International Surgery Journal
Obuhovich AR et al. Int Surg J. 2026 Aug;13(8):1483-1487
https://www.ijsurgery.com
pISSN 2349-3305 | eISSN 2349-2902

DOI: https://dx.doi.org/10.18203/2349-2902.isj20262475

Case Report

When benign mimics malignancy: giant hypervascular focal nodular hyperplasia requiring left hemi-hepatectomy

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Received: 07 June 2026
Accepted: 10 July 2026

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ABSTRACT

Focal nodular hyperplasia of the liver (FNHL) is a benign, non-invasive, and common liver cell tumor with a habitually lazy course of action. The probability of FNHL transforming into malignancy is very unlikely. The prevalence of FNHL globally is reported as 0.4-3% in the general population and the risk of acquiring FNHL increases with age. A 29-year-old woman presented to the hepatobiliary department of the Grodno University Clinic with complaints of intermittent epigastric pain, early satiety and abdominal bloating. No associated fever, jaundice, weight loss, or gastrointestinal bleeding was present. The patient's medical history was not significant, and she had no family history of cancer. A large, well-circumscribed heterogeneous lesion occupying the left lobe of the liver was demonstrated on the abdominal ultrasound scan. Contrast enhanced computed tomography (CT) revealed a giant hypervascular mass with intense arterial phase enhancement and partial washout during the portal venous phase. Furthermore, magnetic resonance imaging further confirmed a hypervascular lesion arising predominantly from segments II, III, and IV of the liver. This case report illustrates the diagnostic challenges aroused by giant hypervascular focal nodular hyperplasia (FNH), a benign liver lesion that can precisely replicate the characteristic features of malignant tumors, both clinically and radiologically.

Keywords: Focal nodular hyperplasia, Benign, Liver, Left hemi-hepatectomy, Hypervascular

INTRODUCTION

Focal nodular hyperplasia in liver (FNHL) is a benign, non-invasive and normal liver cell tumor with a habitually lazy course of action. The probability of FNHL transforming into malignancy is very unlikely.[1] The prevalence of FNHL globally is reported as 0.4-3% in the general population and the risk of acquiring FNHL increases with age.[2] FNHL represents extremely low percentages of rupture and hemorrhage, and it occurs as a result for previous arterial malformations around the area of the liver. Due to the current widespread use of imaging techniques FNHL can be identified occasionally as an accidental finding. The atypical patterns in radiology of FNHL may cause diagnostic uncertainty in the pathophysiology of the condition because it might be a result of hormonal milieu. It is noticed that in around 80% cases of FNHL there is a single lesion which is not encapsulated with a clearly defined boarder. Additionally, in macroscopical examination a large central fibrovascular zone can be detected. This stellate fibrovascular zone is famously known as the "fibrous scar" or "central scar".[2]

FNHL is more predominant in females than in males in the proportion of 8:1. Due to these data, there is a susceptibility to the influence of estrogen in the occurrence of FNHL.[4] Furthermore, the incidence is more common in women of reproductive age (20-50 years). Even though symptomatic cases of FNHL are rare, it is crucial to pay close attention to the diagnosis process because it can be portrayed as a malignant condition in the aspect of clinical presentation.[3]

While most benign FNHL cases are undiagnosed, only one-third of cases are clinically diagnosed with the help of positive symptoms in the abdominal region. The most frequent symptoms in patients with benign FNHL are pain in the left hypochondriac region, abdominal mass, and liver enlargement. Ironically, two-thirds of benign FNHL cases go unnoticed because of the lack of clinical manifestations. Even though there is zero potential of FNHL transforming into malignant form there are certain shared clinical presentations which mimics malignancy such as upper abdominal pain, abdominal swelling and hepatomegaly.[5] Except for these there are specific symptoms for malignancy such as loss of appetite, losing weight without trying, jaundice, and white chalky stools.

Benign tumors should be differentiated from hypervascular malignant liver cancers, such as hepatocellular carcinoma, and other malignant hypervascular conditions, such as melanoma, renal cell carcinoma, and neuroendocrine tumors, using radiological techniques. Lesions that develop in arteries are mostly benign, such as FNHL, adenoma, and small hemangioma, which absorb contrast very quickly. Enhancement patterns in the arterial phase looks similar in both the conditions. The actual differentiation is done in the following phases and by additional gross pathologic features with clinical findings.[6]

In conclusion, this case report illustrates the diagnostic challenges encountered by giant hypervascular focal nodular hyperplasia (FNH), a benign liver lesion that can precisely mimic the characteristic features of malignant tumors, both clinically and radiologically.

CASE REPORT

A 29-year-old woman presented to the hepatobiliary department of the Grodno University Clinic with complaints of intermittent epigastric pain, early satiety, and abdominal bloating. No associated fever, jaundice, weight loss, or gastrointestinal bleeding was present. The patient's medical history was not significant, and she had no family history of cancer.

Tumor markers such as AFP, CEA, and CA 19-9 were not elevated (Table 1). Laboratory investigations, such as complete blood count (CBC), liver function tests, coagulation profile, and renal function tests, were all within normal limits (Table 1). A palpable mass was observed beneath the left costal margin. Clinical examination revealed mild epigastric and left upper quadrant tenderness without any signs of peritoneal irritation or guarding.

Diagnostic workup

Abdominal ultrasound revealed a large, well-circumscribed, heterogeneous lesion in the left lobe of the liver. Contrast enhanced CT revealed a giant hypervascular mass with intense arterial phase enhancement and partial washout during the portal venous phase (Figure 1). Central stellate scarring was suspected in some regions of the lesion. However, the radiological appearance remained atypical. Magnetic resonance imaging further confirmed a hypervascular lesion arising predominantly from segments II, III, and IV of the liver (Figure 2).

Table 1: CBC, biochemical parameters, coagulation profile and tumor markers.

CBC, biochemical parameters and coagulation profile		Reference range
White blood cells/×10^9/l	6.21	4-9
Red blood cells/×10^{12}/l	4.48	3.7-4.9
Hemoglobin/g/l	131	120-160
Platelets/×10^9/l	365	150-450
Hematocrit (%)	38.7	32-47
ESR/mm/hour	8	2-15
C-reactive protein/mg/dl	5	0-6
Creatinine/µmol/l	80	53-97
Urea/mmol/l	3.9	53-97
Aspartate aminotransferase/U/l	23	5-37
Alanine aminotransferase/U/l	16	5-42
Prothrombin time/s	13.4	10-15
Prothrombin activity/%	69.12	60-130
INR	1.17	0.9-1.3
APTT/APTT/s	25.3s	22-38
APTT/APTT ratio	0.8	0-1
Fibrinogen concentration/g/l	4.81	2-4
Tumor markers		
AFP/ng/ml	<0.5	10-20
CEA/ng/ml	1.3	<3.0
CA 19-9/U/ml	29. 1	0-37

Surgical resection was recommended for definitive diagnosis and treatment because of diagnostic uncertainty. Due to the vascularity and heterogeneous enhancement, hepatocellular carcinoma or focal nodular hyperplasia was suspected.

Focal nodular hyperplasia mass

Figures 1 and 2 represent the CT and MRI images of focal nodular hyperplasia in the left lobe of the liver.

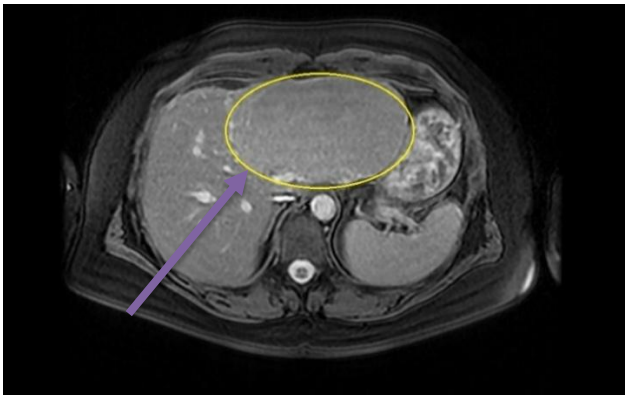

Figure 1: This CT scan shows the focal nodular hyperplasia in the left lobe of the liver.

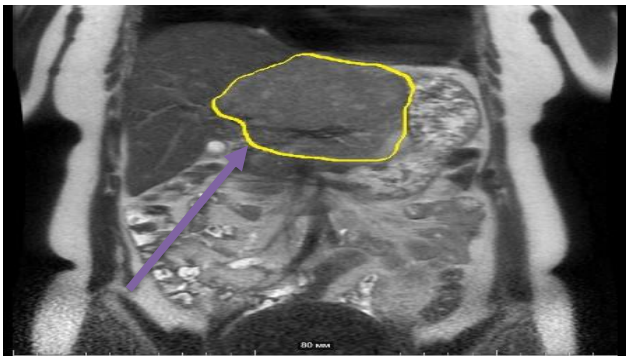

Figure 2: This MRI-scan shows the focal nodular hyperplasia in the left lobe of the liver

Surgical management

Following a comprehensive assessment, elective left hemi-hepatectomy with cholecystectomy was planned. Preoperative planning was done by assessing hepatic vascular anatomy and residual liver volume. Informed consent was obtained prior to surgery. The surgery was performed under general endotracheal anesthesia with standard perioperative antibiotic prophylaxis. The steps were as follows.

The operative field was prepared and draped appropriately. An upper midline laparotomy was performed.

Intraoperative exploration revealed a large, highly vascular mass arising from the left hepatic lobe, compressing adjacent structures. No evidence of extrahepatic invasion or peritoneal dissemination. The liver parenchyma outside the lesion appeared normal.

After mobilization of the left lobe cholecystectomy was performed. The common bile duct and right and left hepatic ducts were isolated.

The left hepatic duct was ligated twice and transected respectively. The left hepatic artery and left branch of the portal vein were isolated, ligated and transected.

Careful hemostasis was maintained throughout the procedure because of the tumor's marked vascularity.

Liver parenchymal transection was performed along Cantlie's line using "LigaSure" 5 mm and bipolar coagulation.

The left and middle hepatic veins were isolated, ligated and transected. Hemostasis was satisfactory, a hemostatic sponge was applied to the resection surface, and a subdiaphragmatic drain was inserted.

The abdominal wall was closed in a single layer. The procedure was completed without any complications.

Postoperative course

The description of the histopathological specimen of mass in the left lobe of the liver taken during USG control biopsy (a brown-gray, thread-like tissue column) revealed liver tissue with focal fatty degeneration of hepatocytes, proliferation of bile ducts, and interportal leukocyte infiltration. Moreover, the morphological findings of this specimen were consistent with focal nodular hyperplasia.

Furthermore, the macroscopic description of resected gallbladder and FNH of the left lobe of the liver was 6×2.8×2.5 cm specimen with a bluish-purple serous membrane comprising of velvety mucosa (wall 2-3 mm thick) and 11×8.5×7 cm specimen with a gray-brown colored consisting of homogeneity of tissues respectively. Thus, the conclusion of this macroscopic specimen was chronic cholecystitis and focal nodular hyperplasia supported by focal cholestasis leading to focal fibrosis of the gallbladder and disseminated large droplet fatty degeneration of hepatocytes respectively.

This patient remained hemodynamically stable, with preserved liver function and no evidence of bile leakage or bleeding. No evidence of dysplasia or malignancy was identified. Furthermore, this patient presented with the complication of abscess together with a serous hemorrhagic discharge in the surgical site following a day after the Hemi-hepatectomy shown in Figure 3. The initial treatment protocol consisted of 0.4 ml Enoxaparin (for prophylaxis of deep vein thrombosis and pulmonary embolism), 10 mg/ml for 1 ml (1 ampoule) Morphine, 20 mg of Omeprazole, infusion therapy to control the

hemodynamics, diuresis of this patient and drainage of the surgical site. Moreover 8 days after the surgery, the patient complained about soreness of the surgical site, rise of temperature to 37.5°C, elevation of CRP to 162 mg/ml and mild leukocytosis (with a WBC of 13×10^9/l) was noticed; therefore, 1200 mg IV drip of Amoxiclav bid was administered. In addition, during an ultrasound scan of the abdominal cavity, fluid was detected. This fluid was later aspirated under the guidance of ultrasound. Also, hepatoprotection was taken into account for this treatment protocol to improve prognosis of the patient, the combination of ademetionine (HEP 500 mg) and 100 ml solution of NaCl was administered to this patient.

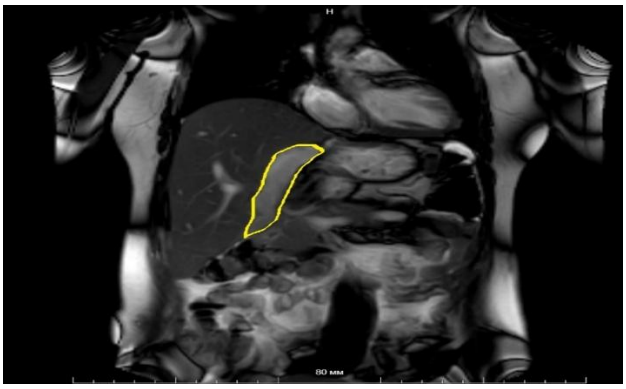

Figure 3: MRI-scan shows the abscess in surgical site.

Abscess in the surgical site

On follow-up, this patient showed resolution of symptoms and satisfactory postoperative recovery without recurrence, dysplasia, or malignancy. However, further follow-ups were planned in the time frame of 3 months, 6 months and 12 months following the surgery to assess the overall prognosis of this patient.

DISCUSSION

The diagnosis of giant hypervascular FNH poses significant challenges, as illustrated in this case. The lesion's size and vascular features usually mimic malignant hepatic tumors such as hepatocellular carcinoma (HCC) or hepatic adenoma, worsening the preoperative categorization.[2,7] Imaging techniques including CT and MRI showed a large, hypervascular mass with atypical features, increasing suspicion for malignancy.[8]

In spite of comprehensive radiological and cytological evaluation, definitive diagnosis remained undefined until histopathological examination post-biopsy and surgery verified FNH.[9] This diagnostic uncertainty highlights the limitations of non-invasive methods in differentiating the benign from malignant hepatic lesions when lesions are large and hypervascular in nature.[10,11]

However, unlike smaller or typical FNH lesions, giant FNH may require major resections, such as left hemi-hepatectomy, for diagnostic confirmation and symptom relief over time. In addition, many cases often report initial misdiagnosis or diagnostic uncertainty, resulting in aggressive surgical interventions.[12,13] Compared with similar case reports in the literature, our case highlights the challenges in treating giant FNH. This case mirrors with reports highlighting the importance of multidisciplinary evaluation and cautious analysis of imaging and biopsy results to prevent overtreatment while ensuring patient safety at all times.[14-16]

The combination of clinical, radiological, and pathological data is important for improving patient outcomes. When imaging and biopsy results remain inconclusive, surgical intervention remains a viable option, particularly when the lesion size or symptoms influence the patient's quality of life. Patient management should stabilize the risks of major hepatic resection in the precancerous state. Clinically, this case underscores the need for increased awareness of giant FNH as a benign imitator of malignancy. Patient management should stabilize the risks of major hepatic resection against the precancerous state. When imaging and biopsy remain inconclusive, surgical intervention remains viable option, particularly when the lesion size or symptoms influence patient quality of life.

The successful outcome following left hemi-hepatectomy demonstrates that, regardless of the benign nature of FNH, vast amounts of surgeries may be required in select cases due to lesion size, location, and the diagnostic uncertainty presented with the lesion.

Reliance solely on imaging and cytology may be inadequate, requiring surgical biopsy or resection for a definitive diagnosis. Furthermore, the key lessons from this case are that giant hypervascular FNH can closely resemble malignant liver tumors, which can delay the timely diagnosis.

However, timely surgical management is considered when malignancy cannot be excluded. Additionally, awareness of the benign nature but atypical presentation of giant FNH can help in decision-making, potentially preventing unnecessary extensive resections in some patients. Clinicians should consider a broad differential diagnosis when encountering large hypervascular liver lesions and adopt a stepwise diagnostic approach involving advanced imaging, biopsy, and multidisciplinary consultation.

Accumulating case series and long-term follow-up data will assist in creating evidence-based guidelines for the treatment of giant hypervascular FNH, elevating patient care and proper resource utilization to improve the prognosis of these patients. Moreover, the development of molecular or imaging biomarkers may decrease the need for invasive procedures. Future studies should focus on improving non-invasive diagnostic criteria and imaging techniques to better differentiate giant FNH from malignant hepatic tumors.

CONCLUSION

This case report illustrates the diagnostic challenges associated with giant hypervascular FNH, a benign liver lesion that can precisely replicate the characteristic features of malignant tumors, both clinically and radiologically. Thus, successful management of the patient via a left hemi-hepatectomy provided a definitive diagnosis and subsequent resolution of the patient's symptoms, highlighting the role of surgical intervention when conservative therapy remained inconclusive. Furthermore, with the given potential for diagnostic uncertainty and the risk of overtreatment, a multidisciplinary strategy should be implemented by combining advanced imaging, biopsy, and careful clinical assessment.

Recommendations

Greater awareness and research are necessary to improve non-invasive diagnostics and develop biomarkers to better distinguish giant FNH from malignancy, elevating patient outcomes and management.

ACKNOWLEDGEMENTS

The authors would like to thank the medical staff at the surgical department of the Grodno University Clinic, Belarus, for their helpful contributions to the diagnosis and management of this patient. They would also like to express their gratitude to the patient and her family for their collaboration and trust in sharing this case to enhance awareness about atypical presentation of giant hypervascular focal nodular hyperplasia managed surgically by left hemi-hepatectomy in the Grodno Region.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Obuhovich AR, Amry MAM, Direcksze NDKN, Dineshkumar PA, Hasnaa MAF. When benign mimics malignancy: giant hypervascular focal nodular hyperplasia requiring left hemi-hepatectomy. Int Surg J 2026;13:1483-7.