, respiratory rate of 22 breaths / minute with Spo<sub>2</sub> of 95% on room air. Systemic examination was normal except altered sensorium. Blood investigations revealed hemoglobin 10.2gm/dl, total leucocytes 4500 cells/cumm and platelets 50,000 cells/cumm. Renal and liver function tests were normal. Coagulation profile was normal and Serum

ammonia level was 114 mg/dl. Further investigations like CT Brain and EEG were normal, Blood culture was normal and urine culture showed E-coli. Patient was treated with antibiotics (Piperacilin-Tazobactum), rifaximin, lactulose syrup, antiepileptic drugs and other supportive measures. His sensorium improved gradually over 2 days (with normalization of serum ammonia) and was shifted to the ward on the third day. Patient was later discharged home on fifth day in a hemodynamically stable condition.

### DISCUSSION

Hyperammonemia is a metabolic disturbance characterized by increased levels of ammonia in the blood. Ammonia is produced by the catabolism of proteins, later excreted in urine as urea which is less toxic substance. There are multiple reasons which can cause increased levels of ammonia in the blood such as hepatic failure, shunts (porto-systemic), gastrointestinal bleeding, vesico-rectal fistula, urinary tract infections, leukemia, surgery, parenteral nutrition [2], hyperinsulinemic hypoglycemia, carnitine deficiency, myeloma, leukemia and antiepileptic



drugs such as divalproic sodium [3]. Hyperammonemia can cause encephalopathy and even death.

In patients having urinary tract infection (UTI) associated with urinary retention can have hyperammonemia. Bacterial infections such as proteus, klebsiella, morganella, providencia, and possibly Serratia species which are urea splitting organisms can hydrolyse urea into ammonia in patients having urinary retention, which later absorbed systemically to cause hyperammonemia [4-8]. In our patient the UTI was caused by Escherichia coli (E-coli) which is not a urea splitting bacteria, still patient had Hyperammonemia. Neurogenic bladder not only predisposes patient to urinary tract infection but also increases the absorption of ammonia from the retained urine through the bladder walls. Cordano et al [9] have reported hyperammonemic encephalopathy in patient with neurogenic bladder having urinary tract

infection caused by Escherichia coli and Enterococcus faecalis which are considered as urease negative bacteria.

This case highlights the rare cause of hyperammonemia with normal liver function tests. Intensivists should have high suspicion of hyperammonemia in patient with neurogenic bladder having fever and altered sensorium. Escherichia coli though considered as non urease producing bacteria can still produce hyperammonemia in patients having urinary retention (neurogenic bladder). Early diagnosis and treatment can reduce morbidity and mortality.

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#### DECLARATION OF INTEREST

None declared.

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