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# Giant Remodeled Hepatic Cavernous Hemangioma Mistaken for Focal Nodular Hyperplasia: A Case Report

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**Abstract** **Case Report**

***Background.*** Hepatic cavernous hemangioma is the most common benign liver tumor, while focal nodular hyperplasia (FNH) is the second most common. Both are usually distinguished on cross-sectional imaging, but a hemangioma that has undergone internal remodeling thrombosis, fibrosis, hyalinization, and hemorrhage may lose its characteristic features and closely simulate FNH, leading to diagnostic uncertainty. ***Case presentation.*** A 26-year-old man with no significant medical history presented with a six-month history of late postprandial vomiting and no other associated symptoms. Physical examination revealed a soft, non-tender abdomen with no palpable mass. Contrast-enhanced CT showed a hypervascular mass of hepatic segment I with exophytic extension toward the hilum, measuring 11.5 × 10 × 9 cm and compressing adjacent vascular structures; the morphological and kinetic features were reported as compatible with FNH. Liver MRI confirmed a large lobulated process without a clearly individualized capsule in the left liver, isointense on T1 and T2, with strong arterial enhancement and portal-phase homogenization, and a central scar that was hypointense on T1, hyperintense on T2, and showed faint delayed enhancement again interpreted as FNH. The patient underwent surgical resection of the exophytic inter-hepatogastric mass arising from segment I (Spiegel lobe). Histopathology demonstrated a benign hepatic vascular lesion consistent with a remodeled cavernous hemangioma, with no evidence of malignancy. ***Conclusion.*** A remodeled giant hepatic hemangioma can mimic FNH on both CT and MRI, particularly when a central scar and atypical enhancement are present. A central scar is not specific to FNH and should be interpreted with caution. Histopathological examination remains the definitive arbiter when imaging is inconclusive or when a large, symptomatic, or anatomically complex lesion warrants resection.

**Keywords:** Cavernous hemangioma; Focal nodular hyperplasia; Central scar; Spiegel lobe; Caudate lobe; Liver MRI; Diagnostic pitfall; Hepatic surgery.

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## INTRODUCTION

Benign focal liver lesions are increasingly encountered as a consequence of the widespread use of abdominal imaging. They form a spectrum of non-malignant entities, most commonly cavernous hemangioma, followed by focal nodular hyperplasia (FNH) and hepatocellular adenoma, and are often discovered incidentally in patients with non-specific symptoms and without risk factors for liver malignancy.

Cavernous hemangioma is the most frequent benign hepatic tumor. It is more common in women and is usually asymptomatic and solitary, although it may be multiple in up to half of patients. Most are diagnosed between the third and fifth decades. FNH is the second most common benign hepatic mass; it is composed of benign hyperplastic hepatocytes arranged around a central fibrous scar, occurring within otherwise normal or nearly normal liver, and is widely regarded as a hyperplastic response to a localized vascular anomaly rather than a true neoplasm. An association between hemangioma and FNH has been repeatedly reported, supporting the view that both may share a vascular basis.

On standard imaging the two lesions are distinct: the typical hemangioma is markedly hyperintense on T2-weighted images and enhances with peripheral, discontinuous, nodular enhancement and progressive centripetal fill, whereas FNH is a homogeneous, strongly arterially enhancing lesion that becomes isointense in later phases and carries a delayed-enhancing central scar. Difficulty arises when a hemangioma is large and internally remodeled, because thrombosis, hyalinization, fibrosis, and hemorrhage modify both its signal and its enhancement kinetics, and

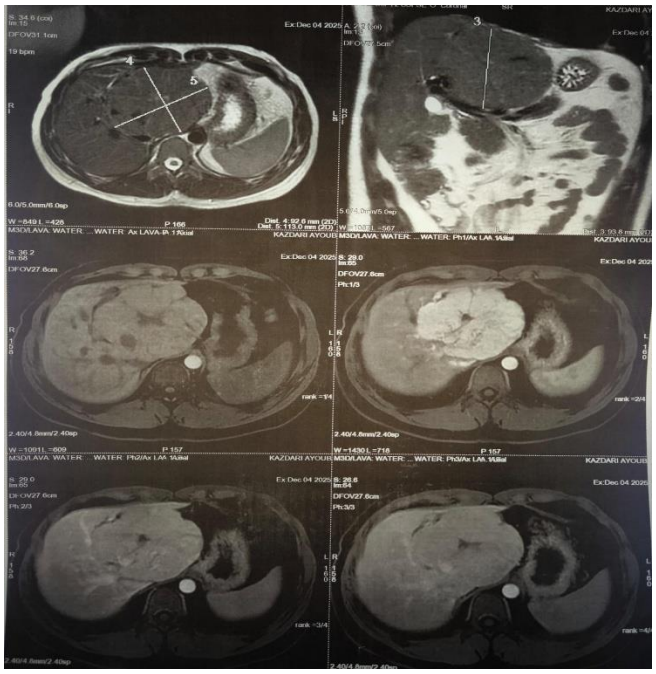
a central scar may then appear an imaging overlap that creates a genuine diagnostic pitfall. We report such a case, in which a giant remodeled hemangioma of the Spiegel lobe was interpreted as FNH on both CT and MRI.

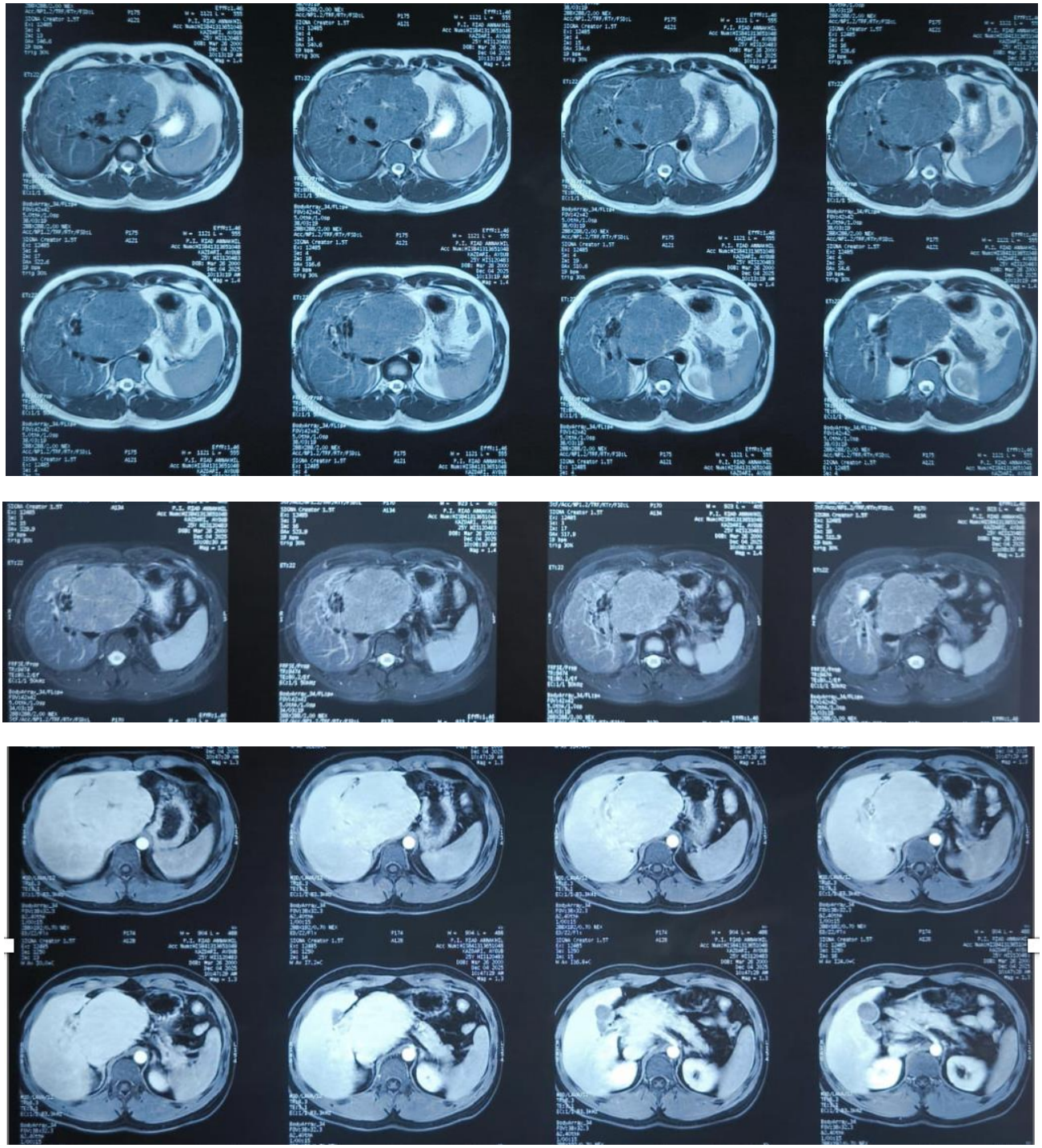
## Case Presentation

A 26-year-old man with no significant past medical history presented with a six-month history of late postprandial vomiting of undigested food, without any other associated symptom no abdominal pain, weight loss, jaundice, or fever and with no chronic medication or relevant family history. On physical examination, the abdomen was soft and non-tender, with no palpable mass and no hepatomegaly, and the remainder of the clinical examination was unremarkable. Contrast-enhanced abdominal CT revealed a hypervascular mass of hepatic segment I with exophytic development toward the hepatic hilum, measuring 11.5 × 10 × 9 cm and compressing the adjacent vascular structures, with morphological and kinetic characteristics reported as compatible with focal nodular hyperplasia (Figure 1). Liver MRI performed subsequently demonstrated a large lesional process with lobulated contours and no clearly individualized capsule in the left liver, isointense on T1- and T2-weighted sequences, showing strong arterial enhancement with portal-phase homogenization and a central scar that was hypointense on T1, hyperintense on T2, and faintly enhancing in the delayed phase; the process measured 92.6 × 113 × 93.8 mm, and the appearance was again considered to favor FNH of the left liver (Figure 2). In view of the lesion's size, the compressive symptoms, and its hilar location, surgical resection was undertaken: the patient underwent resection of the exophytic inter-hepatogastric mass arising from segment I (Spiegel lobe), together with inter-hepatogastric and subhepatic drainage using two Salem tubes. Intraoperative exploration confirmed an exophytic, hypervascular inter-hepatogastric mass arising from the Spiegel lobe and measuring 7 cm in its greatest axis, in intimate contact with the inferior vena cava and the portal vein, supplied by two feeding pedicles draining respectively into the inferior vena cava and the portal vein (Figure 3). Histopathological examination of the specimen showed features consistent with a benign hepatic vascular lesion of the remodeled cavernous hemangioma type, with no histological argument for malignancy (Figure 4). The postoperative course was uneventful, and the patient was discharged on the fourth postoperative day.



**Figure 1: Contrast-enhanced CT: hypervascular exophytic mass of segment I**





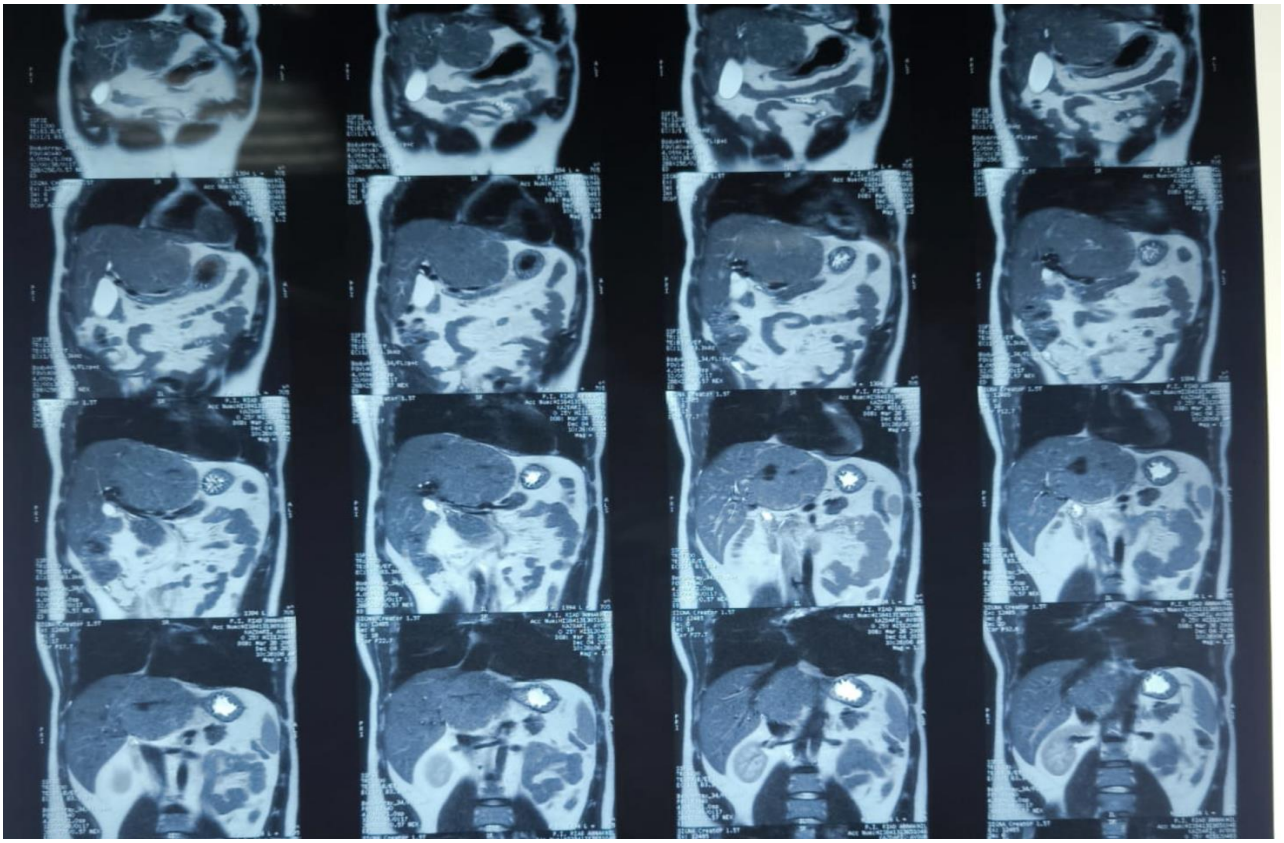


**Figure 2: Liver MRI: lobulated lesion with central scar; arterial enhancement and portal homogenization**

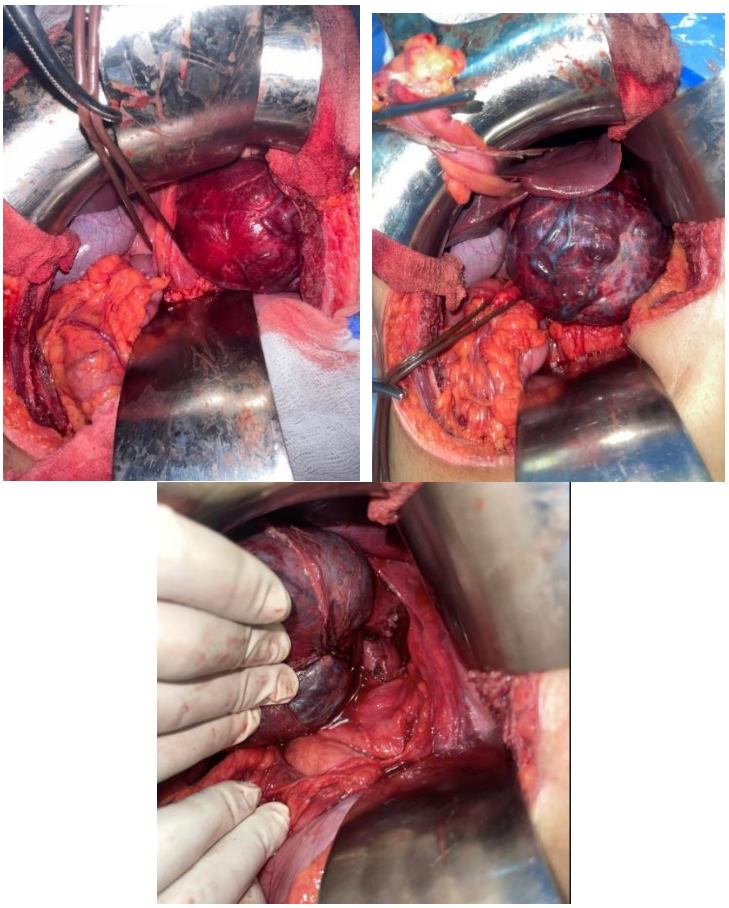


**Figure 3: Intraoperative view of the inter-hepatogastric Spiegel-lobe mass abutting the IVC and portal vein**

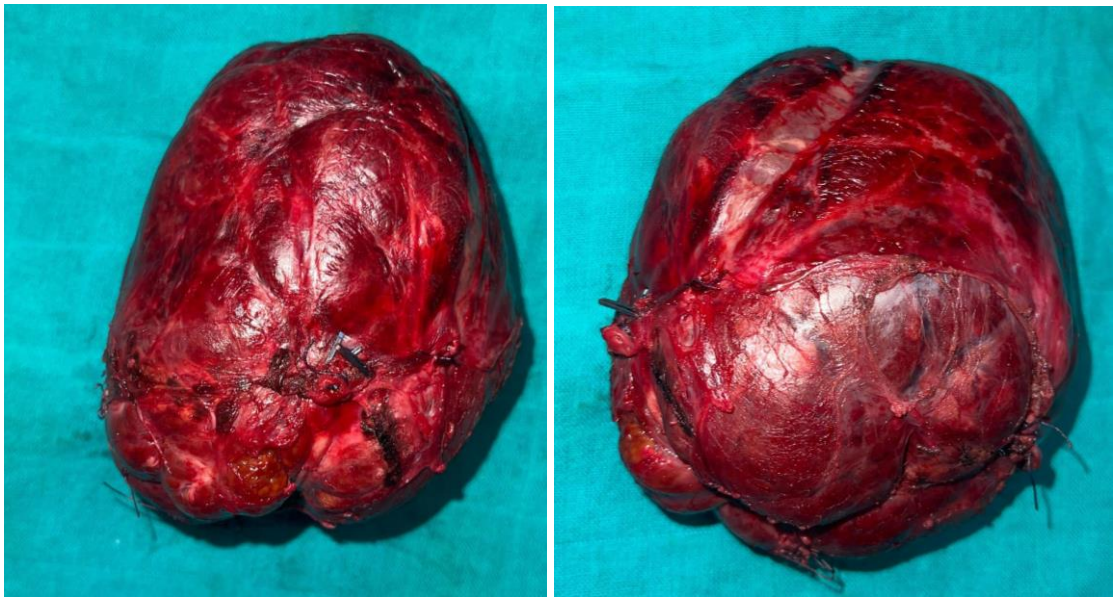


**Figure 4: Anterior and posterior view of the Specimen after extraction**

## DISCUSSION

This case illustrates a recognized but under-appreciated diagnostic trap: a giant hepatic cavernous hemangioma whose internal remodeling produced an imaging phenotype that was read as FNH on two independent modalities, and several aspects of the case warrant discussion.

The demographic profile was itself atypical. Both hemangioma and FNH predominate in women, and FNH in particular is classically described in women in the third and fourth decades, although it can occur at any age and in men [1]. A giant, symptomatic vascular lesion in a 26-year-old man is therefore unusual and, in itself, a reason not to anchor too firmly on a single radiological diagnosis.

The lesion was also symptomatic by mass effect rather than incidental. The late postprandial vomiting in this patient is best explained by extrinsic gastric and duodenal compression from a bulky exophytic mass at the hilum, an uncommon presentation given that most hemangiomas and most FNH lesions are asymptomatic. Symptoms attributable to a benign liver lesion generally arise only when the lesion is large, as it was here [3].

Most instructively, the imaging overlap centered on the central scar and the enhancement pattern. A central or eccentric scar is the imaging hallmark most clinicians associate with FNH, but it is not specific: it is also described in giant hemangiomas, in fibrolamellar hepatocellular carcinoma, and occasionally in conventional hepatocellular carcinoma, cholangiocarcinoma, and some metastases [2,8]. The classic teaching that "not all giant lesions with a central scar are hemangiomas" has an equally important corollary that a central scar does not establish FNH. In FNH the scar is typically hyperintense on T2 and enhances on delayed images, which is precisely what was observed here; yet the same delayed-enhancing T2-bright scar can be seen in a remodeled hemangioma, so the sign loses its discriminating value in large, altered lesions.

The enhancement kinetics were likewise misleading. A typical hemangioma fills peripherally and centripetally and remains markedly T2-hyperintense, whereas this lesion showed strong arterial enhancement with portal-phase homogenization and only iso-signal on T2 an FNH-like behavior. Internal remodeling is the likely explanation: thrombosis, extensive hyalinization, fibrosis, and hemorrhage are well-described in giant hemangiomas and alter both signal intensity and the dynamic enhancement profile, blurring the boundary with hypervascular hepatocellular lesions [2]. The pitfall is not confined to CT and MRI: FDG-avid cavernous hemangiomas have even been reported to mimic malignancy on PET/CT [9]. When ahypervascular lesion does not display the expected progressive peripheral nodular fill, the differential must remain open to FNH, adenoma, and malignant hypervascular tumors, and the clinical context must be weighed alongside the images.

The location further compounded both the diagnostic and the surgical challenge. The Spiegel lobe (the left portion of the caudate lobe, segment I) is an uncommon site, deeply seated between the liver, the stomach, the inferior vena cava, and the portal vein. In this patient the mass was in intimate contact with the inferior vena cava and the portal vein and drained through two feeding pedicles into these vessels, which made resection technically demanding and carried a substantial hemorrhagic risk inherent to vascular lesions in this position; caudate-lobe hemangiomas are recognized as among the most challenging resections in liver surgery [5].

Ultimately, the case underscores the role of histopathology as the definitive arbiter. Cross-sectional imaging, and MRI in particular, resolves most benign focal liver lesions confidently, and asymptomatic, typical FNH and hemangioma require no treatment [1,3]. When the lesion is large, symptomatic, anatomically complex, or radiologically atypical, however, imaging alone may be insufficient, and a similar diagnostic dilemma has been reported for atypical, massive, or pedunculated FNH [7]. Here, resection was justified by the compressive symptoms, the size, and the hilar location independent of the precise preoperative label, and pathology ultimately reclassified the lesion as a remodeled cavernous hemangioma rather than FNH. The surgical strategy for giant hemangiomas, enucleation versus anatomical resection, is likewise dictated by lesion size, symptoms, and the relationship to vascular structures [6]. The practical message is to treat the central scar and FNH-like enhancement of a giant hepatic lesion as suggestive rather than diagnostic, and to let the clinical picture and, when needed, histology complete the assessment.

## Conclusion

A giant cavernous hemangioma that has undergone internal remodeling can convincingly mimic focal nodular hyperplasia on both CT and MRI, especially when it harbors a central scar and shows atypical arterial enhancement. The central scar is not pathognomonic of FNH and should be interpreted within the full clinical and radiological context. In large, symptomatic, or anatomically complex lesions such as a Spiegel-lobe mass in contact with major vessels surgical resection with histopathological confirmation remains the most reliable way to reach a definitive diagnosis and to relieve compressive symptoms.

**Declarations**

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

**Conflicts of interest.** The authors declare no conflicts of interest.

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