

**Title:**

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**Authors:**

Monica Descalzo Alfonso, Alejandro Fernández Soro, Victoria Lobo Antuña, Javier Garrido Gallego, Encarnación Martínez Leandro, María Ángeles Salazar Cabrera, Carlos Alventosa-Mateu, Juan José Urquijo Ponce

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## Multifocal hepatic inflammatory pseudotumor: review

Mónica Descalzo Alfonso<sup>1</sup>

Alejandro Fernández-Soro<sup>2</sup>

Victoria Lobo-Antuña<sup>1</sup>

Javier Garrido Gallego<sup>3</sup>

Encarnación Martínez Leandro<sup>4</sup>

Carlos Alventosa-Mateu<sup>2</sup>

Maria Ángeles Salazar Cabrera<sup>1</sup>

Juan José Urquijo Ponce<sup>2</sup>

1. Internal Medicine Department. General University Hospital Consortium, Valencia (Spain).
2. Hepatology Unit. Digestive Diseases Department. General University Hospital Consortium, Valencia (Spain).
3. Oncology Department. General University Hospital Consortium, Valencia (Spain).
4. Anatomical Pathology Department. General University Hospital Consortium, Valencia (Spain).

Correspondence to Alejandro Fernández Soro, Hepatology Unit, Digestive Diseases Department, General University Hospital Consortium, Valencia (Spain), Av Tres Cruces Nº 2, CP 46014, Valencia (España). E-mail: afdzsoro@gmail.com

## Abstract

**Background:** Inflammatory pseudotumor (IPT) is a rare benign lesion that can closely mimic malignant neoplasms both clinically and radiologically. Hepatic inflammatory pseudotumor (HIPT) is an uncommon manifestation, with fewer than 300 cases reported in the literature.

**Case Presentation:** We present the case of a 67-year-old male with a three-week history of fever and weight loss, and constitutional symptoms. Evaluation with MRI showed multiple hepatic lesions, raising suspicion for metastatic disease or hepatic abscesses. A percutaneous liver biopsy was performed, and histopathological analysis demonstrated a lymphoplasmacytic-rich fibroinflammatory lesion with numerous IgG4-positive plasma cells, consistent with HIPT. The patient was managed conservatively with antibiotics, leading to full clinical recovery and complete radiological resolution after 10 months.

**Conclusion:** Multifocal HIPT poses a significant diagnostic challenge due to its radiologic similarity to hepatic malignancies. Liver biopsy remains critical for establishing an accurate diagnosis and preventing unnecessary surgical intervention.

**Keywords:** Case report. Liver neoplasms. Inflammatory pseudotumor. IgG4-related disease. Liver biopsy. Hepatic mass.

Dear Editor,

Hepatic inflammatory pseudotumor (HIPT) is a rare benign condition that mimics malignant liver tumors. Its cause is unclear and has been linked to multiple factors including IgG4-related disease. Diagnosis depends on histopathology, typically showing mixed inflammatory infiltrates and sometimes IgG4-positive plasma cells.

Due to nonspecific symptoms, HIPT is often misdiagnosed. Treatment can be conservative but surgery may be required in some cases. Accurate diagnosis prevents overtreatment.

#### Case presentation

A 67-year-old man presented with a three-week history of fever (up to 38.5 °C) and 7 kg weight loss. History included daily alcohol intake, treated hepatitis C, resolved hepatitis B, and appendectomy. Blood tests showed leukocytosis ( $19.3 \times 10^9/L$ ), elevated CRP (26.38 mg/dL), and mild liver enzyme abnormalities. Abdominal ultrasound revealed five hypoechoic hepatic lesions. Empirical antibiotics were started at Emergency.

Further studies showed normal IgG4 (81.9 mg/dL), negative tumor markers, and sterile cultures. PET-CT ruled out extrahepatic disease. MRI suggested hepatic abscesses; metastases couldn't be excluded. Liver biopsy revealed 15 IgG4+ plasma cells/HPF and an IgG4/IgG ratio of 60%, compatible with hepatic inflammatory pseudotumor (HIPT). (Figure 1).

Antibiotic treatment was completed despite negative cultures, and corticosteroids were not used due to favorable evolution. The patient improved clinically and was discharged. Follow-up imaging at 10 months confirmed complete resolution.

## Discussion

Inflammatory pseudotumor (IPT) is a rare lesion that mimics malignancy, most commonly found in the lung [1]. Hepatic IPTs (HIPTs) represent up to 8% of extrapulmonary IPTs, with inflammatory or autoimmune etiologies [1,2]. They typically affect middle-aged men and are multifocal in 36% of cases [3].

Typical MRI signs such as the “double ring” can resemble either metastases or abscesses, requiring histological confirmation [1,2]. Two main histological subtypes exist: fibrohistiocytic (xanthogranulomatous inflammation, giant cells, neutrophils) and lymphoplasmacytic, the latter defined by diffuse infiltrates with >10 IgG4+ plasma cells/HPF or >40% IgG4+ cells [4]. Associated findings include eosinophilic infiltration, obliterative phlebitis, and periductal fibrosis.

A subset corresponds to IgG4-related disease (IgG4-RD), marked by tumefactive lesions, elevated serum IgG4, and steroid responsiveness [5,6]. Hepatic involvement in IgG4-RD is variable and includes IPT-like lesions, sclerosing cholangitis, or autoimmune hepatitis [5]. Our patient fulfilled histologic but not serologic criteria for IgG4-RD. IgG4+ cell presence alone is insufficient; serological findings are also needed [5]. Still, IgG4-RD should remain in the differential diagnosis of hepatic masses [6].

Corticosteroids are first-line treatment in confirmed IgG4-RD and effective in HIPTs with IgG4+ infiltrates [2,5]. Although corticosteroids could have been considered in our case, resolution without them questions an IgG4-RD etiology.

## Conclusion

HIPT is a rare benign condition often misdiagnosed. Biopsy confirms diagnosis and helps avoid unnecessary surgery.

#### Declaration of Interests

In the preparation of this work, the author(s) employed ChatGPT-4o to assist with the review and enhancement of the article's writing. The authors declare no other competing interests.

#### References

1. Patnana M, Sevrakov AB, Elsayes KM, Viswanathan C, Lubner M, Menias CO. Inflammatory pseudotumor: the great mimicker. *AJR Am J Roentgenol*. 2012;198(3):W217–W227. doi:10.2214/AJR.11.7288.
2. Patel H, Nanavati S, Ha J, Shah A, Baddoura W. Spontaneous resolution of IgG4-related hepatic inflammatory pseudotumor mimicking malignancy. *Case Rep Gastroenterol*. 2018;12(2):311–316. doi:10.1159/000490100.
3. Nguyen ED, Wen KW, Kakar S, Balitzer DJ. Clinicopathologic features and outcomes of hepatic inflammatory pseudotumour (IPT) and hepatic IPT-like lesions. *Histopathology*. 2025;86(4):525–535. doi:10.1111/his.15357.
4. Schiappacasse FG, Vallejos CA, Figueroa DP, Salcedo RA, Bustamante MC, Humeres AR. Pseudotumor inflamatorio hepático: caso clínico y revisión de la literatura. *Rev Méd Chile*. 2023;151(9):1255–1259. doi:10.4067/S0034-98872023000901255.

5. Lee HE, Zhang L. Immunoglobulin G4-related hepatobiliary disease. *Semin Diagn Pathol.* 2019;36(6):423–433. doi: 10.1053/j.semdp.2019.07.007.
6. Kim SR, Kim SK, Koma YI, Sasaki M, Asai A, Nishikawa H. Hepatic inflammatory pseudotumor—focusing on its heterogeneity. *Diagnostics (Basel).* 2023;13(17):2857. doi:10.3390/diagnostics13172857

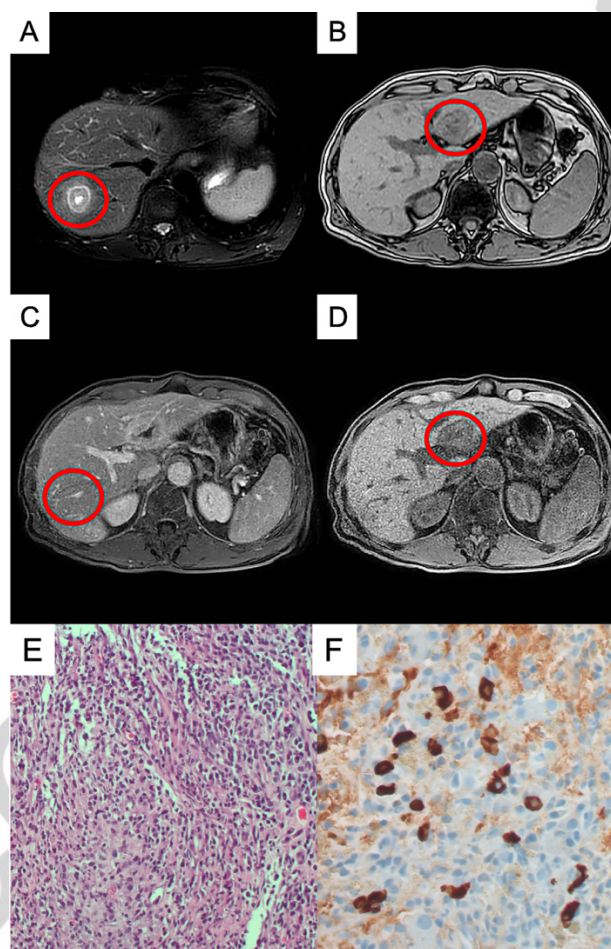


Figure 1. A-D) MRI showing two lesions (red circles) with thick peripheral enhancement and central hypointensity, forming a “double ring” pattern in T1 and hyperintense central signal in T2. E) Hematoxylin-eosin staining showing fibroinflammatory stroma. F) IgG4 immunostaining



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