


“WBC-HBP Dissociation” Unveils Occult Xanthogranulomatous Inflammation Mimicking Hepatocellular Carcinoma in Diabetic Patients

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Background: The reliance on LI-RADS 5 imaging criteria for the non-invasive diagnosis of hepatocellular carcinoma (HCC) can be precarious in the setting of advanced liver fibrosis, where benign inflammatory mimics may exhibit deceptive vascular kinetics. Hepatic xanthogranulomatous inflammation (XGI) represents a rare but significant diagnostic pitfall, as it can strictly simulate the arterial phase hyperenhancement and subsequent washout characteristic of malignancy. This diagnostic ambiguity is exacerbated in diabetic patients by “diabetic immunoparesis” which masks localized infection through a deceptive absence of systemic leukocytosis. This study highlights a novel diagnostic signature, termed “WBC-HBP Dissociation” utilizing Heparin-Binding Protein (HBP) to unveil occult neutrophil activation otherwise obscured by normocytosis.

Case Presentation: A 50-year-old male with chronic hepatitis B and biopsy-confirmed advanced fibrosis (LSM: 11.6 kPa) presented with a solitary, hypervascular liver mass. Despite uncontrolled Type 2 Diabetes (HbA1c: 12.00%) and a broad panel of negative tumor markers (AFP, PIVKA-II, CEA, CA19-9), both CEUS and MRI yielded a definitive LI-RADS 5 classification with suspected local invasion. Notably, while the white blood cell (WBC) count remained within the normal range ($5.83 \times 10^9/L$), a marked elevation in HBP (16.7ng/mL; Ref: <11.4ng/mL) suggested a localized inflammatory response. Guided by the radiological mandate for malignancy, the patient underwent an anatomical hepatectomy. Postoperative histopathology revealed XGI characterized by dense infiltration of lipid-laden foamy histiocytes, with immunohistochemistry (CD68+, SMA+, ALK-, IgG4-) effectively excluding neoplasia and IgG4-related disease.

Conclusion: XGI can authentically mimic the hemodynamic signature of HCC, a challenge compounded by the “relative washout” effect of background fibrosis. The “WBC-HBP Dissociation” identified here serves as a critical serological red flag for occult inflammation in patients with metabolic comorbidities. Integrating HBP assessment into the diagnostic algorithm for LI-RADS 5 masses—especially when tumor markers are negative—may justify advanced metabolic imaging or biopsy, potentially sparing high-risk patients from unnecessary and morbid major hepatic resections.

Plain Language Summary: Diagnosing Hepatocellular Carcinoma usually relies on specific patterns seen on CT or MRI scans. Yet, this method has a flaw. In patients with liver fibrosis, benign inflammatory lumps can mimic the exact appearance of a tumor. Such mimicry creates a dangerous trap, often leading to unnecessary major surgeries. The risk is even higher for diabetic patients. Diabetes causes “diabetic immunoparesis” a condition where the immune system reacts slowly. Consequently, standard blood tests may show normal white blood cell counts despite an active, hidden infection. We report a case where a diabetic patient’s liver mass mimicked cancer on all imaging modalities. His normal blood counts initially masked the infection. However, we detected a sharp rise in Heparin-Binding Protein (HBP). Unlike routine markers, HBP is released early and remains elevated even in diabetic patients. We term this mismatch “WBC-HBP Dissociation”. It was the critical clue identifying the mass as Xanthogranulomatous Inflammation, not cancer. Testing for HBP offers a safety net, helping surgeons avoid operating on inflammatory mimics.

Keywords: xanthogranulomatous inflammation, heparin-binding protein, hepatocellular carcinoma, diagnostic biomarkers, neutrophil activation, hemodynamic illusion

Introduction

The non-invasive diagnosis of hepatocellular carcinoma (HCC) primarily relies on the LI-RADS 5 imaging criteria: Arterial Phase Hyperenhancement (APHE) followed by washout.¹ While HCC pathogenesis involves complex cell signaling pathways,^{2,3} clinical diagnosis is largely morphological. However, the specificity of these imaging features can be compromised in patients with background liver fibrosis. In such cases, benign inflammatory lesions may exhibit a “pseudo-washout” appearance due to differential contrast kinetics.⁴ This occurs when contrast clears rapidly from the lesion while being retained in the surrounding fibrotic parenchyma due to expanded extracellular volume (ECV).⁵ False-positive diagnoses of LI-RADS 5 are clinically significant as they can lead to unnecessary major hepatic resections and increased patient morbidity.⁶ Among the mimics of malignancy, hepatic Xanthogranulomatous Inflammation (XGI) is a rare entity that is frequently overlooked in the differential diagnosis of HCC.⁷

The diagnostic difficulty is further increased in patients with metabolic comorbidities, particularly diabetes. In patients with uncontrolled diabetes, “diabetic immunoparesis” impairs neutrophil mobilization from the bone marrow,⁸ often resulting in normal White Blood Cell (WBC) counts even during active infection.⁹ This normal WBC count can mislead clinical decision-making by obscuring the infectious etiology of a hepatic mass. Clinicians currently face a blind spot: distinguishing these “radiologically aggressive” but “hematologically silent” lesions from HCC without invasive biopsies.¹⁰ Standard biomarkers often fail in this context.

In this report, we analyze a case of solitary hepatic XGI in a patient with concurrent liver fibrosis and diabetes. We propose a novel clinical sign: “WBC-HBP Dissociation.” Our data suggests that Heparin-Binding Protein (HBP), a pre-formed granule protein, provides superior sensitivity over routine leukocyte counts for detecting localized inflammation in this specific demographic.^{11,12}

Case Presentation

Clinical History and Laboratory Findings

A 50-year-old male was admitted for the management of uncontrolled Type 2 Diabetes Mellitus (T2DM). His medical history included chronic Hepatitis B virus (HBV) infection of 10 years’ duration. On admission, glycemic control was poor, with a Glycated Hemoglobin (HbA1c) level of 12.00%.

Routine preoperative laboratory tests were performed. Hematological parameters showed a normal White Blood Cell (WBC) count of $5.83 \times 10^9/L$ (Reference range: $3.5\text{--}9.5 \times 10^9/L$). Liver function tests revealed 14.6 IU/L for Alanine Aminotransferase (ALT) (Reference: 9–50 IU/L), 13.3 IU/L for Aspartate Aminotransferase (AST) (Reference: 15–40 IU/L), and 13.4 $\mu\text{mol/L}$ for Total Bilirubin (Reference: 0–26.0 $\mu\text{mol/L}$). Additionally, Alkaline Phosphatase (ALP) was 93 IU/L (Reference: 45–125 IU/L), Gamma-Glutamyl Transferase (GGT) was 46.3 IU/L (Reference: 10–60 IU/L), and Albumin was 36.5 g/L (Reference: 40–55 g/L).

The patient presented with severe hyperglycemia (HbA1c 12.00%). We suspected “diabetic immunoparesis,” a state where normal WBC counts fail to signal active inflammation. To investigate, we assessed Heparin-Binding Protein (HBP) as a direct marker of neutrophil activation. The results were significant: HBP levels were elevated at 16.7 ng/mL (Reference: 0–11.4 ng/mL), and C-reactive protein (CRP) measured 13.90 mg/L. These values confirmed neutrophil degranulation, even though systemic leukocytosis was absent.

The tumor marker panel was negative. Alpha-fetoprotein (AFP) was <2.00 ng/mL (Reference: 0–7 ng/mL), Protein Induced by Vitamin K Absence or Antagonist-II (PIVKA-II) was 8.38 ng/mL (Reference: 0–40 ng/mL), Carcinoembryonic Antigen (CEA) was 1.75 ng/mL (Reference: 0–5 ng/mL), and Carbohydrate Antigen 19–9 (CA19–9) was 9.89 U/mL (Reference: 0–30 U/mL). Transient elastography (FibroScan) indicated a Liver Stiffness Measurement (LSM) of 11.6 kPa, consistent with advanced hepatic fibrosis (F3–F4).

Imaging Assessment

Gray-scale ultrasound identified a hypoechoic nodule measuring approximately 34 mm × 29 mm in Segment V. The lesion exhibited indistinct boundaries and abutted the gallbladder wall (Figure 1A). Contrast-Enhanced Ultrasound (CEUS) demonstrated a “fast-in, fast-out” vascular pattern: rapid, homogeneous hyperenhancement in the arterial phase (19 s) (Figure 1B) followed by washout in the portal venous phase (60 s) (Figure 1C). This profile was consistent with hepatocellular carcinoma.

Dynamic CT scan findings were concordant with ultrasound. The non-contrast phase revealed a hypoattenuating nodule in Segment V (Figure 2A). The lesion displayed arterial phase enhancement (Figure 2B) and washout in the portal venous phase relative to the liver parenchyma (Figure 2C). This vascular profile met the LI-RADS 5 criteria for malignancy.

Magnetic Resonance Imaging (MRI) showed a mass measuring 4.7 cm × 3.5 cm spanning segments IVb/V. Diffusion-Weighted Imaging (DWI) demonstrated marked hyperintensity (Figure 3A), indicating restricted diffusion. On axial T1-weighted imaging, the mass appeared hypointense (Figure 3B). These findings supported a preoperative diagnosis of neoplasm.

Surgical Intervention and Pathological Diagnosis

Despite negative tumor markers, the lesion was classified as LI-RADS 5 based on arterial phase hyperenhancement and washout in a high-risk patient. In accordance with AASLD guidelines, the vascular pattern was considered diagnostic for

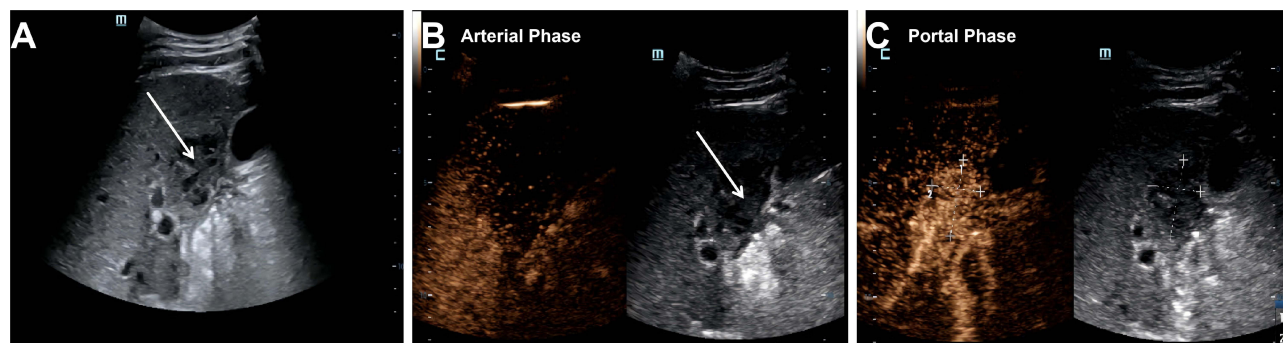


Figure 1 Contrast-enhanced ultrasound (CEUS) findings. (A) Conventional B-mode ultrasound reveals a hypoechoic nodule (arrow) in liver Segment V with indistinct boundaries adjacent to the gallbladder. (B) Arterial phase image (19 s) shows rapid hyperenhancement of the nodule (arrow). (C) Portal venous phase image (60 s) demonstrates washout (hypo-enhancement) of the lesion (arrow) compared to the surrounding liver parenchyma.

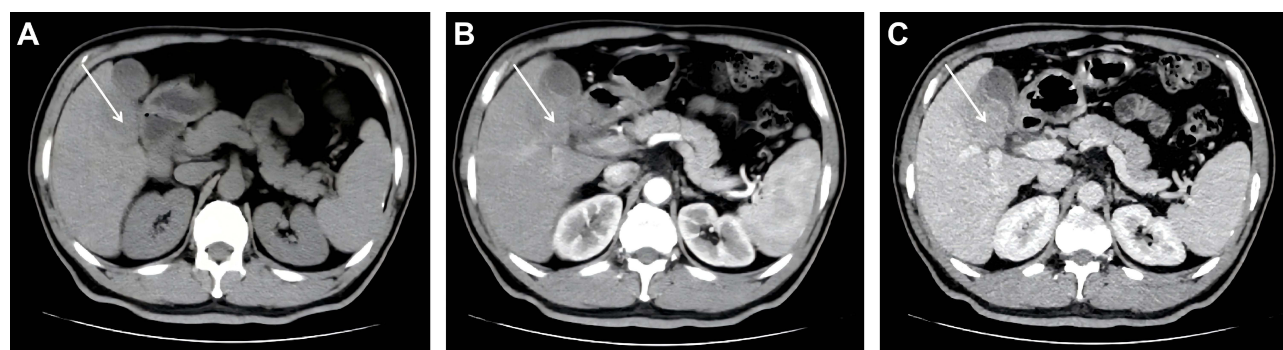


Figure 2 Dynamic Contrast-Enhanced CT (CECT) findings. (A) Non-contrast CT showing a hypoattenuating nodule (arrow) in Segment V. (B) Arterial phase imaging reveals intense enhancement of the lesion (arrow). (C) Portal venous phase imaging demonstrates distinct washout (hypo-attenuation) of the lesion (arrow) relative to the liver parenchyma, consistent with the LI-RADS 5 vascular profile.

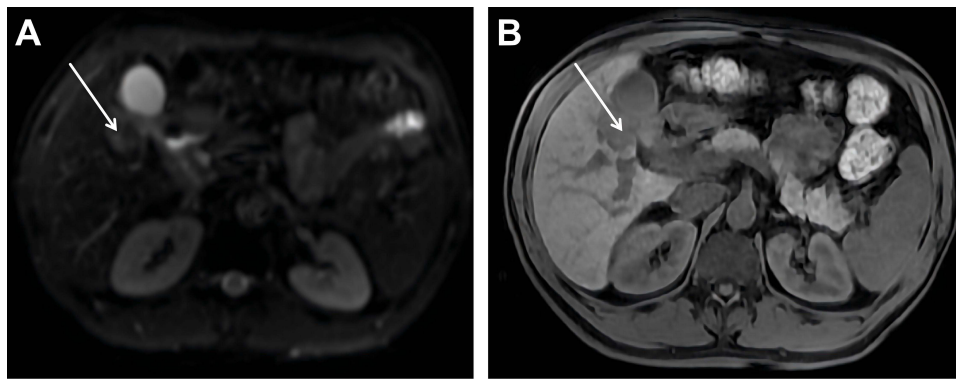


Figure 3 Magnetic Resonance Imaging (MRI) features. **(A)** Diffusion-weighted imaging (DWI) demonstrates marked hyperintensity (arrow), indicating restricted diffusion. **(B)** Axial T1-weighted imaging reveals a hypointense lesion (arrow) in Segment V.

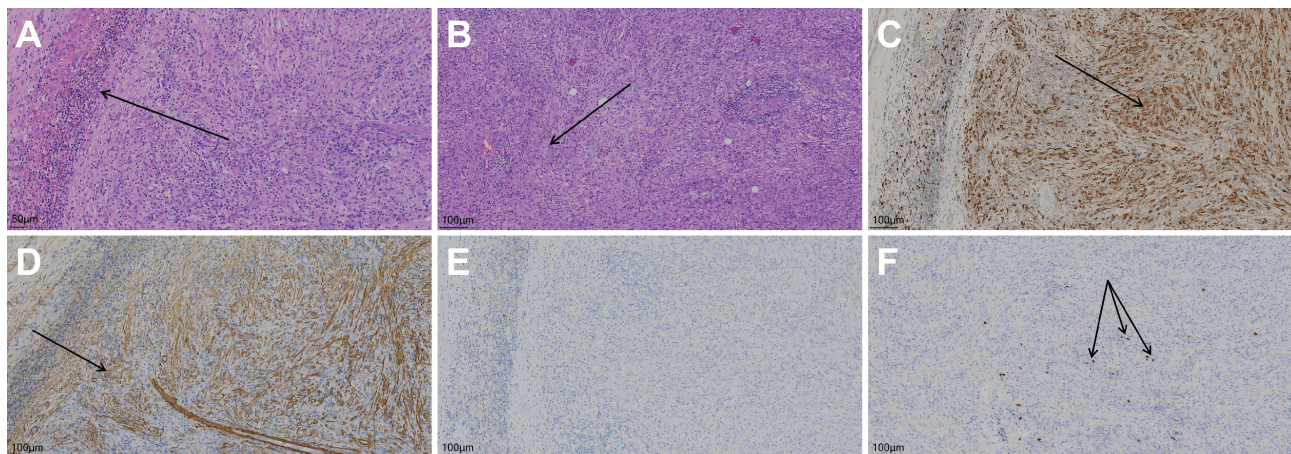


Figure 4 Histopathological and immunohistochemical characterization. **(A)** Low-power Hematoxylin and Eosin (H&E) staining ($\times 200$) demonstrates inflammatory infiltrates replacing the hepatic parenchyma (arrow). **(B)** High-power H&E staining ($\times 400$) reveals sheets of lipid-laden foamy histiocytes (arrow) with abundant clear cytoplasm. **(C)** Immunohistochemistry (IHC) for CD68 shows strong cytoplasmic positivity (arrow). **(D)** IHC for Smooth Muscle Actin (SMA) highlights spindle-shaped myofibroblasts (arrow) within the stroma. **(E)** IHC for Anaplastic Lymphoma Kinase (ALK) is negative. **(F)** IHC for IgG4 reveals only scattered positive plasma cells (arrows).

HCC, and biopsy was deferred. Given the subcapsular location, a laparoscopic anatomical liver resection (Segment 5) combined with cholecystectomy was performed.

Postoperative histopathology diagnosed Xanthogranulomatous Inflammation (XGI). Microscopically, the hepatic parenchyma was replaced by inflammatory infiltrates (Figure 4A). The lesion consisted predominantly of sheets of lipid-laden foamy histiocytes with clear cytoplasm (Figure 4B), intermingled with lymphocytes, plasma cells, and occasional multinucleated giant cells.

Immunohistochemical staining was performed to exclude malignancy and other inflammatory lesions. Foamy histiocytes were positive for CD68 (Figure 4C), and stromal spindle cells were positive for Smooth Muscle Actin (SMA) (Figure 4D). Anaplastic Lymphoma Kinase (ALK) was negative (Figure 4E), excluding Inflammatory Myofibroblastic Tumor (IMT). IgG4 staining revealed only scattered positive cells (Figure 4F), ruling out IgG4-related disease. Masson's trichrome staining confirmed background bridging fibrosis.

Discussion

Imaging Characteristics and Diagnostic Challenges

The primary diagnostic challenge in this case was the vascular profile observed on CEUS and CECT, which mimicked the classic “fast-in, fast-out” pattern of HCC. In patients with advanced fibrosis (LSM 11.6 kPa), the specificity of this pattern is reduced. The “washout” appearance observed in this case of XGI likely represents “pseudo-washout”.¹³ This

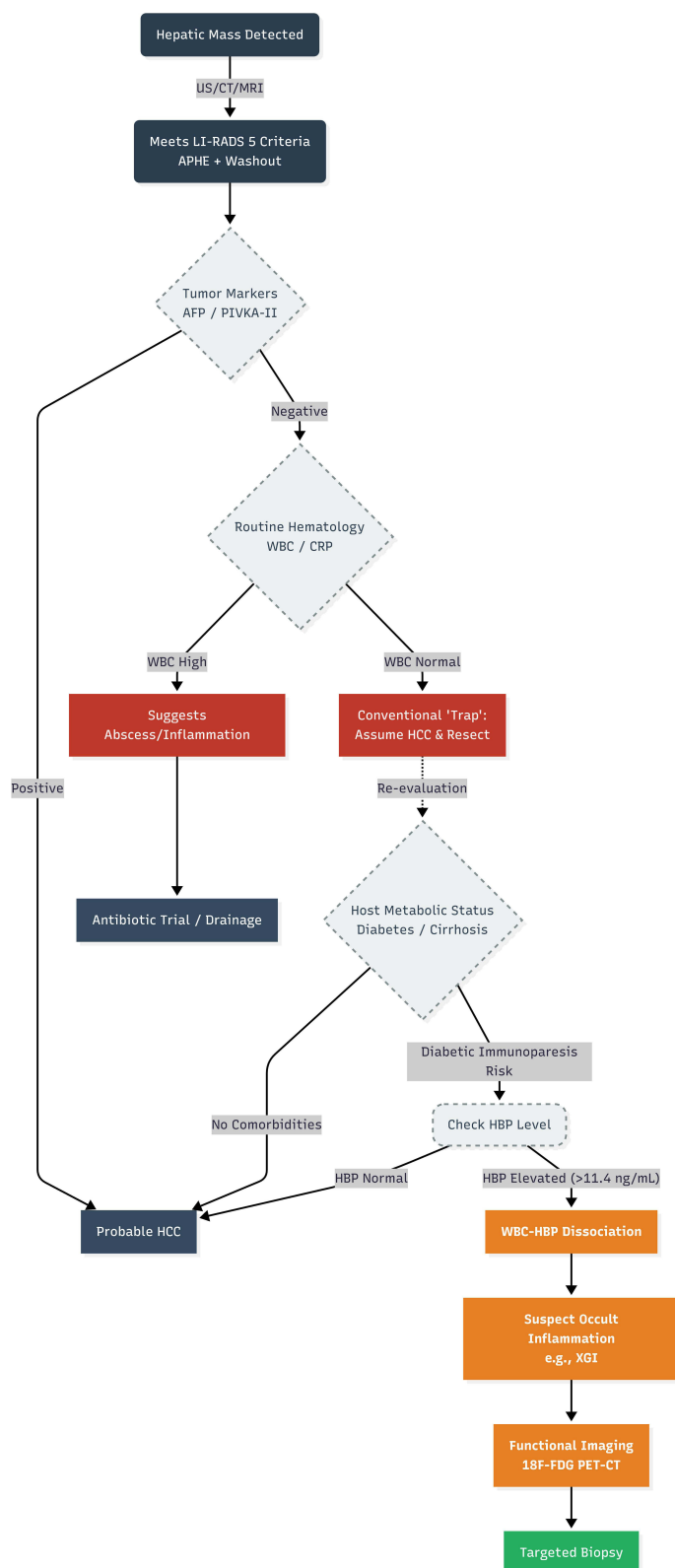


Figure 5 Proposed diagnostic algorithm. The flowchart illustrates the clinical decision-making pathway. Patients with LI-RADS 5 imaging features but negative tumor markers and normal WBC counts (Steps 1–3) represent a diagnostic challenge. In patients with metabolic comorbidities like diabetes (Step 4), assessing Heparin-Binding Protein (HBP) is recommended. An elevated HBP level (>11.4 ng/mL) despite normocytosis (“WBC-HBP Dissociation”) suggests occult inflammation, justifying the use of functional imaging (18F-FDG PET-CT) or targeted biopsy (Step 5) to avoid unnecessary hepatectomy.

Abbreviations: APHE, Arterial Phase Hyperenhancement; HCC, Hepatocellular Carcinoma; XGI, Xanthogranulomatous Inflammation.

phenomenon occurs when rapid contrast clearance from the hypervascular inflammatory lesion is contrasted against prolonged retention in the fibrotic parenchyma.¹⁴ While arterial hyperenhancement in XGI is caused by inflammatory hyperemia, the relative hypodensity in the venous phase can easily be mistaken for malignancy. Other inflammatory lesions, such as inflammatory pseudotumors and hepatic tuberculosis, can also present with similar imaging features, complicating the differential diagnosis.¹⁵

Mechanism of WBC-HBP Dissociation The discrepancy between normal WBC counts and elevated HBP levels (“WBC-HBP Dissociation”) reflects differences in their release kinetics. Systemic leukocytosis relies on a delayed bone marrow response, which is often impaired in diabetic patients due to “diabetic immunoparesis”.^{16,17} Hyperglycemia can blunt neutrophil chemotaxis and mobilization, resulting in normal cell counts despite active infection. In contrast, HBP is a pre-formed protein stored in neutrophil granules and is released immediately upon activation.^{18,19} Crucially, HBP is released by activated neutrophils regardless of the inflammation site. It is not specific to hepatic pathology but serves as a systemic indicator of neutrophil degranulation. In this patient, HBP leaked into the circulation from the localized hepatic inflammation, providing a sensitive marker that was detectable even when the systemic cellular response was suppressed.²⁰

Clinical Implications Strict adherence to LI-RADS criteria without considering clinical context can lead to misdiagnosis and overtreatment. Major hepatic resections for benign lesions carry significant risks of morbidity, emphasizing the need for accurate preoperative differentiation.^{21,22} We suggest that in high-risk patients (eg., those with diabetes) presenting with “radiologically aggressive” but “hematologically silent” (normal WBC) lesions, HBP should be assessed. An elevated HBP level in the absence of leukocytosis may warrant a deviation from the standard resection algorithm. In such cases, 18F-FDG PET-CT or targeted biopsy should be considered before surgery, as XGI typically demonstrates intense FDG uptake distinct from many well-differentiated HCCs.²³ Based on these clinical implications, we propose a diagnostic algorithm to guide the preoperative evaluation of indeterminate hepatic masses in high-risk patients (Figure 5).

Limitations

This study has several limitations. First, as a single case report, the findings require validation in larger cohorts to establish the diagnostic accuracy of HBP for hepatic inflammatory mimics. Second, HBP is a non-specific marker of inflammation; its elevation can be caused by infections in other organs, which must be ruled out clinically. Third, preoperative PET-CT and biopsy were not performed in this case. Their inclusion might have altered the surgical management and avoided anatomical resection.

Conclusion

This case underscores a critical diagnostic pitfall: Heparin-Binding Protein (HBP) levels can reveal inflammation when standard counts fail. Hepatic Xanthogranulomatous Inflammation (XGI) can indistinguishably mimic the hemodynamic profile of Hepatocellular Carcinoma, especially in fibrotic livers. For diabetic patients, “diabetic immunoparesis” further complicates diagnosis by masking serological signs of infection. We identified that elevated HBP combined with normal WBC counts—the “WBC-HBP Dissociation”—serves as a robust indicator of occult inflammation. Integrating HBP assessment into the diagnostic algorithm for indeterminate hepatic masses provides a crucial filter. This strategy helps identify inflammatory mimics, justifying the use of PET-CT or biopsy, and ultimately spares high-risk patients from unnecessary hepatic resections.

Ethics Approval and Informed Consent

The protocol for this clinical case study was formally reviewed and granted approval by the Medical Research Ethics Committee of The First Affiliated Hospital of the University of Science and Technology of China (Institutional Review Board No: 2026-RE-109). Institutional approval was required to publish the case details and was granted by the aforementioned committee. This study was conducted in strict accordance with the ethical standards of the responsible committee on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008. Furthermore, we confirm that comprehensive written informed consent was obtained from the patient prior to the submission, specifically authorizing the publication of clinical details and associated radiological and histopathological imagery. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Consent for Publication

Written informed consent was obtained from the patient for the publication of this case report and any accompanying images.

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Disclosure

The authors report no conflicts of interest in this work.

References

- Kulkarni AM, Kruse D, Harper K, et al. Current state of evidence for use of MRI in LI-RADS. *J Magn Reson Imaging*. 2025;62:640–653. doi:10.1002/jmri.29336
- Das J, Barman B, Sarma P, Das BK, Chetia R, Kalita PP. Signalling pathways in hepatocellular carcinoma (HCC) metastasis and invasion: molecular mechanisms and therapeutic implications. *Biochem Biophys Rep*. 2025;44:102403.
- Habeeb M, Vasanthan M. Formulation, interaction analysis, and invitro hepatocellular carcinoma studies of Rutin loaded lipid and polymeric nanoparticles. *Int J Nano Dimens*. 2025;16(2). doi:10.57647/ijnd.2025.1602.09
- Joo I. Preventing false positive imaging diagnosis of HCC: differentiating HCC from mimickers and practical strategies. *J Liver Cancer*. 2025;25(2):217–232. doi:10.17998/jlc.2025.07.29
- Obmann VC, Ardoino M, Klaus J, et al. MRI extracellular volume fraction in liver fibrosis—a comparison of different time points and blood pool measurements. *J Magn Reson Imaging*. 2024;60(4):1678–1688. doi:10.1002/jmri.29259
- An C, Rakhmonova G, Choi J-Y, Kim M-J. Liver imaging reporting and data system (LI-RADS) version 2014: understanding and application of the diagnostic algorithm. *Clin Mol Hepatol*. 2016;22(2):296–307. doi:10.3350/cmh.2016.0028
- Okamoto T, Suda T, Ishigami K, et al. Hepatic xanthogranuloma that originated from a liver cyst and mimicked a malignant tumor. *Am J Case Rep*. 2020;21:e925827.
- Dou Q-L, Liu J, Zhang W, et al. Dynamic changes in heparin-binding protein as a prognostic biomarker for 30-day mortality in sepsis patients in the intensive care unit. *Sci Rep*. 2022;12(1):10751. doi:10.1038/s41598-022-14827-1
- Casqueiro J, Casqueiro J, Alves C. Infections in patients with diabetes mellitus: a review of pathogenesis. *Indian J Endocrinol Metab*. 2012;16(Suppl 7):S27–S36. doi:10.4103/2230-8210.94253
- Hamed AE, Elwan N, Naguib M, et al. Diabetes association with liver diseases: an overview for clinicians. *Endocr Metab Immune Disord Drug Targets*. 2019;19(3):274–280. doi:10.2174/1871530318666181116111945
- Fisher J, Kahn F, Wiebe E, et al. The dynamics of circulating heparin-binding protein: implications for its use as a biomarker. *J Innate Immun*. 2022;14(5):447–460. doi:10.1159/000521064
- Yang W, Dong W. Heparin-binding protein as a diagnostic and prognostic marker of infections: a systematic review and meta-analysis. *Mediterr J Hematol Infect Dis*. 2025;17(1):e2025029. doi:10.4084/MJHID.2025.029
- Yang HK, Burns PN, Jang H-J, et al. Contrast-enhanced ultrasound approach to the diagnosis of focal liver lesions: the importance of washout. *Ultrasonography*. 2019;38(4):289–301. doi:10.14366/usg.19006
- Terzi FVO, Parente DB, Camargo GC, et al. MRI-derived extracellular volume to assess liver fibrosis in patients with metabolic-associated steatotic liver disease. *Abdom Radiol*. 2025;50(11):5223–5231. doi:10.1007/s00261-025-04945-5
- Zhang J, Lin H, Li X, et al. Xanthogranulomatous inflammation involving the gallbladder, bile duct, and pancreas: a case report. *Front Oncol*. 2023;13:1191181. doi:10.3389/fonc.2023.1191181
- Berbudi A, Rahmadika N, Tjahjadi AI, Ruslami R. Type 2 diabetes and its impact on the immune system. *Curr Diabetes Rev*. 2020;16(5):442–449. doi:10.2174/1573399815666191024085838
- Margalin B, Arfijanto MV, Hadi U. Effector function and neutrophil cell death in the severity of sepsis with diabetes mellitus. *Narra J*. 2024;4(1):e532. doi:10.52225/narra.v4i1.532
- Dowey R, Iqbal A, Heller SR, Sabroe I, Prince LR. A bittersweet response to infection in diabetes; targeting neutrophils to modify inflammation and improve host immunity. *Front Immunol*. 2021;12:678771. doi:10.3389/fimmu.2021.678771
- Tapper H, Karlsson A, Mörgelin M, Flodgaard H, Herwald H. Secretion of heparin-binding protein from human neutrophils is determined by its localization in azurophilic granules and secretory vesicles. *Blood*. 2002;99(5):1785–1793. doi:10.1182/blood.V99.5.1785
- Linder A, Christensson B, Herwald H, Björck L, Åkesson P. Heparin-binding protein: an early marker of circulatory failure in sepsis. *Clin Infect Dis*. 2009;49(7):1044–1050. doi:10.1086/605563
- Lee Y-T, Wang JJ, Zhu Y, Agopian VG, Tseng H-R, Yang JD. Diagnostic Criteria and LI-RADS for Hepatocellular Carcinoma. *Clin Liver Dis*. 2021;17(6):409–413. doi:10.1002/cld.1075
- Vengateswaran HT, Habeeb M, You HW, Aher KB, Bhavar GB, Asane GS. Hepatocellular carcinoma imaging: exploring traditional techniques and emerging innovations for early intervention. *Med Novel Technol Dev*. 2024;24:100327. doi:10.1016/j.medntd.2024.100327
- Shono N, Otomi Y, Otsuka H, Shinya T, Harada M. Multicystic biliary hamartoma with xanthogranulomatous inflammation on 18F-FDG PET/CT. *Clin Nucl Med*. 2022;47(10):882–884. doi:10.1097/RLU.0000000000004204

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