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Case Report

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Successful Management of Acute Viral Hepatitis A complicated by Hypokalemia, Thrombocytopenia, Respiratory Alkalosis and Ascites: A Case Report

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Abstract

Introduction: Acute viral hepatitis A is a self-limiting liver infection caused by the hepatitis A virus (HAV). Although most pediatric cases are benign, some children may develop severe complications, including hypokalaemia, thrombocytopenia, respiratory alkalosis, and ascites. Prompt recognition and management of these complications are essential to improving outcomes. This case report discusses a 15-month-old child with acute viral hepatitis A who presented with these complications, highlighting the importance of early intervention.

Case Report: A 15-month-old boy was admitted to the Pediatric Intensive Care Unit (PICU) with a 3-day history of jaundice, vomiting, and bradycardia. Laboratory tests revealed severe hypokalaemia (K^+ = 2.84 mmol/L), deranged liver function tests, and thrombocytopenia. The patient was treated with intravenous fluids, potassium correction, broad-spectrum antibiotics, and supportive care. Imaging confirmed mild ascites, gallbladder wall edema, and splenomegaly. HAV infection was confirmed serologically (IgM positive). Over the next 48 hours, his condition improved, with the resolution of bradycardia, stabilization of electrolytes, and normalization of liver function. He was discharged after 8 days in stable condition, with follow-up planned for nutritional support and further liver monitoring.

Conclusion: This case illustrates the potential for complications such as hypokalaemia, thrombocytopenia, respiratory alkalosis, and ascites in pediatric patients with hepatitis A. Early identification and comprehensive supportive care can lead to favourable outcomes. Similar cases emphasize the need for careful monitoring and prompt intervention to prevent severe consequences in children with acute viral hepatitis A.

Keywords:- Hepatitis A, Hypokalaemia, Thrombocytopenia, Ascites

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INTRODUCTION

Acute viral hepatitis A is a self-limiting infection caused by the hepatitis A virus (HAV), primarily affecting the liver and leading to a wide range of clinical manifestations, from mild flu-like symptoms to severe jaundice and liver dysfunction. Although the infection is typically benign, certain pediatric patients can develop complications that require close monitoring and intervention. These complications, including hypokalaemia, thrombocytopenia, and respiratory alkalosis, are not uncommon in severe cases of hepatitis A and can significantly affect patient outcomes if not addressed promptly.[1]

Hepatitis A can lead to hypokalaemia due to a variety of mechanisms. One possible cause is vomiting, a common symptom of hepatitis A, which leads to the loss of potassium from the body. Additionally, patients with hepatic dysfunction may experience an imbalance in renal function, impairing the kidneys' ability to retain potassium. Hypokalaemia is a serious complication as it can result in life-threatening arrhythmias, such as bradycardia, which was observed in the present case. Early correction of hypokalaemia is crucial to prevent further cardiac complications.[2]

Thrombocytopenia is another complication that can occur in hepatitis A due to the virus's impact on the liver's ability to produce clotting factors. Additionally, the immune-mediated destruction of platelets can occur as a result of the viral infection, leading to decreased platelet counts. Thrombocytopenia increases the risk of bleeding, particularly in patients with compromised liver function, as the liver is also responsible for producing clotting proteins. In this case, the child presented with significant thrombocytopenia upon admission, necessitating careful monitoring and supportive care.[3]

Respiratory alkalosis is a less common but notable complication of viral hepatitis. It occurs due to hyperventilation, which can be triggered by hepatic dysfunction and its effects on the central nervous system. In patients with severe liver involvement, hepatic encephalopathy may alter the respiratory drive, leading to increased respiratory rates and the subsequent exhalation of excess carbon dioxide, causing an imbalance in blood pH. Respiratory alkalosis, if untreated, can lead to further electrolyte imbalances, complicating the patient's clinical course.[4]

In addition, mild ascites in hepatitis A can result from the liver's decreased synthetic function, particularly affecting albumin production, which plays a crucial role in maintaining oncotic pressure. Reduced oncotic pressure leads to fluid accumulation in the peritoneal cavity, resulting in ascites.[5]

This case report discusses a 15-month-old male child diagnosed with acute viral hepatitis A (HAV positive, IgM positive) who developed hypokalaemia, thrombocytopenia, respiratory alkalosis, and mild ascites. The case illustrates the importance of recognizing and managing these complications promptly, as they can significantly affect the clinical course and recovery. Through appropriate supportive care, electrolyte correction, and close monitoring, the patient showed a favourable outcome and was discharged in stable condition. The case report highlights the complex interplay between liver dysfunction and the systemic complications associated with acute hepatitis A in pediatric patients.

CASE REPORT

A 15-month-old male child, was admitted to the Pediatric Intensive Care Unit (PICU) with a 3-day history of jaundice, vomiting, and persistent bradycardia. Upon admission, an initial electrocardiogram (ECG) revealed bradycardia, and routine blood investigations showed severe hypokalaemia (K^+ = 2.84 mmol/L), thrombocytopenia (platelet count 20,000/cumm) and deranged liver function tests (LFTs). These findings, along with his clinical presentation, necessitated immediate admission to the PICU for close monitoring and management. On admission, he was immediately started on intravenous (IV) fluids to maintain hydration and correct the electrolyte imbalance. Given the clinical suspicion of infection, the patient was empirically started on IV Taxim (Cefotaxime) and IV Vancomycin for broad-spectrum antibiotic coverage, pending blood culture results.

To correct the hypokalaemia and prevent further cardiac complications, IV potassium correction was administered. Additionally, IV 3% Normal Saline (NS) was provided to manage his volume status, and IV Vitamin K was initiated in three doses to address the coagulopathy secondary to liver dysfunction, indicated by his prolonged prothrombin time. As part of his liver support, Lactulose syrup was prescribed to prevent hepatic encephalopathy by reducing ammonia production. Supportive care was also provided in the form of calcium supplementation, multivitamin and IV Pantoprazole was also given. Ondansetron was administered to control vomiting, and IV Paracetamol was used for fever control.

Over the first 48 hours his condition showed gradual improvement. His bradycardia resolved, and he became afebrile. With correction of his hypokalaemia and ongoing IV fluids, his oral intake improved, and his electrolyte levels began to stabilize. His blood cultures remained negative, prompting the discontinuation of IV antibiotics after five days. An abdominal ultrasound revealed gallbladder wall edema, mild splenomegaly (spleen measuring 6.8 cm), mild ascites, and mesenteric lymphadenopathy. These changes were consistent with the diagnosis of acute viral hepatitis. A serological test confirmed the presence of HAV IgM antibodies, confirming the viral etiology of his hepatitis. Patient continued to show significant clinical improvement. His SGOT and SGPT levels, though elevated initially, began to stabilize, and his ascites reduced. Regular ECG assessments confirmed no recurrence of bradycardia, and his electrolyte levels, particularly potassium, were maintained within normal limits.

By 7th day of admission his liver function tests were improving, and his oral intake had normalized. His platelet count, which was low at admission, stabilized over the course of his stay, and his electrolyte balance was restored. The IV fluids were gradually tapered, and he was transitioned to oral medications and supportive care.

On Day 8th day he was discharged with an advice to follow up after 5 days. During hospital stay he was noted to have dysmorphic features, although this was not a major concern during his hospitalization. His family was advised on the importance of continued nutritional support and follow-up monitoring of liver function tests to ensure a full recovery from hepatitis A. Further follow-up was arranged to address the failure to thrive and monitor for any complications related to liver dysfunction.

Day of Admission	Parameter	Value
Day 1	Serum Potassium (K^+)	2.32 mmol/L (Severe hypokalaemia)
	Hemoglobin (HB)	11.7 g/dL
	Total Leukocyte Count (TLC)	17,070 /Cumm (High)
	Platelet Count	20,000 /mm³ (Severe Thrombocytopenia)
	Blood Urea	20 mg/dL (normal)
	Serum Creatinine	0.5 mg/dL (normal)
	Total Bilirubin	7.5 mg/dL (High)
	SGOT (AST)	450 U/L (High)
	SGPT (ALT)	1190 U/L (High)
Day 3	Serum Potassium (K^+)	3.94 mmol/L (corrected)
	Platelet Count	Improving
	SGOT (AST)	133.9 U/L (High)
	SGPT (ALT)	561.4 U/L (High)
Day 7	Hemoglobin (HB)	11.5 g/dL (normal)
	Total Leukocyte Count (TLC)	7,200 /Cumm (normal)
	Platelet Count	90,000 /mm³ (improving from 20,000/mm³)
	Serum Potassium (K^+)	4.01 mmol/L (normal)
	Total Bilirubin	7.68 mg/dL (High)
	Direct Bilirubin	6.03 mg/dL (High)
	SGOT (AST)	108.5 U/L (High)
	SGPT (ALT)	289 U/L (High)
	Gamma-GT	551.4 U/L (High)
	Albumin	3.24 g/dL (Low)

Table 1 :- Summary of improvements in lab parameters in the case.

Discussion:-

Hepatitis A is generally considered a self-limiting disease in children, but severe complications such as hypokalaemia, thrombocytopenia, respiratory alkalosis, and ascites can arise in some pediatric patients, as demonstrated in this case of a 15-month-old male child. Early recognition and

intervention are critical to prevent serious outcomes.[6] This discussion elaborates on the clinical course, management, and review of similar complications seen in other pediatric cases of hepatitis A reported in the literature.

The primary complication in this case was hypokalaemia which can arise due to vomiting and hepatic impairment, which affects the body's ability to maintain normal potassium homeostasis. Hypokalaemia in hepatitis A has been linked to both renal dysfunction and excessive gastrointestinal losses, leading to further complications such as arrhythmias and bradycardia, as seen in this patient. Vidyut Bhatia et al conducted a comparative study to determine urinary potassium loss in children with acute liver failure (ALF) and acute viral hepatitis (AVH). For this purpose, the authors undertook a study comprising 25 patients with ALF and 84 with AVH, analyzing their clinical features, liver function, and potassium loss at presentation and day 45 follow-up. The study found that 60% of ALF patients were hypokalaemic compared to 12% of AVH patients, with inappropriate kaliuresis present in 80-100% of hypokalaemic individuals. On the basis of these findings, the authors concluded that potassium loss in ALF and AVH reverses with recovery.[7]

Thrombocytopenia, another significant finding in this case, is a known complication of hepatitis A, often attributed to both immune-mediated destruction of platelets and the liver's inability to produce clotting factors. A case series published by Urganci U reported a similar case of Immune Thrombocytopenic Purpura Associated with Hepatitis A Infection in a Five-year Old Boy similar to the patient in this case . Importantly, while thrombocytopenia can increase bleeding risk, the majority of pediatric patients recover without the need for platelet transfusions if managed conservatively.

Respiratory alkalosis in viral hepatitis is a less common but recognized complication. It often occurs secondary to hyperventilation, which can be triggered by hepatic encephalopathy and metabolic disturbances. There are various studies which have reported that children with severe hepatitis may experience hyperventilation and subsequent respiratory alkalosis . In our patient, early detection and management of the respiratory alkalosis helped in stabilizing the electrolyte imbalances and preventing further complications.[9]

The development of mild ascites in the context of acute hepatitis A is usually secondary to reduced albumin production and impaired liver function. Dagan R et al presented a case report of a seven-year-old child who developed acute ascites during recovery from hepatitis A. For this purpose, the authors described the child's progression, noting that the ascites resolved spontaneously within a few days without any medical intervention. The study found that no recurrence of ascites occurred over a five-year follow-up period. On the basis of these findings, the authors concluded that acute ascites can occur with hepatitis A but is not associated with poor prognosis. In this case also the ascites resolved with supportive care, including IV fluids, nutritional supplementation, and careful monitoring of liver function.[10]

Through prompt intervention, our patient’s complications, including hypokalaemia, thrombocytopenia, and respiratory alkalosis and ascites were effectively managed, leading to a favourable outcome. Similar cases of pediatric hepatitis A with these complications underscore the importance of early recognition and supportive care.

CONCLUSION

This case illustrates the successful management of a pediatric patient with acute viral hepatitis A, complicated by hypokalaemia, persistent bradycardia, and thrombocytopenia. Early intervention with electrolyte correction, liver support, and close monitoring of cardiac and liver function allowed for a favourable outcome. The patient was discharged in stable condition after seven days of comprehensive care, with a plan for follow-up to ensure continued recovery.

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