

Monitoring Drug-Induced Hepatic Steatosis: Challenges and Non-Invasive Methods

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Abstract

Drug candidates can induce hepatic steatosis, which is characterized by the accumulation of lipids in liver cells without any other morphological changes. Clinical studies have reported two types of fat deposition patterns: diffuse and non-diffuse. The diffuse pattern is more common, while the non-diffuse pattern includes several subtypes such as geographic, focal, sub-capsular, multifocal, and perivascular patterns. Since drug-induced hepatic steatosis can have limited safety margins, it is crucial to develop methods to monitor its occurrence and severity during preclinical and clinical studies. However, liver biopsy, which is considered the gold standard for diagnosing hepatic steatosis, has several limitations, such as invasiveness, risk of hemorrhage and morbidity, and sampling errors due to the heterogeneity of fat distribution in the liver. Therefore, liver biopsy is not suitable for monitoring drug-induced hepatic steatosis, and there is currently no established monitoring method for this condition.

Keywords: Drugs • Clinical medicine • MRI

Introduction

Hepatic steatosis, also known as fatty liver disease, is a condition characterized by the accumulation of lipids in liver cells. While the diffuse pattern of hepatic steatosis is more common, non-diffuse patterns, such as geographic, focal, subcapsular, multifocal, and perivascular patterns, can also occur. Monitoring the occurrence and severity of drug-induced hepatic steatosis is crucial during preclinical and clinical studies. Liver biopsy is considered the gold standard for diagnosing hepatic steatosis. However, it is an invasive procedure that carries risks of hemorrhage, morbidity, and sampling errors. Therefore, non-invasive monitoring methods are preferred. Here are some non-invasive methods that can be used to monitor non-diffuse hepatic steatosis.

MRI is a non-invasive imaging technique that can detect hepatic steatosis with high sensitivity and specificity. MRI can detect the degree and distribution of fat deposition in the liver, including non-diffuse patterns. This imaging technique is safe, does not involve radiation exposure, and can be used to monitor the progression of hepatic steatosis over time. H-Magnetic Resonance Spectroscopy (H-MRS): H-MRS is a non-invasive technique that can quantify the amount of fat in the liver. It provides information on the type of lipid present in the liver, which can help distinguish between non-alcoholic fatty liver disease (NAFLD) and alcoholic fatty liver disease (AFLD). H-MRS can also be used to monitor the efficacy of treatment for hepatic steatosis [1,2].

Description

Methionine Choline Deficient (MCD) Diet: The MCD diet is a dietary intervention that induces hepatic steatosis in animal models. The MCD diet is low in methionine and choline, which are essential nutrients required for proper

liver function. This diet causes the accumulation of lipids in the liver, mimicking the pathophysiology of NAFLD. The MCD diet can be used to induce non-diffuse hepatic steatosis in animal models, which can be monitored using non-invasive methods such as MRI and H-MRS [3].

Blood tests can be used to monitor liver function and detect markers of hepatic steatosis, such as elevated levels of alanine aminotransferase (ALT) and aspartate aminotransferase (AST). However, blood tests are not specific for hepatic steatosis and may not be sensitive enough to detect non-diffuse patterns of hepatic steatosis. Non-invasive monitoring methods such as MRI and H-MRS can be used to monitor non-diffuse hepatic steatosis in preclinical and clinical studies. The MCD diet can be used to induce non-diffuse hepatic steatosis in animal models. Blood tests can be used to monitor liver function, but they may not be sensitive enough to detect non-diffuse patterns of hepatic steatosis. Overall, these non-invasive monitoring methods can provide valuable information on the occurrence and severity of drug-induced hepatic steatosis. Hepatic steatosis, a condition characterized by the accumulation of lipids in hepatocytes, is a growing concern worldwide. It can be caused by various factors such as obesity, type 2 diabetes, alcohol consumption, and certain medications. Drug-induced hepatic steatosis has become a significant concern for the pharmaceutical industry due to the increasing number of drugs that have been associated with this condition. Therefore, there is a need for non-invasive methods to monitor the occurrence and severity of drug-induced hepatic steatosis [4].

The gold standard for diagnosing hepatic steatosis is liver biopsy. However, this method is invasive and carries some risks, such as hemorrhage and morbidity. Sampling error is also a significant limitation of liver biopsy, as the fat distribution in the liver is heterogeneous. Therefore, it may not represent the whole liver accurately, leading to false-negative results. MRI is a non-invasive imaging technique that can be used to visualize the liver's fat content. The proton density fat fraction (PDFF) is a quantitative MRI technique that has been used to assess hepatic steatosis in both preclinical and clinical studies. PDFF values above 5% are considered indicative of hepatic steatosis. However, MRI is not always accessible, and the equipment is expensive. H-MRS is a non-invasive technique that can assess hepatic lipid content by measuring the signal intensity of hydrogen atoms in specific lipid molecules. This technique has been shown to be reliable in detecting hepatic steatosis, with high sensitivity and specificity. However, the equipment for H-MRS is not widely available, and the technique is expensive.

The MCD diet is a nutritional approach that induces hepatic steatosis in animal models. The diet is deficient in methionine and choline, which are essential components of phosphatidylcholine, a major component of cellular membranes [5,6].

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Conclusion

The MCD diet causes liver injury and fat accumulation, making it an ideal model for studying drug-induced hepatic steatosis. However, this model is limited by its ability to mimic human physiology accurately. Several blood biomarkers, such as alanine aminotransferase (ALT), aspartate aminotransferase (AST), and gamma-glutamyl transferase (GGT), have been used to assess liver function and detect hepatic injury. These biomarkers can also be used to monitor drug-induced hepatic steatosis. However, they are not specific to hepatic steatosis and may be influenced by other factors such as inflammation and viral infections. Drug-induced hepatic steatosis is a growing concern, and the development of non-invasive methods to monitor this condition is crucial. Although liver biopsy is the gold standard, it is invasive and carries risks. Non-invasive methods such as MRI, H-MRS, the MCD diet, and blood biomarkers have been shown to be effective in detecting hepatic steatosis. However, these methods have their limitations, and more research is needed to improve their accuracy and availability.

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Conflict of Interest

None.

References

1. Siegelman, Evan S and Mark A Rosen. "Imaging of hepatic steatosis." *In Sem in Liver Dis* 21 (2001): 071-080.

2. Nassir, Fatiha, R Scott Rector, Ghassan M Hammoud and Jamal A Ibdah. "Pathogenesis and prevention of hepatic steatosis." *Gastroenterol Hepatol* 11 (2015): 167.
3. Browning, Jeffrey D., Lidia S Szczepaniak, Robert Dobbins and Pamela Nuremberg, et al. "Prevalence of hepatic steatosis in an urban population in the United States: Impact of ethnicity." *Hepatology* 40 (2004): 1387-1395.
4. Hooper, Amanda J., Leon A Adams and John R Burnett. "Genetic determinants of hepatic steatosis in man." *J Lipid Res* 52 (2011): 593-617.
5. Browning, Jeffrey D and Jay D Horton. "Molecular mediators of hepatic steatosis and liver injury." *J Clin Investigat* 114 (2004): 147-152.
6. Mazhar, Sameer M., Masoud Shiehorteza and Claude B. Sirlin. "Noninvasive assessment of hepatic steatosis." *Clin Gastroenterol Hepatol* 7 (2009): 135-140.

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