Hepatocellular adenomas (HCAs) are monoclonal, hepatocellular neoplasms that are typically described in young women on oral contraceptives (OCs). Because HCAs have an increased proclivity to hemorrhage and to rarely undergo malignant transformation, they warrant either a periodic imaging surveillance or surgical resection. Until recently, the pathogenesis of HCAs was poorly understood. Recent developments in pathology and genetics have contributed to better understanding of the distinctive oncological pathways involved in HCAs. It has now been shown that HCAs possess unique genetic signatures that are distinct from normal hepatocytes and hepatocellular carcinomas (HCCs). Hepatocellular adenomas are now classified into 4 subtypes, including a miscellaneous category. The 3 distinct subtypes of HCAs include hepatocyte nuclear factor-1α (HNF-1α)-mutated HCAs (HNF-HCAs), HCAs characterized by β-catenin mutations, and inflammatory HCAs (I-HCAs; due to mutations involving interleukin-6 [IL-6] signal transducer). Inflammatory HCAs and HNF-HCAs are the 2 major subgroups of HCAs, which together constitute for up to 80% of all adenomas. The characteristic genetic abnormalities, epidemiologic features, histomorphology, clinicobiologic behavior, and imaging findings of these 3 subtypes of HCAs are summarized in Table 1 and discussed in detail in the article.

**Epidemiology, Pathology, and Clinical Manifestations**

Hepatocellular adenoma is the second most common benign hepatocellular neoplasm after focal nodular hyperplasia. The exact incidence and prevalence of HCA is difficult to predict because of the introduction of low-dose OCs and increased rate of incidental detection at least in part due to the widespread use of multiphase computed tomography (CT) or magnetic resonance imaging (MRI). Sex hormones, particularly estrogens, seem to play a major role in the pathogenesis of HCAs. Hepatocellular adenomas occur predominantly in young adult women (with a male-to-female ratio of 1.8–10), with a mean age of 41 years, and are rarely seen in adult men and children. Hepatocellular adenomas have been reported to grow remarkably during pregnancy, sometimes with catastrophic consequences from rupture or hemorrhage. Medical literature has shown a strong association between long-term OC use and the development of HCA. Although early data suggested a definitive correlation between the duration of OC pill use and risk of HCAs (relative risk ranging from 1.3 for 1–3 years of OC use to 25 for >11 years of OC use), more recent data on low-estrogen OC pills have demonstrated an overall relative risk of 1.25 to 2.8. Overall, 85% to 95% of patients with HCAs have history of OC pill use of more than 2 years. Hepatocellular adenomas may show partial or complete regression after cessation of OC pills. There is an increased risk of development of HCAs in patients with anabolic androgen steroid intake, familial adenomatosis polyposis and metabolic liver diseases such as glycogen storage diseases (types Ia, III, VI), tyrosinemia, galactosemia, steatohepatitis, and hemochromatosis.

Pathologically, most HCAs are solitary, unencapsulated tumors that vary from less than 1 cm to up to 20 cm in size. Multiple HCAs may occur in adrenocortical origin, a condition defined by the presence of more than 10 adenomas in a normal liver (discussed later). Histologically, HCAs are characterized by layers of mildly thickened or irregular liver cell cords and plates separated by sinusoids. In contradistinction to HCCs, the hepatocytes in HCAs lack cytologic and nuclear atypia. Typically, HCAs do not have portal tract elements including the portal venules or bile ductules. Clinically, up to 50% of patients with HCAs may present with a wide variety of symptoms of which right upper quadrant discomfort is the most common (up to 43%). Palpable mass and severe abdominal pain (secondary to rupture or hemorrhage) are uncommonly encountered. Nearly 60% of patients who are symptomatic have signs of intratumoral or peritumoral hemorrhage.

Inflammatory HCAs

Inflammatory HCAs, which form the largest subgroup of HCAs, comprise 40% to 55% of all HCAs. These tumors are predominantly seen in women, in association with obesity, alcohol use, and hepatic steatosis. More than 90% of women with I-HCAs do not have portal tract elements including the portal venules or bile ductules. Clinically, patients with I-HCAs may manifest a

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**Table 1 and discussed in detail in the article.**

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TABLE 1. Salient Epidemiological, Clinical, Pathological, and Imaging Features of 3 Major Subtypes of HCA

<table>
<thead>
<tr>
<th>Epidemiology</th>
<th>Inflammatory HCA (I-HCA)</th>
<th>HCA With HNF-1α Gene Mutation</th>
<th>HCA With β-Catenin Activation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical features</td>
<td>40%-55% of HCA; majority are women; rarely seen in men.</td>
<td>35%-50% of HCA; almost exclusively in women.</td>
<td>10%-18% of HCA. Affects men and women.</td>
</tr>
<tr>
<td>Pathological features</td>
<td>Increased risk of bleeding and small risk of malignant transformation.</td>
<td>May bleed; no risk of malignant transformation.</td>
<td></td>
</tr>
<tr>
<td>Imaging features</td>
<td>Marked sinusoidal dilatation/peliosis.</td>
<td>No inflammatory infiltrates or significant peliosis.</td>
<td></td>
</tr>
</tbody>
</table>

“systemic inflammatory syndrome” including fever, leukocytosis, and elevated serum C-reactive protein (CRP) levels. Inflammatory HCA carry a definite increased risk of bleeding (up to 30%) and a small risk of malignant transformation (5%-9%).

Inflammatory HCA comprise a prototype example of tumors induced by hepatobiliary inflammation. More than 40% of around 285 genes overexpressed in I-HCA are associated with inflammation and immune response. Interleukin-6 is a proinflammatory cytokine that regulates acute phase inflammatory response and liver regeneration through activation of the JAK-STAT (Janus kinase- signal transducers and activators of transcription) pathway. Sustained activation of IL-6 receptor signaling due to somatic mutations of IL-6 signal transducer gene (IL-6ST gene), which encodes glycoprotein-130 (gp-130), is one of the major pathogenetic mechanisms proposed in the development of I-HCA. The resultant overactivation of acute phase inflammatory response results in overexpression of markers such as serum amyloid A and CRP in neoplastic hepatocytes and, in some cases, in serum. Serum levels of γ-glutamyl transferase may also be elevated in these patients. Somatic, activating mutations of IL-6ST gene are seen in up to 60% of I-HCAs. The remainder of the I-HCAs show STAT3 activation independent of gp-130 activation. Interestingly enough, activation of IL-6–STAT3 pathway is also described in development of HCCs especially when combined with inactivation of the transforming growth factor-β signaling pathway. Around 10% of I-HCAs may also show mutations involving β-catenin gene.

Histologically, I-HCAs are characterized by marked sinusoidal dilatation, polymorphous inflammatory infiltrates, peliosis, and thickened tortuous arteries. The presence of prominent ductular reaction is a distinct histologic feature. Steatosis within I-HCA is less extensive (compared with HNF-HCA) and variable. Historically, HCA with these histologic features were misclassified as “telangiectatic focal nodular hyperplasias (FNHs).” Polymorphous inflammatory infiltrate seen in I-HCAs likely results from a significant increase in CCL20, a chemokine that attracts a wide spectrum of immune cells.

On imaging, I-HCAs manifest as hypervascular hepatic masses with persistent enhancement in the portal venous and delayed phases. Inflammatory HCA are markedly hyperintense on T2-weighted images corresponding to areas of sinusoidal dilatation (Fig. 1). Marked T2 hyperintensity and persistent delayed enhancement have been shown to have a sensitivity of up to 85.2% and specificity of up to 87.5% for detection and characterization of I-HCAs. Focal areas of microscopic fat may be seen in a small subset of patients (11%). On contrast-enhanced ultrasound (CEUS), I-HCAs show arterial hypervascularity with centripetal filling and peripheral rim of sustained enhancement with delayed central washout. These findings have a diagnostic significance given that other benign hepatic lesions rarely exhibit such a pattern.

**HNF-1α MUTATED HCAs**

Hepatocyte nuclear factor-1α–mutated HCAs (HNF-HCAs) account for 35% to 50% of all HCA and arise because of bi-allelic inactivation of transcription factor 1 gene located in chromosome 12. Hepatocyte nuclear factor-1α–mutated HCAs...
are exclusively seen in women; more than 90% patients have history of OC use.\textsuperscript{13} Hepatocyte nuclear factor-1α-mutated HCA may be multiple in up to 50% of cases.\textsuperscript{6}

Hepatocyte nuclear factor-1α mutations may be somatic (up to 90% of cases) or germline (5%–10% of cases) in origin. Germline mutations of HNF-1α gene result in maturity-onset diabetes of the young- type 3 (MODY-3), a type of diabetes mellitus syndrome that is also characterized by familial adenomatosis.\textsuperscript{7,31,32} Transcription factor 1 encodes the tumor suppressor gene, hepatocyte nuclear factor-1α (HNF-1α). Hepatocyte nuclear factor-1α mutation promotes lipogenesis by suppression of gluconeogenesis, activation of glycolysis, and promotion of fatty acid biosynthesis.\textsuperscript{33} The down-regulation of fatty acid binding protein-1 leads to “faulty” transport of fatty acids and to intracellular deposition of fat. Therefore, HNF-HCAs are characterized by diffuse intralesional steatosis. The putative role of OCs in the induction and progression of HCAs is partly explained by genotoxic effects of estrogen, which result in HNF-1α mutations. Alternatively, HNF-1α mutation may be the primary inciting event that results in the accumulation of estrogen metabolites that unconditionally stimulate hepatocyte proliferation.\textsuperscript{34,35}

On multidetector computed tomography (MDCT), HNF-HCAs may appear as liver masses with either macroscopic fat or low-density masses on unenhanced scans because of microscopic fat. Steatosis within HCAs is better demonstrated on MRI than on CT scans because of superior tissue differentiation on MRI. In contrast to a high (35%–77% of adenomas) albeit variable detection of fat within HCAs on MRIs, fat deposition was seen in only 7% of 44 adenomas studied by CT scanning.\textsuperscript{36–38} On MRI, HNF-HCAs manifest as heterogeneous lesions with areas of T1 high signal intensity that may correspond to the presence of fat, glycogen or, less commonly, hemorrhage. The characteristic finding of HNF-HCAs is that of diffuse intralesional steatosis that is classically demonstrated as diffuse signal dropout on out-of-phase T1-weighted gradient echo imaging (Fig. 2). The sensitivity, specificity, positive predictive value, and negative predictive value of diffuse signal drop on T1 out-of-phase chemical shift...
imaging for predicting the HNF-HCA is 86.7%, 100%, 100%, and 94.7%, respectively. On T2-weighted MR images, HNF-HCAs appear as homogenous masses that may be hypointense or hyperintense to the surrounding liver. On contrast-enhanced CT or MRI, moderate contrast enhancement is seen in the arterial phase, which does not persist on to the portal venous and delayed phases. On CEUS, HNF-HCAs are homogeneously hyperechoic on grayscale imaging (likely because of diffuse steatosis) with mild to moderate arterial hypervascularity and isoechoic appearance to the liver on the portal venous phase.

**β-CATENIN MUTATED HCAs**

A small subset of HCAs (up to 10%-18%) originates from sustained activation of β-catenin because of mutations involving the CTNNBI gene (catenin, beta-1). These tumors primarily affect patients with glycogen storage disease and on androgen treatment and have a greater propensity than the other subtypes of HCAs to undergo malignant transformation to HCCs. β-Catenin plays a major role in hepatocyte development, differentiation, zonation, proliferation, and regeneration. Activation of β-catenin in normal hepatocytes is usually transient and is regulated by its rapid degradation. Adenomatous polyposis coli (APC) and Axin family genes in concert with glycogen synthase kinase-1β forms the cytoplasmic destruction complex responsible for prompt degradation of β-catenin. Excessive nuclear accumulation and sustained activation of β-catenin may result from mutations in β-catenin itself (with the mutant β-catenin resisting the degradation) or from mutations involving the cytoplasmic degradation complex. Biallelic mutations of the APC gene seen in familial polyposis syndrome therefore predispose an individual for the development of HCAs. Excessive β-catenin activity results in autonomous growth of hepatocytes and accelerates HCA formation. Hepatocellular adenomas with β-catenin activation show cytologic abnormalities and pseudoglandular formation on histology. Aberrant activation of β-catenin is seen in HCCs and hepatoblastomas as well. Although β-catenin mutations are implicated in malignant transformation of HCAs, only 20% to 30% of malignant HCAs show β-catenin mutations suggesting alternate pathways of malignant transformation of HCAs.

Glycogen storage disease is a well-described independent risk factor for the development of HCAs and HCCs on account of background chronic inflammation. Up to 75% of patients with glycogen storage disease (IA, II, IV, and VI) may develop HCAs. Several chromosomal and genetic alterations have been described in HCAs in glycogen storage disease, which include gain of chromosome 6q involving multiple oncogenes and loss of chromosome 6p, involving multiple tumor suppressor genes like IGF2R (insulin-like growth factor 2 receptor) and LATS1 (large tumor suppressor, homolog 1). These chromosomal/genetic changes are also frequently described in HCCs and preneoplastic dysplastic nodules, thereby explaining the higher risk of malignant transformation associated with HCAs in patients with glycogen storage disease. Given the rarity of glycogen storage disease, the exact incidence of malignant transformation of HCA is, however, difficult to predict in this context. Hepatocellular adenomas with β-catenin gene mutations are less frequently encountered in glycogen storage disease (4/14 cases reported so far in the literature).

Patients with glycogen storage disease manifest with diffusely increased attenuation of the liver on MDCT (Fig. 3). Hepatocellular adenomas due to β-catenin mutations may appear as homogeneous or heterogeneous hypervascular masses and lack intratumoral fat (Fig. 4). Intense arterial enhancement is seen, which may or may not persist into the delayed phase. Frankly malignant, non-inflammatory type of HCAs with β-catenin mutations (up to 10%-15% of all HCAs) simulates HCCs on imaging.

**MISCELLANEOUS HCAs (UNCLASSIFIED)**

The miscellaneous group of HCAs include the remainder of HCAs that have none of the distinct genetic alternations described above. These HCAs are poorly understood; detailed studies are required to evaluate specific pathogenetic and clinical characteristics of this disparate entity.

**HEPATIC ADENOMATOSIS**

Hepatic adenomatosis (HA) is defined as the presence of more than 10 adenomas in an otherwise normal liver. First described by Flejou et al in 1985, patients with glycogen storage disease are typically excluded from this definition. Hepatic adenomatosis may be characterized by the development of either a steatotic or an inflammatory type of HCAs and shows a definite female preponderance (Figs. 5 and 6). The role of OCS in the etiopathogenesis of HA is, however, still unclear. Hepatic adenomatosis was initially thought to be an independent risk factor.

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**FIGURE 3.** Hepatocellular adenoma with malignant change in a 39-year-old man with glycogen storage disease. A, Axial unenhanced CT image of the liver showing a large heterogeneous lesion in the right lobe of the liver (white arrow). Note the mild diffuse hyperattenuation of the background liver. Multiple additional steatotic as well as nonsteatotic focal lesions were seen (not shown). Percutaneous ultrasound-guided biopsy of the largest lesion revealed HCC in the background of glycogen storage disease. B and C, Images from digital subtraction angiograms of the right hepatic artery injection before (B) and after (C) chemoembolization show intense vascular tumor blush in the right hepatic lobe on the pre-embolization image (arrows in B), which disappears after successful embolization (C).
for complications like hemorrhage or malignant transformation with a reported risk of bleeding of up to 63%. However, a more recent analysis has shown that the rates of intrahepatic bleeding and intratumoral bleeding in asymptomatic incidentally discovered HA are 2% and 13%, respectively; rates that are similar to solitary HCAs. Recent studies thus suggest that the potential risk of complications is related to the underlying histologic subtype and tumor size rather than the number of HCAs.

HCAs: DIFFERENTIAL DIAGNOSES AND SUBTYPE DIFFERENTIATION

The major imaging differential diagnoses of HCAs include FNH and HCC. Focal nodular hyperplasia, a polyclonal tumor-like lesion that occurs predominantly in young women, commonly manifests as a lobulated, homogenous, solid liver mass that shows intense arterial phase enhancement and becomes iso-attenuating to the liver on portal venous phase imaging. The presence of a central scar is a useful sign, which can be readily detected in FNH on MRI in up to 80% of large FNHs (>3 cm). In contrast to FNHs, HCAs manifest as heterogeneous masses with variable presence of fat, hemorrhage, or necrosis. Magnetic resonance imaging is the current criterion standard imaging technique in noninvasively differentiating FNH from HCA, especially with the advent of hepatobiliary-specific contrast agents like gadoxetic acid (EOVIST; Bayer Schering Pharma, Germany). Unlike HCAs that are hypointense in the hepatobiliary phase (20 minutes after injection), most FNHs remain isointense or become hyperintense on delayed phase imaging after administration of gadoxetic acid (Fig. 7). Gadobenate dimeglumine enhanced MRI has also been found to be highly accurate with sensitivity of up to 96.9% and specificity of up to 100% for differentiation of HCA and FNH. In contradistinction to HCAs, HCCs develop against the background of liver cirrhosis or chronic hepatitis. Hepatocellular carcinomas may have variable imaging manifestations; they commonly appear as heterogeneously enhancing solid masses that show arterial phase enhancement and washout during portal venous phase. Hepatocellular carcinomas may have variable proportion of fat, hemorrhage, and necrosis and may at times be indistinguishable from HCAs. The presence of infiltrative growth pattern, portal or hepatic venous thromboses, lymph node, and/or distant metastases helps discriminate HCCs from HCAs. Accurate differentiation may warrant biopsy and histopathologic evaluation.

Traditionally, HCAs were considered as lesions with widely pleomorphic imaging appearance. Hepatocellular adenomas were usually described as heterogeneous, hypervascular masses with areas of fat, hemorrhage, and necrosis on multiphase CT or MRI. Recent findings suggest that 2 major subtypes of HCAs, namely I-HCA and HNF-HCAs, can be reliably differentiated...
from one another on MRI (described above under specific sub-types) thereby significantly affecting patient management. Immunohistochemistry is also a reliable tool to differentiate various subtypes of HCAs. Lack of staining for liver fatty acid binding protein is 100% sensitive and specific for HNF-1α-mutated HCA. Strong diffuse overexpression of glutamine synthetase (which is encoded by the β-catenin gene) and nuclear β-catenin staining is highly specific (100% specificity) and sensitive (85% sensitivity) for the detection of β-catenin-activated HCAs and hepatocellular expression of serum amyloid A2 correlates closely with inflammatory adenomas (91% sensitivity and specificity).25

MANAGEMENT OF HCAs: CURRENT UPDATE

Current taxonomical schemata and genotype-phenotype correlation have resulted in a paradigm shift in the management of HCAs. Management guidelines are now based on the histologic subtype and size rather than the number of HCAs. It has been shown that HCAs more than 5 cm tend to bleed, and tumors more than 8 cm tend to undergo malignant change.6 Risk of malignancy within HCAs is also higher in patients with glycogen storage disease and in men (up to 50% of HCAs in men).7,33 Hepatocyte nuclear factor-1α–mutated HCAs do not carry any significant risk of malignant transformation; the risk of bleeding in HNF-HCA was 9% versus 16% in I-HCAs according to a study.6,18,42 Hemorrhage associated with HCAs may be intratumoral or may result in subcapsular hematoma and/or hemoperitoneum (Fig. 8). Hemodynamic instability is rarely associated with ruptured HCAs, and selective hepatic artery embolization is sufficient to restore hemodynamic stability and to reduce tumor size as well as operative risk during surgery.55-57 Embolization may also obviate the need for aggressive surgical resection, which is associated with an increased morbidity.6,58

Hepatocellular adenomas less than 5 cm in size are rarely associated with risk of hemorrhage or malignant transformation and hence can safely be managed conservatively with serial imaging follow-up.1,42 Stopping the offending drugs (OC pills, androgens, barbiturates) and dietary modification as in glycogen storage disease are the recommended initial steps that may help by halting HCA growth and reducing the tendency to bleed. Most small HCAs remain stable during surveillance, and a small number of them may disappear.1 Currently, there are no clear-cut guidelines for the optimal interval and duration of imaging surveillance in HCAs. Yearly surveillance with multiphase CT or MRI seems to be the most appropriate strategy, which should probably be continued until menopause.4,59 Currently, surgical resection is recommended for HCAs more than 5 cm, HCAs that do not regress after stopping the offending drugs, HCAs with malignant change or evidence of β-catenin activation as demonstrated on biopsy, and all HCAs in men.60 Rates of tumor recurrence are found to be very low after surgical resection.6,18 Alternative approaches for the treatment of HCAs include transarterial embolization and radiofrequency ablation. Radiofrequency ablation has been found to be safe and effective in achieving complete tumor ablation in a recently published series of 10 patients with HCAs.61 A recent analysis showed that, for small HCAs that continue to grow despite stopping the offending drugs, radiofrequency ablation may be the most cost-effective option, in comparison to surgery, transarterial embolization, and watchful waiting.62

Image-guided biopsy with immunohistochemical and cytogenetic evaluation has been proposed to enable identification of patients “at risk” for malignant transformation. Biopsy may be performed for small I-HCAs that continue to grow despite

FIGURE 7. Role of gadoxetic acid–enhanced MRI in the differentiation of focal nodular hyperplasia from HCA. A, Axial gadoxetic acid–enhanced MR image in the hepatobiliary phase (20 minutes after injection) in a 35-year-old woman revealing a focal hepatic lesion that is isointense to slightly hyperintense to the hepatic parenchyma (white arrow) consistent with focal nodular hyperplasia. A central scar is also seen. B, Axial gadoxetic acid–enhanced MR image in the hepatobiliary phase (20 minutes after injection) in a 32-year-old woman revealing multiple focal hepatic lesions throughout the liver, which are hypointense to the hepatic parenchyma (white arrows). Histopathology confirmed the diagnosis of HCA.

FIGURE 8. Ruptured HCA in a 42-year-old woman presenting with acute right upper quadrant pain. Axial contrast-enhanced image of the hepatic arterial phase reveals a large right hepatic mass with focal disruption along the right anterolateral aspect, intralesional and perihepatic hematoma (arrowheads), and hemoperitoneum (arrows). Note residual enhancement of the lesion along its medial aspect.
Widespread application of the guidelines on percutaneous biopsy of HCAs is still controversial because of several factors including lack of sophisticated techniques in pathology and cytogenetics in most hospitals around the world. Other potential problems with biopsy include sampling errors, the risk of hemorrhage or tumor seeding along the needle-track, and the occasional difficulty in differentiating HCA from normal hepatocytes as well as well-differentiated HCCs on histopathologic examination of limited core biopsy samples.

Management of HCAs in women of child-bearing age is more complicated given the increased risk of growth and bleeding during pregnancy. Adenomas that bleed or rupture during pregnancy are frequently found to be large (>6.5 cm). Major catastrophic events because of HCAs in pregnancy comes from case reports from 1970s to 1990s, which may be attributable to the delay in clinical and imaging diagnoses. Overall, up to 59% of large HCAs may rupture with resultant maternal and fetal mortality in the range of 30% to 40%. Although pregnancy is not contraindicated in patients with HCAs, given the risk of growth and subsequent increased risk of hemorrhage during pregnancy, HCAs in young women should be managed more aggressively. Some authors recommend early intervention even for small HCAs (<5 cm) in young women who wish to become pregnant, although others reserve it for HCAs larger than 5 cm or HCAs, which continue to grow despite stopping the offending drugs.

Management of HA should be similar to solitary HCAs with aggressive approach (surgery or embolization) for symptomatic lesions or asymptomatic lesions larger than 5 cm and “watchful waiting” for smaller lesions. Withdrawal of exogenous steroid intake has been found to decrease the risk of bleeding but does not affect the lesion size or growth. Liver transplantation is no more indicated as the primary treatment strategy for all patients with HA and is reserved for cases with progressive liver failure or malignant transformation.

CONCLUSIONS

Recent insights into distinctive cytogenetic abnormalities and genotype-phenotype correlation in HCAs have brought paradigm shifts in our understanding of the imaging findings with significant implications for management. Adenomas with specific genetic abnormalities demonstrate characteristic clinicobiologic behavior, natural history, response to treatment, and prognosis. Although I-HCAs frequently undergo hemorrhage, HCAs with β-catenin mutations show increased predisposition to transformation to HCCs. Dynamic, multiphase MDCT and MRI play a key role in the diagnosis, characterization, and staging of HCAs as well as in surveillance after management with one of many therapeutic options.

REFERENCES


