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Cerebral conundrum: A deep dive into acute encephalopathy

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Abstract

This case report delves into the intricate clinical presentation, diagnostic challenges, and management strategies of a patient diagnosed with acute encephalopathy of metabolic origin, accompanied by severe dehydration, acute kidney injury (AKI) secondary to rhabdomyolysis, old coronary artery disease (CAD) with inferior wall myocardial infarction (IWMI), type 2 diabetes mellitus (T2DM), hyponatremia, hypokalaemia, and hyperchloremia. Through a thorough examination of the patient's medical history, diagnostic findings, and treatment interventions, this report aims to shed light on the complexities involved in managing such multifaceted cases.

Keywords: Hypokalaemia, Hyperchloremia, Diagnostic Findings.

Introduction

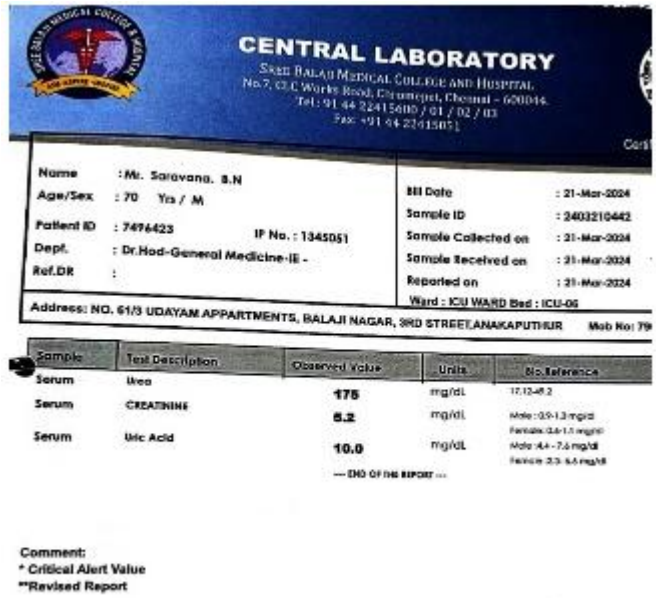
Acute encephalopathy, a condition characterized by rapid alterations in mental status ranging from mild confusion to coma, represents a formidable challenge in clinical practice. While its etiology can be diverse, metabolic disturbances emerge as prominent contributors, weaving a complex tapestry of symptoms and diagnostic hurdles. This introduction serves as a gateway to unraveling the intricacies of acute encephalopathy with a focus on its multifactorial origins, diagnostic conundrums, and therapeutic interventions. The clinical landscape of acute encephalopathy is fraught with ambiguity, as patients may present with a myriad of symptoms reflecting underlying metabolic derangements. These disturbances can stem from electrolyte imbalances, renal dysfunction, metabolic acidosis, or a combination thereof, adding layers of complexity to the diagnostic process. Moreover, the overlap in clinical manifestations between encephalopathy and other conditions such as sepsis, cerebral ischemia, or drug toxicity poses a diagnostic dilemma, necessitating a meticulous evaluation to discern the primary precipitating factors. In this context, the case presentation serves as a focal point, illustrating the intricate interplay between metabolic abnormalities and neurological manifestations. We encounter a 65-year-old male presenting with altered mental status, profound

lethargy, and a history of type 2 diabetes mellitus (T2DM), hypertension, and old coronary artery disease (CAD) with a prior inferior wall myocardial infarction (IWMI). The clinical examination reveals electrolyte imbalances—hyponatremia, hypokalemia, and hyperchloremia—alongside acute kidney injury (AKI) and metabolic acidosis. Such a constellation of findings underscores the multifactorial nature of acute encephalopathy and underscores the importance of a comprehensive diagnostic approach to disentangle its complexities. The diagnostic challenges inherent in managing acute encephalopathy are manifold. The simultaneous presence of multiple comorbidities and electrolyte imbalances complicates the identification of the underlying precipitating factors. Electrolyte disturbances, renal impairment, and metabolic acidosis can each contribute to the development of encephalopathy, necessitating a systematic evaluation to elucidate the primary etiology. Furthermore, the clinical overlap with conditions such as sepsis or cerebral ischemia underscores the importance of thorough clinical assessment and diagnostic testing to differentiate among potential causes. Effective management of acute encephalopathy hinges upon a multidisciplinary approach tailored to address the underlying metabolic abnormalities and precipitating factors. Immediate interventions, such as fluid resuscitation and electrolyte repletion, are paramount to correct dehydration, restore hemodynamic stability, and address electrolyte imbalances cautiously to mitigate the risk of complications such as osmotic demyelination syndrome or cardiac arrhythmias. Moreover, supportive measures aimed at optimizing renal function and acid-base balance play a pivotal role in mitigating further neurological deterioration. The outcome and follow-up of patients with acute encephalopathy underscore the importance of timely recognition and intervention. Serial monitoring of clinical status and laboratory parameters facilitates the assessment of response to treatment and guides further management decisions. In our case, aggressive fluid and electrolyte management, coupled with hemodialysis for AKI secondary to rhabdomyolysis, led to a gradual improvement in clinical status and resolution of metabolic derangements. Such outcomes underscore the significance of a vigilant monitoring strategy and the potential for favourable outcomes with prompt and targeted interventions.

Case Presentation

A 65-year-old male presented to the emergency department with altered mental status and profound lethargy. The patient's medical history included T2DM, hypertension, and old CAD with a history of IWMI five years ago. On examination, the patient was drowsy with a Glasgow Coma Scale (GCS) score of 10 (E3V3M4). Vital signs revealed hypotension (blood pressure 90/60 mmHg) and tachycardia (heart rate 110 bpm). Laboratory investigations showed profound electrolyte disturbances: hyponatremia (Na 125 mmol/L), hypokalemia (K 2.8 mmol/L), and hyperchloremia (Cl 110 mmol/L). Additionally, the patient exhibited acute kidney injury (serum creatinine 2.5 mg/dL) and metabolic acidosis (pH 7.25, bicarbonate 16 mmol/L). A CT scan of the brain ruled out acute intracranial pathology, confirming a diagnosis of metabolic encephalopathy. The simultaneous presence of multiple comorbidities and electrolyte imbalances in our patient posed diagnostic dilemmas. Acute encephalopathy can result from various metabolic derangements, including electrolyte disturbances, renal dysfunction, and metabolic acidosis, making it crucial to identify the underlying precipitating factors. Moreover, the overlap in symptoms between encephalopathy and other conditions such as sepsis,

cerebral ischemia, or drug toxicity necessitated a comprehensive diagnostic approach to differentiate the primary cause. The management of our patient involved a multidisciplinary approach targeting the underlying metabolic abnormalities and precipitating factors. Immediate interventions included fluid resuscitation with isotonic saline to correct dehydration and improve hemodynamic stability. Electrolyte repletion was initiated to address hyponatremia and hypokalemia cautiously, considering the risk of osmotic demyelination syndrome and cardiac arrhythmias. Additionally, given the AKI secondary to rhabdomyolysis, two cycles of hemodialysis were performed to enhance clearance of myoglobin and metabolic waste products. Supportive measures were employed to optimize renal function and acid-base balance. With aggressive fluid and electrolyte management, as well as hemodialysis for AKI secondary to rhabdomyolysis, the patient's clinical condition gradually improved over the subsequent days. Serial laboratory monitoring demonstrated correction of electrolyte imbalances and improvement in renal function. Neurological examination revealed a progressive return to baseline mental status, with the patient becoming alert and oriented. Cardiac evaluation showed no evidence of acute ischemia or arrhythmias. The patient was discharged with close outpatient follow-up and recommendations for lifestyle modifications and medication adherence to prevent future metabolic decompensations.


CENTRAL LABORATORY

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Tel : 91 44 22415600 / 01 / 02 / 03

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Name : Mr. Saravana. B.N

Age/Sex : 70 Yrs / M

Patient ID : 7496423 IP No. : 1345051

Dept. : Dr.Hod-General Medicine-III -

Ref.DR :

Bill Date : 21-Mar-2024

Sample ID : 2403210442

Sample Collected on : 21-Mar-2024

Sample Received on : 21-Mar-2024

Reported on : 21-Mar-2024

Ward : ICU WARD Bed : ICU-06

Address: NO. 61/3 UDAYAM APPARTMENTS, BALAJI NAGAR, 3RD STREET,ANAKAPUTHUR

Sample	Test Description	Observed Value	Units	Bio.Reference
Serum	Urea	175	mg/dL	17.12-49.2
Serum	CREATININE	8.2	mg/dL	Male : 0.9-1.3 mg/dl Female: 0.6-1.1 mg/dl
Serum	Uric Acid	10.0	mg/dL	Male :4.4 - 7.6 mg/dl Female :2.5 - 6.6 mg/dl

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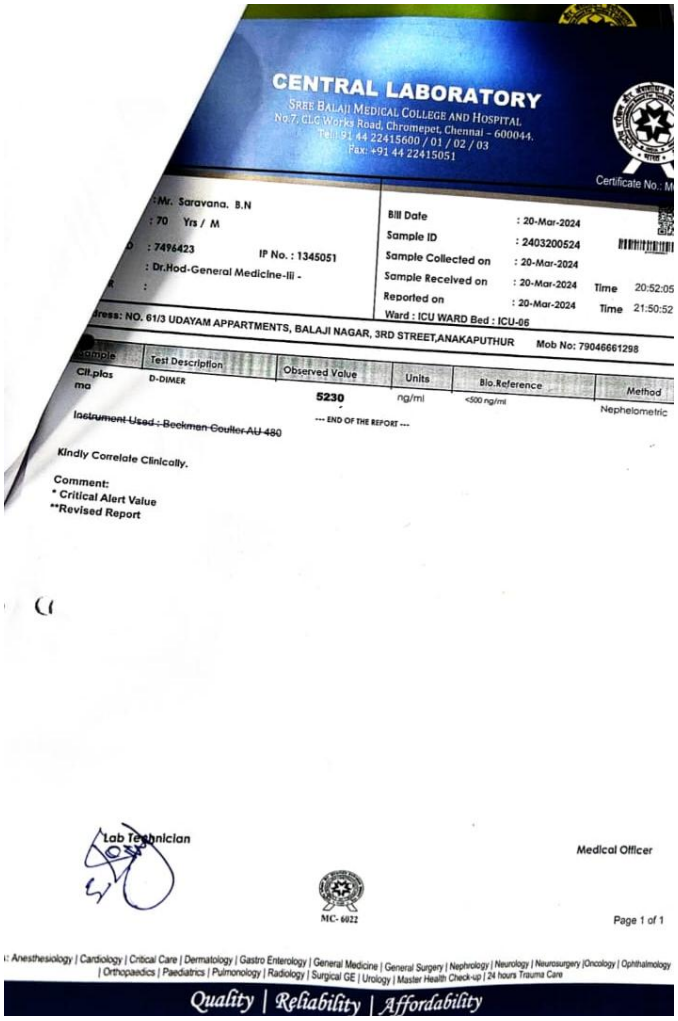
Comment:
* Critical Alert Value
**Revised Report

Image 1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Certificate No.: M

Mr. Saravana. B.N

: 70 Yrs / M

: 7496423 IP No. : 1345051

: Dr.Hod-General Medicine-III -

Bill Date : 20-Mar-2024

Sample ID : 2403200524

Sample Collected on : 20-Mar-2024

Sample Received on : 20-Mar-2024 Time 20:52:05

Reported on : 20-Mar-2024 Time 21:50:52

Ward : ICU WARD Bed : ICU-06

ress: NO. 61/3 UDAYAM APPARTMENTS, BALAJI NAGAR, 3RD STREET,ANAKAPUTHUR Mob No: 79046661298

Sample	Test Description	Observed Value	Units	Bio.Reference	Method
Cit.plas ma	D-DIMER	5230	ng/ml	<300 ng/ml	Nephelometric

--- END OF THE REPORT ---

Instrument Used : Beckman Coulter AU 480

Kindly Correlate Clinically.

Comment:
* Critical Alert Value
**Revised Report

Lab Technician

Medical Officer

MC- 6022

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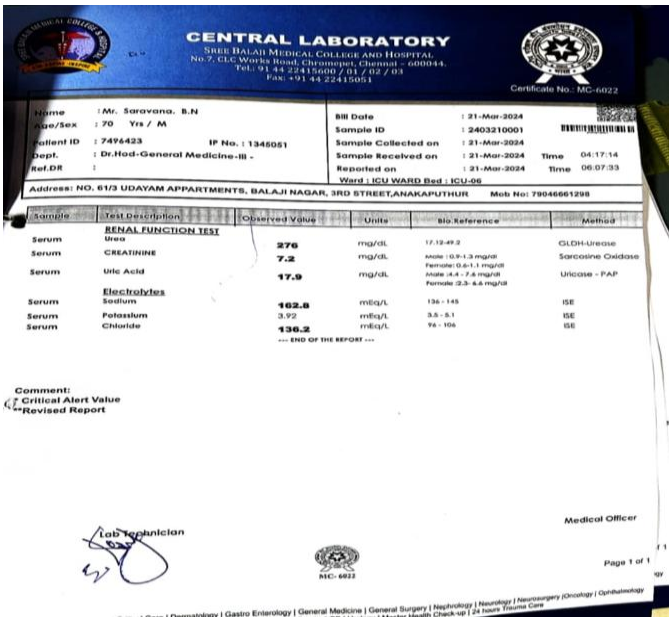
Anesthesiology | Cardiology | Critical Care | Dermatology | Gastro Enterology | General Medicine | General Surgery | Nephrology | Neurology | Neurosurgery | Oncology | Ophthalmology | Orthopaedics | Paediatrics | Pulmonology | Radiology | Surgical GE | Urology | Master Health Check-up | 24 hours Trauma Care

Quality | Reliability | Affordability

Image 2



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Certificate No.: MC-6022

Name	: Mr. Saravana. B.N	Bill Date	: 21-Mar-2024
Age/Sex	: 70 Yrs / M	Sample ID	: 2403210001
Patient ID	: 7496423 IP No. : 1345051	Sample Collected on	: 21-Mar-2024
Dept.	: Dr.Hod-General Medicine-III -	Sample Received on	: 21-Mar-2024 Time 04:17:14
Ref.DR	:	Reported on	: 21-Mar-2024 Time 06:07:33
		Ward : ICU WARD Bed : ICU-06	

Address: NO. 61/3 UDAYAM APPARTMENTS, BALAJI NAGAR, 3RD STREET,ANAKAPUTHUR Mob No: 79046661298

Sample	Test Description	Observed Value	Units	Bio.Reference	Method
	RENAL FUNCTION TEST				
Serum	Urea	276	mg/dL	17.12-49.2	GLDH-Urease
Serum	CREATININE	7.2	mg/dL	Male : 0.9-1.3 mg/dl Female: 0.6-1.1 mg/dl	Sarcosine Oxidase
Serum	Uric Acid	17.9	mg/dL	Male :4.4 - 7.6 mg/dl Female :2.3- 6.6 mg/dl	Uricase - PAP
	Electrolytes				
Serum	Sodium	162.8	mEq/L	136 - 145	ISE
Serum	Potassium	3.92	mEq/L	3.5 - 5.1	ISE
Serum	Chloride	130.2	mEq/L	96 - 106	ISE

--- END OF THE REPORT ---

Comment:
Critical Alert Value
**Revised Report

Lab Technician

Medical Officer

MC- 6022

Page 1 of 1

Image 3

Discussion

The discussion delves deeper into the complexities of acute encephalopathy with multifactorial complications, elucidating the diagnostic challenges, management strategies, clinical implications, and avenues for future research. Acute encephalopathy presents a diagnostic conundrum, particularly when compounded by multiple comorbidities and overlapping symptoms. In our case, the patient presented with altered mental status, electrolyte imbalances, renal dysfunction, and metabolic acidosis, necessitating a meticulous evaluation to discern the primary precipitating factors. The simultaneous presence of conditions such as hyponatremia, hypokalemia, hyperchloremia, AKI, and metabolic acidosis underscores the multifactorial nature of acute encephalopathy and highlights the importance of a comprehensive diagnostic approach. Distinguishing acute encephalopathy from other conditions with similar clinical manifestations, such as sepsis, cerebral ischemia, or drug toxicity, poses a significant challenge. Differentiating between primary and secondary causes of encephalopathy is crucial for targeted management. Moreover, the role of metabolic disturbances, including electrolyte imbalances and acid-base disorders, necessitates a systematic assessment to identify and address the underlying etiology. Diagnostic modalities such as imaging studies, laboratory investigations, and clinical assessment aid in delineating the primary precipitating factors and guiding appropriate management strategies. The management of acute encephalopathy revolves around addressing the underlying metabolic abnormalities and precipitating factors while mitigating further neurological deterioration. Immediate interventions aim to correct dehydration, restore hemodynamic stability, and normalize electrolyte imbalances cautiously to prevent complications. Fluid resuscitation with isotonic saline is essential to replenish intravascular volume and improve perfusion, particularly in the setting of hypotension and dehydration. Electrolyte repletion is a cornerstone of management, with careful attention to correcting hyponatremia and hypokalemia while avoiding rapid shifts that may precipitate osmotic demyelination syndrome or cardiac arrhythmias. In our case, the presence of AKI secondary to rhabdomyolysis necessitated hemodialysis to enhance clearance of myoglobin and metabolic waste products, highlighting the importance of addressing underlying precipitating factors. Supportive measures targeting renal function and acid-base balance play a pivotal role in optimizing clinical outcomes. Monitoring of renal function, electrolyte levels, and acid-base status guides the titration of interventions and ensures appropriate response to treatment. Close observation of neurological status facilitates the assessment of mental status and cognitive function, providing insights into the effectiveness of management strategies. The complexities of acute encephalopathy with multifactorial complications have

significant clinical implications, including the potential for adverse outcomes and prolonged hospitalizations if not promptly recognized and managed. The overlap in symptoms with other conditions underscores the importance of a thorough diagnostic evaluation to elucidate the primary etiology and guide targeted therapy. Moreover, the case highlights the need for a multidisciplinary approach involving neurology, nephrology, cardiology, and critical care to optimize patient care. Collaboration among specialists facilitates the integration of expertise and the development of comprehensive management plans tailored to individual patient needs. The successful management of acute encephalopathy hinges upon timely recognition, appropriate intervention, and vigilant monitoring. Early identification of metabolic disturbances and precipitating factors allows for prompt initiation of therapy, thereby minimizing the risk of complications and optimizing outcomes. Moreover, close follow-up and patient education are essential to prevent recurrence and promote long-term management strategies. Despite advances in the understanding and management of acute encephalopathy, several areas warrant further investigation. Research focusing on the pathophysiology of metabolic encephalopathy and its impact on neurological function may elucidate novel therapeutic targets and treatment modalities. Additionally, prospective studies evaluating the efficacy of different management strategies, including fluid resuscitation protocols, electrolyte repletion strategies, and renal replacement therapies, are needed to optimize clinical outcomes and minimize complications. Furthermore, exploring the role of biomarkers in predicting the development and severity of acute encephalopathy may aid in early diagnosis and risk stratification. Furthermore, research aimed at elucidating the long-term neurological sequelae and cognitive outcomes of acute encephalopathy is essential for understanding the full spectrum of the disease and guiding rehabilitation strategies.

Conclusion

Acute encephalopathy with metabolic underpinnings represents a clinical challenge requiring a nuanced understanding of its multifactorial etiology and tailored management strategies. Through this case report, we have elucidated the diagnostic intricacies and therapeutic considerations involved in managing such patients, underscoring the importance of a multidisciplinary approach and vigilant monitoring to optimize outcomes and prevent recurrence. Further research is warranted to explore novel therapeutic modalities and refine existing protocols for the management of acute encephalopathy in the context of metabolic disturbances.

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