

Metabolic Impact of Tuberculosis on Liver and Blood Sugar

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DESCRIPTION

The liver is a vital organ responsible for regulating metabolism, detoxification, and maintaining blood sugar levels. When Tuberculosis (TB) spreads beyond the lungs, it can affect the liver, leading to inflammation and altered metabolic processes. Even in cases where the infection remains localized to the lungs, systemic inflammation triggered by TB can indirectly impact liver function. One of the key changes seen in TB is the disruption of glucose metabolism. The liver plays a central role in maintaining glucose levels through glycogen storage and gluconeogenesis (the production of glucose). TB related inflammation can interfere with these processes, leading to metabolic imbalances that can push the body toward insulin resistance—a precursor to diabetes. This disruption in glucose homeostasis highlights the liver's vulnerability during TB infection, even when the disease is primarily pulmonary. The inflammatory cascade can impair the liver's ability to balance glucose production and utilization, contributing to elevated blood sugar levels. These changes underscore the need for metabolic monitoring in TB patients, particularly those at risk for diabetes.

TB, inflammation and insulin resistance

Inflammation is an indicator of TB. The immune system's response to the infection involves the release of cytokines, which are proteins that help fight infection but also promote inflammation. Chronic inflammation can damage cells and impair the body's ability to use insulin effectively. In the liver, this can manifest as increased production of glucose, even when the body doesn't need it. This condition, known as hepatic insulin resistance, can contribute to persistently high blood sugar levels. Over time, this dysfunction can strain the pancreas, which produces insulin, potentially leading to the development of type 2 diabetes. The chronic inflammation in TB and metabolic disturbances underscores a complex relationship that can contribute to diabetes development. Elevated levels of pro-inflammatory cytokines, such as TNF-alpha and IL-6, not only impair insulin signalling but also disrupt lipid metabolism,

further exacerbating insulin resistance. In the liver, the combination of increased glucose output and reduced insulin sensitivity sets the stage for hyperglycemia. Concurrently, pancreatic beta cells may become overburdened as they try to compensate for rising blood sugar levels, eventually leading to beta-cell dysfunction. This interplay highlights the bidirectional relationship between TB and diabetes, where each condition can worsen the other, necessitating integrated management strategies.

Anti-TB treatment and its role in liver health

The medications used to treat TB can also impact liver metabolism. Many anti-TB drugs, such as isoniazid and rifampin, are metabolized in the liver and can cause liver toxicity or dysfunction in some patients. This adds an additional layer of complexity, as a compromised liver is less capable of managing blood sugar levels effectively. Furthermore, prolonged use of these medications may exacerbate metabolic disturbances, particularly in individuals who already have risk factors for diabetes, such as obesity or a family history of the disease. This interplay between TB medications and liver metabolism highlights the need for careful monitoring of liver function during treatment. Patients with pre-existing risk factors for diabetes or metabolic disorders may require more frequent evaluations to mitigate potential complications. Additionally, the hepatotoxic effects of drugs like isoniazid and rifampin can lead to altered glucose metabolism, increasing the risk of insulin resistance or hyperglycemia. Managing TB effectively while minimizing adverse metabolic effects necessitates a comprehensive, individualized treatment approach, including lifestyle interventions and possible adjunctive therapies.

The link between TB and diabetes

The relationship between TB and diabetes appears to be bidirectional. People with diabetes are more susceptible to TB due to their weakened immune systems. Conversely, TB can increase the risk of developing diabetes, as it disrupts normal metabolic processes. This dual interaction presents a significant public health challenge, especially in countries where TB and

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diabetes are prevalent. Understanding the connection between TB, liver metabolism, and diabetes opens avenues for more comprehensive treatment strategies. Individuals diagnosed with TB should be screened for diabetes, especially those with additional risk factors. Early detection can help manage both conditions more effectively. Regular monitoring of liver function in TB patients is essential to detect and address metabolic disturbances early. Healthcare providers should adopt a multidisciplinary approach, combining infectious disease specialists, endocrinologists, and dietitians to manage TB patients at risk of diabetes. Patients undergoing TB treatment should be encouraged to adopt a healthy diet and exercise

regimen to support liver health and reduce the risk of insulin resistance.

CONCLUSION

Tuberculosis is more than a lung disease is a systemic condition that can significantly impact liver metabolism and potentially lead to diabetes. The intricate interplay between inflammation, liver dysfunction, and glucose regulation underscores the need for holistic care in managing TB patients. By addressing these metabolic complications alongside the infection, healthcare providers can improve outcomes and reduce the burden of TB on global health.