Case Report

Focal hepatic steatosis as a rare complication of microwave ablation-induced portal vein thrombosis: A radiologic case report

Running head: Hepatic steatosis after portal vein thrombosis

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Abstract

Microwave ablation (MWA) is a widely used treatment for liver metastases, particularly for lesions smaller than 3 cm. Although generally safe, rare complications such as segmental or lobar portal vein thrombosis (PVT) can occur. We present a unique case of tumor-like focal fat deposition in the left hepatic lobe following left PVT, a rare complication of MWA, in a

59-year-old female with pancreatic adenocarcinoma liver metastasis. Post-ablation imaging revealed geographic-shaped areas of focal hepatic steatosis confined to the left lobe, accompanied by left PVT. Dual-echo MRI, a routine but highly effective imaging sequence for liver fat characterization, was instrumental in distinguishing benign fat deposition from tumor recurrence. This case highlights the importance of recognizing focal hepatic steatosis as a rare but significant complication of MWA and PVT, with implications for clinical management and follow-up strategies.

Keywords: Focal hepatic steatosis, microwave ablation, portal vein thrombosis

Introduction

MWA is an established, minimally invasive treatment for liver metastases, offering high efficacy for lesions smaller than 3 cm.[1] While generally well tolerated, rare complications such as segmental or lobar PVT have been reported.[2] Hepatic steatosis following PVT is an exceedingly rare phenomenon, with only one reported case in the context of liver transplantation.[3] Focal hepatic steatosis and sparing are frequently encountered in radiology practice and are often related to regional perfusion alterations. Dual-echo MRI, a routine sequence in liver imaging, is crucial for identifying fat-containing hepatic lesions and differentiating them from tumor recurrence. This report presents a rare instance of focal hepatic steatosis following left PVT, a complication of MWA performed for liver metastasis from pancreatic adenocarcinoma. In addition to presenting the clinical and imaging findings, we discuss the underlying pathophysiology of steatosis in the context of altered hepatic perfusion and portal venous flow.

Case Presentation

A 59-year-old female patient underwent distal pancreatectomy and splenectomy for pancreatic adenocarcinoma. During routine follow-up one year later, a metastatic lesion measuring 23 □ 18 mm was detected in liver segment 2 on abdominal magnetic resonance imaging (MRI) (Fig. 1). MWA was performed on the lesion (2+1 minutes, 150 watts) using a Covidien-brand antenna under ultrasound guidance by an interventional radiologist with over 9 years of experience. General anesthesia was preferred as the anesthesia method. Written informed consent was obtained from the patient, who voluntarily agreed to participate in the study.

Post-procedure abdominal CT was obtained on the same day to assess ablation efficacy and complications. The same-day CT confirmed the patency of the portal vein and inclusion of the metastatic lesion within the ablation zone. The patient was discharged the following day without complications. One month later, dynamic contrast-enhanced MRI and CT were performed as part of our institutional routine follow-up protocol. MRI revealed newly developed, T2 mildly hyperintense, geographic-shaped areas confined to the left lobe, without enhancement or diffusion restriction. Additionally, left PVT was observed. Fat suppression on out-of-phase images in the dual-echo sequence supported the diagnosis of focal hepatic steatosis (Fig. 2).

The patient exhibited a striking post-procedural elevation in CA 19-9 levels (>200-fold above ULN), which may be related to emerging pulmonary metastases, although this remains

speculative without confirmatory imaging. No local tumor recurrence or new hepatic lesions were identified. Elevated GGT and AST levels post-procedure, along with thrombocytosis, may reflect a combination of hepatic stress and systemic inflammatory response. The timing of laboratory tests ranged from one week before to one week after the procedure. A table summarizing laboratory values is provided (Table 1).

Discussion

Focal hepatic steatosis is a common imaging finding and is often related to regional alterations in hepatic perfusion.[3] In this rare case, focal hepatic steatosis developed in the left hepatic lobe following PVT induced by MWA. The pathophysiology likely involves decreased portal venous flow leading to localized hypoxia, impairment of lipid metabolism, and compensatory increase in hepatic arterial flow.[4,5] These changes may result in hepatocyte lipid accumulation and focal fatty infiltration. Experimental studies have supported the notion that regional hypoperfusion can induce hepatic steatosis by disrupting lipoprotein transport and altering hepatocellular metabolism.[6,7]

Although dual-echo MRI is a routine liver imaging sequence, its importance in characterizing focal hepatic fat is underscored in this case. The absence of enhancement and diffusion restriction, along with the presence of fat suppression in out-of-phase images, was key in differentiating benign steatosis from tumor recurrence, guiding appropriate clinical management.

MWA is generally safe, but PVT may occur due to thermal injury to adjacent vascular structures or the thrombogenic effects of ablated tissue. To minimize the risk of PVT, meticulous technique is crucial: maintaining a safe distance from major portal branches, using lower power settings near vascular structures, and ensuring proper needle placement are recommended. Routine follow-up after MWA in our institution includes an abdominal CT on the day of the procedure and dynamic contrast-enhanced MRI at 1 month to assess treatment response and detect early complications.

In this case, the left portal vein was patent on immediate post-procedure CT. The subsequent development of PVT at 1 month suggests a delayed thrombotic process, possibly related to endothelial injury or local inflammation induced by the ablation. Laboratory parameters are typically not discussed in detail in the case presentation section. Here, they are summarized in Table 1 for clarity. While CA 19-9 elevation may indicate disease progression or distant metastasis (e.g., pulmonary), a definitive link requires further imaging. Further follow-up in this patient includes serial imaging to monitor for tumor recurrence and resolution or progression of PVT. MRI with dual-echo and diffusion-weighted imaging, in combination with laboratory markers, remains the mainstay of follow-up.

Conclusion

This case underscores the importance of recognizing focal hepatic steatosis as a rare but significant complication following MWA-induced PVT. Although dual-echo MRI is a routine imaging tool, its utility in differentiating fat deposition from tumor recurrence is critical for clinical decision-making. Awareness of this rare complication, combined with thoughtful imaging follow-up and careful procedural planning, can help optimize patient outcomes.

Further research is needed to clarify the mechanisms linking altered portal perfusion to focal fat deposition and to refine post-ablation monitoring protocols.

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Table 1. Comparative analysis of laboratory parameters in pre- and post-ablation follow-up evaluations

Laboratory Parameters	Pre-Procedure	Post-Procedure Follow up
AST (U/L)	23	46
ALT (U/L)	15	35
GGT (U/L)	239	266
CA 19-9 (U/mL)	113	9397
Platelet Count (cells/□L)	649000	434000
Bilirubin (mg/dL)	0.36	0.61
INR	0.97	1.03
AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; GGT: Gamma-glutamyl transferase; INR: International normalized ratio.		

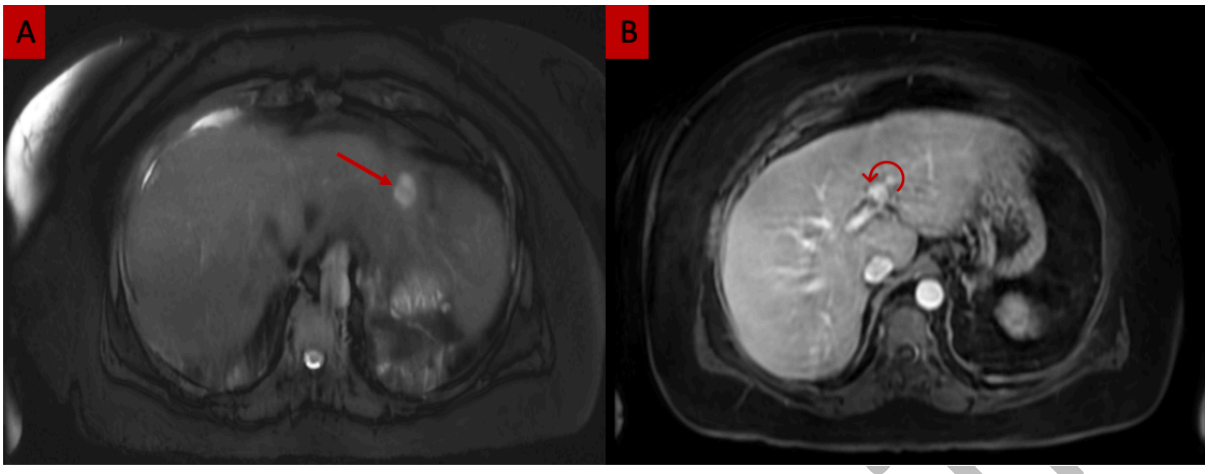

Figure 1. Pre-procedure T2-weighted MR imaging reveals a metastatic lesion in segment 2 (arrow) (A). Portal phase MRI demonstrates that the left portal vein remains patent (curved arrow) (B)

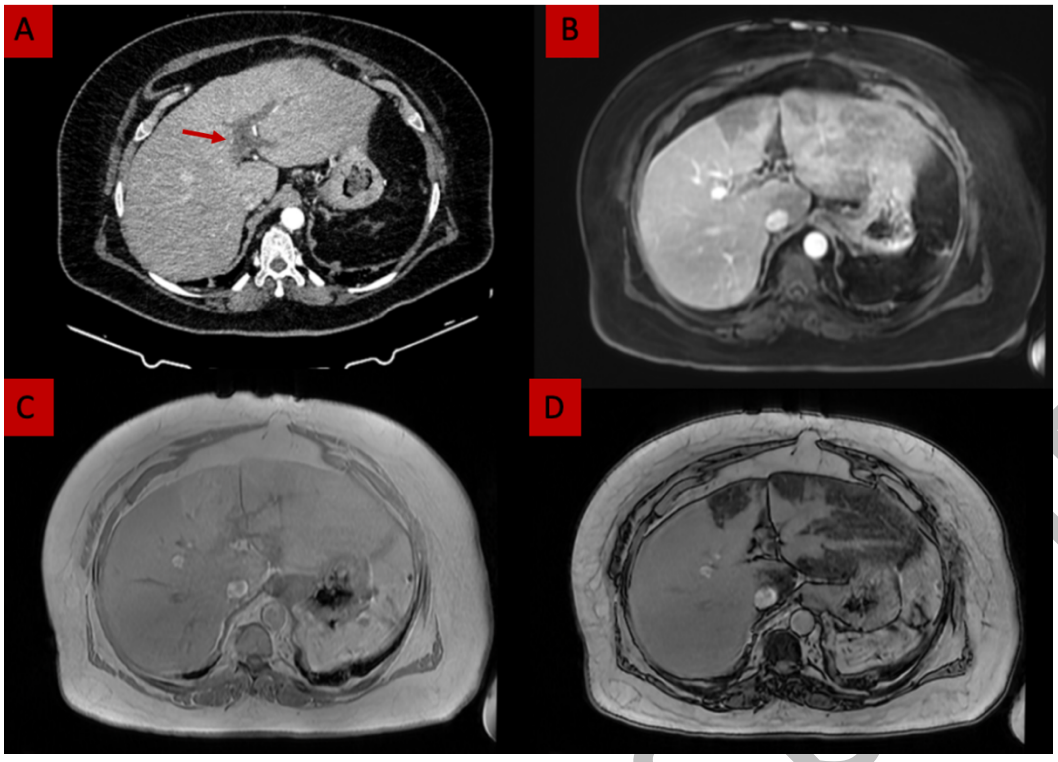

Figure 2. CT imaging demonstrates left portal vein thrombosis (arrow) (A). MR images (B-D) reveal patchy, nonenhancing areas (B), observed on dual-echo in-phase images (C), corresponding to focal fatty infiltration in the left liver lobe, confirmed by out-of-phase imaging (D)