Diagnostic challenges in drug-induced hyperammonaemia.

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Introduction

Drug-induced hyperammonaemia is a significant clinical condition characterized by elevated levels of ammonia in the blood, often resulting from the administration of certain medications. Ammonia, a by-product of protein metabolism, is ordinarily detoxified in the liver through the urea cycle. However, several drugs can disrupt this process, leading to ammonia accumulation and subsequent clinical manifestations. This condition poses a challenge in clinical pathology and laboratory medicine due to its varied etiology, complex diagnostic criteria, and diverse clinical presentations [1].

Several mechanisms contribute to drug-induced hyperammonaemia. First, drugs such as valproic acid and topiramate inhibit enzymes critical to the urea cycle, impairing ammonia detoxification in the liver [2]. Second, antibiotics alter the composition of gut flora, leading to increased ammonia production from bacterial metabolism. Additionally, drugs like diuretics or ACE inhibitors can impair renal function, reducing ammonia excretion and contributing to hyperammonaemia[3].

Clinical presentation of drug-induced hyperammonaemia varies widely but commonly includes neurological symptoms such as confusion, lethargy, seizures, and coma. Gastrointestinal symptoms such as nausea, vomiting, and abdominal pain may also manifest, along with respiratory symptoms like respiratory alkalosis due to cerebral edema [4]. Recognizing these clinical signs is crucial for early diagnosis and intervention.

Laboratory investigations play a pivotal role in diagnosing drug-induced hyperammonaemia. Elevated blood ammonia levels are diagnostic, though levels may vary depending on the severity and chronicity of the condition[5]. Liver function tests help assess liver function and detect underlying liver disease, while arterial blood gas analysis evaluates acid-base status and lactate levels. Neuroimaging techniques such as MRI may reveal cerebral edema in severe cases, aiding in the assessment of neurological complications [6].

Management of drug-induced hyperammonaemia involves both discontinuing the offending drug and providing supportive care. Prompt cessation of the causative drug is essential to halt further ammonia accumulation[7]. Ammonia-lowering therapies such as intravenous sodium benzoate or sodium phenylacetate facilitate ammonia excretion and metabolic detoxification. Supportive care includes hydration, correction of electrolyte imbalances, and management of neurological symptoms through symptomatic therapy [8].

Prevention strategies focus on patient education and monitoring. Educating patients about symptoms of hyperammonaemia and the importance of medication adherence is crucial [9]. Regular monitoring of liver function and ammonia levels in high-risk patients allows for early detection and intervention. Careful consideration of alternative medications with a lower risk of causing hyperammonaemia, especially in patients with pre-existing liver or renal impairment, can mitigate the risk of this condition [10].

Conclusion

In conclusion, drug-induced hyperammonaemia presents significant challenges in clinical pathology and laboratory medicine due to its diverse etiology, complex clinical presentation, and potential for severe neurological sequelae. A multidisciplinary approach integrating clinical assessment, biochemical analysis, and therapeutic intervention is essential for effective management. By enhancing understanding and awareness of this condition, healthcare providers can optimize patient care and outcomes, ensuring safe and informed medication practices.

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