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The Hepatic adenomatosis and steroid-induced liver tumors – a modern clinical challenge in gynecology and sports medicine: a narrative review

Damian Stencelewski1, Łukasz Wójcik2, Aleksandra Gorzynska-Schulz3, Natalia Gorzynska4, Michał Falis1, Alicja Nierychlewska2, Agata Bronisławska2, Robert Bronisławski2, Alicja Solecka1, Lidia Kiełbratowska4

Damian Stencelewski[1] (D.S.)

e-mail: damian.sten777@gmail.com

ORCID: https://orcid.org/0009-0002-7359-5562

Łukasz Wójcik[2] (Ł.W.)

E-mail lukaszjaw571@gmail.com

ORCID: https://orcid.org/0009-0003-1516-3478

Aleksandra Gorzynska-Schulz[3] (A.G.S.)

e-mail: amjgorzynska@gmail.com

ORCID https://orcid.org/0009-0005-1719-8548

Natalia Gorzynska[4] (N.G).

e-mail: nataliagorzynska05@gmail.com

ORCID https://orcid.org/0009-0007-3891-9636

Michał Falis[1] (M.F.)

e-mail: michaljakubfalis@gmail.com

ORCID: https://orcid.org/0009-0002-1074-6036

Alicja Nierychlewska[2] (A.N.)

e-mail: alicjanierychlewska99@gmail.com

ORCID: https://orcid.org/0009-0009-6783-4601

Agata Bronisławska[2] (A.B.)

e-mail: agata.blach2@gmail.com

ORCID: https://orcid.org/0009-0003-3833-0638

Robert Bronisławski[2] (R.B.)

e-mail: robertbronislawski@gmail.com

ORCID: https://orcid.org/0009-0004-6494-4307

Alicja Solecka[1] (A.S.)

e-mail: alicjasolecka222@gmail.com

ORCID: https://orcid.org/0009-0001-0688-309X

Lidia Kiełbratowska[4] (L.K.)

e-mail: Lidkie03@gmail.com

ORCID https://orcid.org/0009-0000-6322-7446

Affiliation:

1 University Clinical Centre, Gdańsk, Poland

2 Nicolaus Copernicus Hospital, Copernicus PL Sp. z o.o., Gdańsk, Poland

3 Medical University of Gdańsk, Poland

4 Pomeranian Medical University, Szczecin, Poland

Corresponding author:

Damian Stencelewski

damian.sten777@gmail.com

ABSTRACT

Background

Hepatocellular adenoma (HCA) is a rare benign liver tumor strongly associated with hormonal exposure, particularly estrogen-containing oral contraceptives and anabolic-androgenic steroids. Although benign, HCA is clinically significant because of the risks of hemorrhage and malignant transformation into hepatocellular carcinoma (HCC), especially in high-risk molecular subtypes.

Aim

This narrative review summarizes current evidence on the molecular classification, pathogenesis, diagnosis, and management of hepatocellular adenoma and hepatic adenomatosis.

Material and Methods

A literature review was conducted using the PubMed and Scopus databases. Studies on epidemiology, molecular mechanisms, diagnostic imaging, and therapeutic strategies were reviewed.

Results

Hormonal exposure promotes hepatocellular proliferation through dysregulated estrogen and androgen signaling. β-catenin–activated adenomas carry the highest risk of malignant transformation, while inflammatory and HNF1A-inactivated subtypes show distinct molecular and imaging features. Hepatic adenomatosis is associated with an increased risk of complications. Magnetic resonance imaging is the diagnostic modality of choice, whereas biopsy is reserved for selected cases because of hemorrhagic risk. Management includes withdrawal of hormonal triggers and, when indicated, surgical resection or embolization.

Conclusions

HCA is a molecularly heterogeneous, hormonally driven liver tumor requiring risk-adapted management. Early identification of high-risk lesions is essential to prevent hemorrhage and malignant transformation.

Keywords: Hepatocellular adenoma; Hepatic adenomatosis; Hormonal exposure; Liver tumor imaging; signaling pathway ;

1. Introduction

HCA is a relatively rare, benign liver tumor of epithelial origin that predominantly develops in han otherwise normal hepatic parenchyma[1]. Historically considered an isolated clinical finding, the clinical landscape of this disease fundamentally shifted with the recognition of hepatic adenomatosis. First described as a distinct clinical entity by Alexandre et al. in 1984, hepatic adenomatosis is classically defined by the presence of ten or more adenomas scattered throughout the liver tissue. While HCA is not malignant by definitione[1,2,3], it represents a critical topic in contemporary gastroenterology, hepatology, and sports medicine due to its high propensity for life-threatening complications, including spontaneous intra-tumoral or intra-abdominal hemorrhage and malignant transformation into HCC[2,4].

The clinical significance of HCA and hepatic adenomatosis (table 1) has increased exponentially in recent years, driven by a rising incidence correlated with widespread exogenous hormone exposure[3,5,6]. The historical cornerstone of HCA epidemiology dates back to the 1970s, following the introduction of first-generation oral contraceptive pills (OCPs). Early landmark epidemiological studies established a definitive, dose-dependent causal relationship between high-dose ethinylestradiol formulations and the proliferation of benign hepatocyte tumors in young women[5,7].

However, the modern era has introduced a profound paradigm shift in the risk factor profile of this disease[8]. While OCP use remains a classic trigger, the rising prevalence of metabolic syndrome, obesity, and, most notably, the widespread abuse of anabolic-androgenic steroids (AAS) among amateur and professional bodybuilders has altered the demographic landscape of hepatic adenomatosis. Today, what was once considered a disease almost exclusive to young females is increasingly diagnosed in young men presenting with highly aggressive molecular subtypes[9,10,11]. This narrative review aims to comprehensively synthesize current knowledge on the pathophysiology, diagnostic challenges, and evolving management strategies for hormone-associated hepatic adenomatosis in the era of expanding OCP and AAS exposure (table 1) [10,12].

Feature	**Hepatocellular adenoma (HCA)**	**Hepatic adenomatosis**
Definition	Single benign hepatocellular tumor	≥10 adenomas in the liver
Typical population	Young women (historically)	Broader, increasing male incidence
Major risk factors	Oral contraceptives, estrogens	OCP, AAS, metabolic syndrome
Key molecular subtypes	HNF1A-inactivated, inflammatory, β-catenin	Same spectrum, often multiple lesions

Feature	Hepatocellular adenoma (HCA)	Hepatic adenomatosis
Malignant potential	Low–moderate (β-catenin subtype highest risk)	Higher overall risk due to multiplicity
Major complication	Hemorrhage, rare malignant transformation	Higher bleeding + management complexity
Imaging of choice	MRI with contrast	MRI with contrast
Biopsy role	Generally avoided	Generally avoided

Table 1. Key features of hepatocellular adenoma and hepatic adenomatosis

2. Epidemiology and Risk Factors

2.1 Sex-Based Discrepancies and Demographic Shifts

HCA has exhibited one of the most striking sex-based discrepancies in oncology, with an annual incidence of approximately 3 to 4 per 100,000 in women using oral contraceptives, compared to only 0.1 per 100,000 in men. In the classic female-dominated cohort, HCAs typically present as solitary lesions[2,4,6].

However, the epidemiological landscape of hepatic adenomatosis (multiple lesions) reveals a different trend[9,11]. When multiple adenomas develop, the stark female-to-male ratio narrows, reflecting a systemic, metabolic, or exogenous hormonal drive rather than an isolated, sporadic event. Moreover, the absolute number of male patients diagnosed with HCA has risen sharply over the last two decades. This demographic shift is directly tied to the diversification of modern risk factors.[12,13]

2.2 Primary Risk Factors: Estrogens, Androgens, and Metabolic Status

Oral Contraceptive Pills (OCPs)

The administration of exogenous estrogens remains the most extensively documented trigger for HCA development in females. The risk is strictly dose- and duration-dependent. Long-term use of OCPs (exceeding 5 years) multiplies the risk of adenoma formation up to 25-fold compared to non-users. Ethinylestradiol stimulates hepatocyte proliferation directly through estrogen receptors, promoting cellular hyperplasia and vascular alterations within the hepatic parenchyma[13,14,15].

Anabolic-Androgenic Steroids (AAS)

While OCPs drive the female cohort[3,8], AAS abuse is the primary engine behind the emerging male cohort of HCA and hepatic adenomatosis. This phenomenon is no longer restricted to

professional athletes; it has become a widespread public health issue due to illicit image- and performance-enhancing drug use among amateur bodybuilders. Synthetic androgens—particularly 17alpha-alkylated steroids (such as oxymetholone or stanozolol)—undergo intensive hepatic metabolism, exerting profound hepatotoxic and mitogenic effects. AAS-induced adenomas are notoriously aggressive, frequently presenting as multiple tumors (adenomatosis) with a significantly higher baseline risk of life-threatening rupture or malignant transformation compared to OCP-linked variants[3,5,11,12,14,16].

Metabolic Syndrome, Obesity, and MAFLD

With the global rise in body mass index (BMI), metabolic health has emerged as a major independent driver of HCA. Obesity, insulin resistance, systemic low-grade inflammation, and Metabolic Dysfunction-Associated Steatohepatitis (MASH—formerly NASH) stimulate hepatocyte growth through altered adipokine signaling and elevated insulin-like growth factors. This metabolic milieu uniquely predisposes patients to the inflammatory sub-type of HCA (ih-HCA) and is a frequent driver of multi-centric adenomatosis in both sexes[1,8,11].

Glycogen Storage Diseases (GSD)

Though rare from a public health perspective, GSDs (most notably Type Ia and Ib) represent a highly critical academic and clinical model for hepatic adenomatosis. Up to 70-80% of adult GSD Type I patients develop multiple hepatic adenomas due to chronic hypoglycemia, elevated glucagon-to-insulin ratios, and altered intracellular carbohydrate metabolism. GSD-associated adenomas typically present early in life and carry an exceptionally high risk of transformation into HCC[13,14].

Why Adenomatosis? Systemic vs. Focal Initiation

Understanding why some patients develop a single, solitary HCA while others present with diffuse hepatic adenomatosis requires a shift from local genetic concepts to systemic pathophysiological models.

Single HCA (Sporadic)	**Hepatic Adenomatosis (Systemic)**
Driven by a localized somatic mutation	Driven by a continuous, systemic stimulus
Initiated by a single clones of hepatocytes	Multicentric initiation across the whole parenchyma
Often static after hormone withdrawal	Higher risk of recurrence and progression

Table 2. **Comparative Features of Sporadic HCA vs. Hepatic Adenomatosis**

In sporadic cases (table 2), a localized genetic mishap occurs within a single hepatocyte clone. Conversely, hepatic adenomatosis is the product of a widespread background fields defect[4,9,14,15]**.** The entire hepatic parenchyma is continuously bathed in elevated levels of circulating hormones (either OCs or AAS) or subjected to persistent metabolic stress (hyperinsulinemia, glycogen accumulation). Because there is no single, localized initiating event, multiple independent clusters of hepatocytes cross the threshold into autonomous proliferation simultaneously. This systemic background explains why adenomatosis is inherently multicentric, harder to manage surgically, and highly prone to recurrence if the underlying hormonal or metabolic driver is not completely eliminated.[10,13]

3. Pathogenesis and Molecular Classification

3.1 Molecular Subtypes of Hepatocellular Adenoma

The management of HCA and hepatic adenomatosis has been revolutionized by the transition from purely histological descriptions to a robust molecular classification[15,17]. Based on specific genetic alterations, HCAs are categorized into four distinct major subgroups, each exhibiting unique clinical behaviors and varying risks of malignant transformation[1,4,15,18].

$HNF1A$-Inactivated HCA (H-HCA)

This subtype accounts for approximately 35–40% of all HCA cases and is characterized by bi-allelic inactivating mutations of the *HNF1A* gene, which encodes hepatocyte nuclear factor. This mutation leads to a complete down-regulation of liver fatty acid-binding protein (LFABP)[13,17]. H-HCA is strongly associated with oral contraceptive use in women, typically presents with marked intratumoral steatosis, and carries a negligible, near-zero risk of malignant transformation[3,10,16].

Inflammatory HCA (ih-HCA)

Representing roughly 40–50% of cases, ih-HCA is the most prevalent subtype. It is driven by activating mutations in genes involved in the IL-$6/JAK/STAT$ signaling pathway (most commonly *IL6ST*, *STAT3*, or *JAK1*), leading to chronic, autonomous inflammatory signaling and overexpression of acute-phase proteins like serum amyloid A (SAA) and C-reactive protein (CRP). This subtype is highly correlated with obesity, alcohol consumption, and metabolic syndrome. Notably, it has the highest propensity for spontaneous, life-threatening intratumoral hemorrhage[1,2,4,17,19].

β-Catenin Activated HCA (β-HCA)

Accounting for 10–15% of adenomas, this is the most oncologically critical subtype. It is driven by activating mutations in the *CTNNB1* gene (encoding β-catenin), which leads to the constitutional activation of the Wnt/β-$catenin$ signaling pathway. Histologically, it is identified by the diffuse nuclear and cytoplasmic accumulation of β-catenin and the overexpression of its target, glutamine synthetase (GS). β-HCA is strongly associated with male sex, glycogen storage diseases, and, most importantly, the use of AAS[10].

3.2 Clinical and Oncological Significance: The Malignant Shift

The primary clinical utility of this molecular breakdown lies in its power to stratify the risk of transformation into HCC.

While $H\text{-}HCA$ and the majority of $ih\text{-}HCA$ lesions follow a benign clinical course, the $\beta\text{-}HCA$ **subtype is a borderline malignant entity**. The constitutive activation of the $Wnt/\beta\text{-}catenin$ pathway disrupts cell-cell adhesion, promotes metabolic remodeling, and drives rapid, uncontrolled hepatocyte proliferation. Consequently, up to 40–50% of $\beta\text{-}HCA$ lesions show signs of high-grade dysplasia or frank malignant conversion to HCC[14,15,18]. Because AAS use preferentially drives this specific pathway, any male patient presenting with steroid-associated hepatic adenomatosis must be treated as a high-risk oncological case, regardless of the tumor's size.

3.3 Molecular Mechanisms: Estrogens vs. AAS

The disparate clinical behaviors of female (OCP-driven) and male (AAS-driven) adenomatosis are rooted in the distinct intracellular pathways activated by these exogenous hormones (Figure 1).

Estrogens: Cellular Hyperplasia and Vascular Engineering

Oral contraceptives drive hepatocyte growth primarily via Estrogen Receptor alpha ($ER\alpha$). Estrogens act as promoters of cellular hyperplasia rather than true mutagenic initiators. They upregulate local growth factors (such as $VEGF$ and $TGF\text{-}\alpha$), leading to the creation of thin-walled, poorly supported, dilated sinusoidal blood vessels. This "vascular engineering" explains why OCP-driven tumors, particularly when exposed to additional inflammatory triggers ($ih\text{-}HCA$), are highly prone to rupture and bleeding, but rarely progress to malignancy once the hormonal stimulus is withdrawn[3,20].

Anabolic-Androgenic Steroids: Mitogenic Signaling and β-Catenin Hijacking

AAS interact directly with the cytoplasmic Androgen Receptor (AR) in hepatocytes, but their pathogenetic footprint is far more aggressive. 17-alpha-alkylated steroids bypass normal hepatic degradation, leading to profound intracellular stress[21.

At the molecular level, AAS abuse actively hijacks the $Wnt/\beta\text{-}catenin$ signaling cascade. Androgen receptor signaling cross-talks with β-catenin, stabilizing the protein and preventing its destruction complex from degrading it. This mimics a genetic mutation, forcing β-catenin into the nucleus where it binds to T-cell factor/lymphoid enhancer factor (TCF/LEF) transcription factors. This results in the relentless transcription of oncogenes such as *c-Myc* and *Cyclin D1[14,17,20]* Therefore, while estrogens gently push hepatocytes toward hyperplasia, AAS act as potent mitogens that systematically destabilize genomic integrity, directly predisposing the liver parenchyma to multicentric $\beta\text{-}HCA$ and rapid malignant progression[7,18].

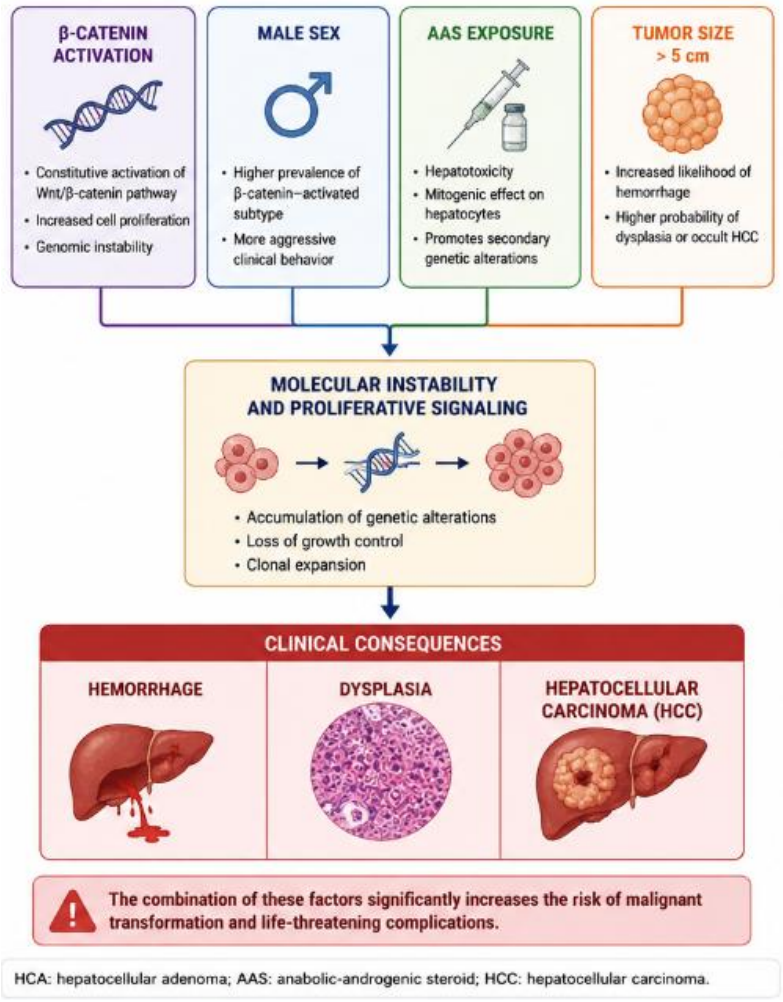


Figure 1. Risk stratification model of malignant transformation in HCA

4. Hepatic Adenomatosis as a Distinct Clinical Entity

The clinical and pathological characterization of hepatic adenomatosis remains a debated topic in modern hepatology. First defined by Alexandre et al. in 1984, hepatic adenomatosis is

diagnosed when ten or more synchronous hepatocellular adenomas are identified within a non-cirrhotic liver. In contrast to solitary hepatocellular adenoma, hepatic adenomatosis represents a multifocal disease with distinct clinical and therapeutic implications.

From a clinical perspective, hepatic adenomatosis is associated with a higher cumulative risk of complications, particularly spontaneous intra-tumoral or intra-abdominal hemorrhage. Although the risk of individual lesions is largely size-dependent, multiplicity increases the overall probability of clinically significant events. In addition, diffuse liver involvement limits the feasibility of curative surgical approaches, as extensive resection carries a substantial risk of post-hepatectomy liver failure. Consequently, surgical strategies are typically restricted to selected lesions with high-risk features, while the remaining nodules are managed conservatively[7,19].

The nosological classification of hepatic adenomatosis remains controversial [4,13]. The spectrum hypothesis proposes that lesion multiplicity reflects prolonged exposure to external stimuli such as exogenous hormones, including oral contraceptives or anabolic-androgenic steroids. In contrast, emerging evidence supports a distinct entity model, suggesting that affected patients may have an underlying predisposition, potentially related to genetic, metabolic, or vascular factors that promote a liver-wide susceptibility to adenoma formation[5,6,10].

For clinical practice, this distinction is less important than its therapeutic implication. Given the multifocal nature of the disease, management should focus on elimination of hormonal triggers, selective treatment of high-risk lesions, and lifelong radiological surveillance of the entire liver[20].

5. Clinical Presentation and Complications

Hepatocellular adenoma and hepatic adenomatosis are frequently asymptomatic, with many cases discovered incidentally during routine abdominal imaging. When symptoms do occur, the most common presentation is a dull, chronic pain or a feeling of fullness in the right upper quadrant, typically caused by the stretching of Glisson's capsule due to tumor growth or minor intratumoral bleeding[2,7,10].

The most critical and dramatic clinical manifestation is the acute rupture of an adenoma, leading to a life-threatening hemoperitoneum. This emergency presents with sudden, excruciating abdominal pain, rapidly followed by signs of hypovolemic shock, such as tachycardia, severe hypotension, and cold, clammy skin. Inflammatory adenomas ($ih\text{-}HCA$) and tumors larger than 5 cm carry the highest baseline risk for this catastrophic event [21].

Beyond acute hemorrhage, the primary long-term complication is the malignant transformation into HCC. This risk is exceptionally high in men using AAS due to the strong association with the oncogenic β-catenin activated subtype ($\beta\text{-}HCA$). Conversely, tumors driven by oral

contraceptives in women carry a much lower risk of malignancy and frequently exhibit significant regression following hormone withdrawal[20,22].

6. Diagnostics and Differential Diagnosis

6.1 Imaging Modalities

Ultrasonography (USG) is the first-line screening tool for hepatic masses but is non-specific for HCA, often showing hyperechoic or heterogeneous lesions that require further evaluation. Contrast-enhanced CT is useful in acute settings, particularly for detecting rupture and hemoperitoneum, but has limited ability to subtype HCA[3,4,10,11].

MRI with hepatobiliary-specific contrast agents (e.g., Gd-EOB-DTPA) is the gold standard for characterization and subtyping. It can identify typical features such as intracellular fat in H"-" HCA (signal drop on out-of-phase imaging) and T2 hyperintensity in inflammatory (ih"-" HCA). The β- HCA subtype often has a non-specific appearance and may mimic malignancy(15,20,23].

6.2 Differential Diagnosis

A key challenge is distinguishing HCA from focal nodular hyperplasia (FNH). On hepatobiliary-phase MRI, FNH retains contrast and appears hyperintense due to preserved hepatocytes, whereas HCA is typically hypointense due to absent biliary function[10,21].

Differentiation from HCC is also crucial, especially in patients using anabolic-androgenic steroids. HCC typically shows arterial phase enhancement with venous washout, although β"-" HCA can sometimes mimic this pattern. Other considerations include hepatic adenomatosis, hypervascular metastases, and hemangiomas, which classically show peripheral nodular enhancement with progressive fill-in [1,2,4].

6.3 Liver Biopsy

Liver biopsy is generally avoided due to the risk of bleeding and potential tumor seeding, particularly in hypervascular lesions. It is reserved for cases where MRI is inconclusive and histological or molecular subtype identification will alter management, such as suspected β"-" HCA in small lesions or when differentiating HCA from well-differentiated HCC. If required, biopsy should be performed under imaging guidance, targeting the least [4,14,20].

7. The Role of Exogenous Hormones and Anabolic Compounds

7.1 Oral Contraceptives (OCPs): The Historical Blueprint

The OCPs and HCA is characterized by a dose- and duration-dependent relationship[2,5,15,17]. Early high-dose formulations containing ethinylestradiol (>50 μg) were associated with a markedly increased risk of adenoma development, particularly with prolonged use exceeding five years. Estrogen exposure promotes hepatocyte proliferation and localized hyperplastic changes[4,15,16].

In modern gynecological practice, the incidence of OCP-associated HCA has significantly decreased due to the widespread use of low-dose estrogen formulations (<30 μg) and progestin-only contraceptives, which exert a reduced proliferative effect on hepatic tissue (figure 2).

7.2 Anabolic-Androgenic Steroid (AAS)s: Emerging Risk Factor

AAS abuse is an increasingly recognized risk factor for hepatocellular adenoma and hepatic adenomatosis, particularly in the context of recreational bodybuilding. Orally active 17α-alkylated androgens are associated with hepatotoxic effects and may contribute to hepatocyte proliferation and genetic instability[14,15].

Clinical observations suggest that AAS-associated adenomas are more frequently multifocal and may exhibit an increased association with β-catenin activation, a molecular subtype linked to higher malignant potential. These lesions may demonstrate a more aggressive clinical course, including a higher risk of hemorrhage and malignant transformation into HCC[1,7].

7.3 Reversibility of Lesions Upon Cessation

The clinical course following cessation of exogenous hormonal exposure differs between etiologies. In patients with OCP-associated adenomas, lesion regression or stabilization is frequently observed within 6–12 months after discontinuation, supporting a conservative management strategy in selected cases[24].

In contrast, regression of AAS-associated adenomas is less predictable. While partial tumor regression may occur following steroid withdrawal, molecular alterations—particularly β-catenin pathway activation—may persist, potentially contributing to continued growth or delayed complications. Therefore, these patients require individualized risk assessment and long-term imaging surveillance[7,9,22].

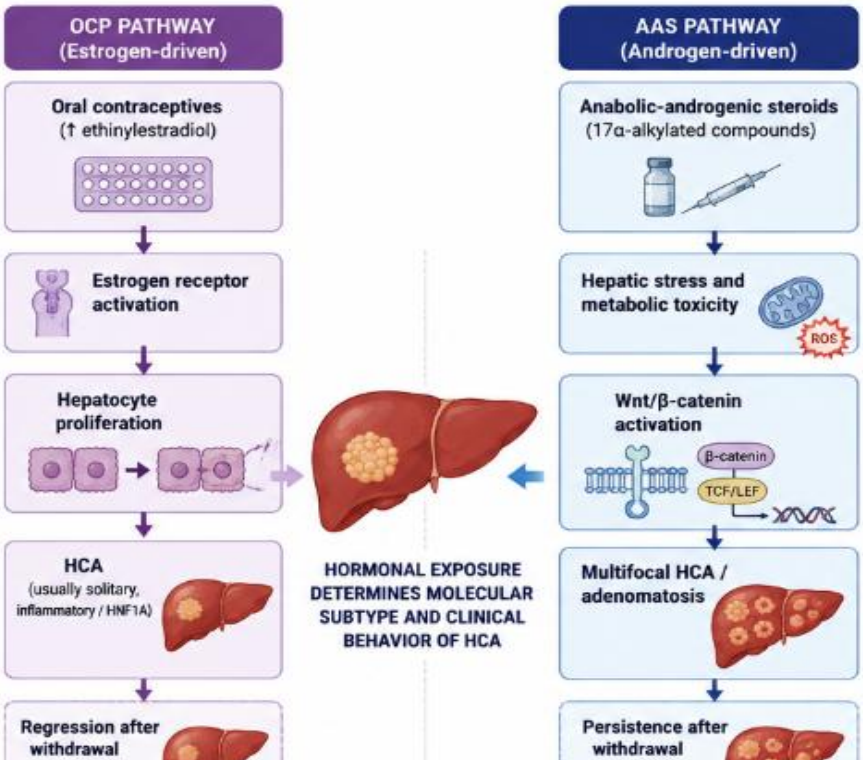


Figure 2. Hormone-driven pathogenesis of hepatocellular adenoma

8. Management and Therapeutic Strategies

8.1 Conservative Management

The initial step in the management of hormone-associated hepatic adenomatosis is cessation of the offending exogenous compound, including OCPs or AAS. Lifestyle modifications targeting metabolic risk factors, such as weight reduction and glycemic control, may contribute to improvement of systemic inflammatory status[9,19,23].

In female patients with OCP-associated adenomas, conservative management is often associated with lesion regression. Serial monitoring with contrast-enhanced MRI at 6–12 month intervals is recommended. In asymptomatic patients with lesions <5 cm, a watchful waiting strategy may be considered under close imaging surveillance[20,24,25].

8.2 Interventional and Surgical Treatment

Surgical resection, preferably via minimally invasive laparoscopic techniques, is indicated in cases of large lesions (>5 cm), persistent growth after hormone withdrawal, or high-risk imaging features[20,21].

Due to the increased prevalence of β-catenin–activated subtypes, male patients are generally considered at higher risk of malignant transformation, and therefore a lower threshold for interventional management is recommended[6,13,17].

In acute hemorrhage with hemoperitoneum, transarterial embolization (TAE) is the first-line therapeutic option, providing effective hemostasis and allowing for delayed elective surgical planning. For selected cases, thermal ablation techniques such as radiofrequency ablation (RFA) or microwave ablation (MWA) may serve as parenchyma-sparing alternatives to resection[3,18].

8.3 Liver Transplantation

Orthotopic liver transplantation (OLT) represents a rare treatment option reserved for patients with diffuse hepatic adenomatosis not amenable to resection[1,11,24].

Indications include progressive disease despite complete hormonal withdrawal, recurrent hemorrhagic complications refractory to embolization, or suspicion of malignant transformation in non-resectable, multifocal disease[18].

9. Risk of Malignant Transformation

The risk of malignant transformation into hepatocellular carcinoma (HCC) represents the most clinically relevant long-term concern in hepatic adenomatosis[1,4]. This risk is heterogeneous and strongly influenced by molecular subtype and patient-specific factors. The β-catenin–activated HCA subtype is consistently associated with the highest oncological risk, driven by activation of the Wnt/β-catenin signaling pathway and increased cellular proliferation. Male sex is recognized as an independent risk factor for malignant transformation, with a significantly higher incidence of HCC compared with female patients. This association is largely explained by the higher prevalence of β-catenin–activated lesions in men[1,5]. In this context, clinical management often favors early intervention rather than prolonged surveillance in male patients.Exposure to AAS has been associated with an increased risk of adenoma formation and may contribute to more aggressive clinical behavior, potentially through hormonal stimulation of hepatocyte proliferation and secondary genetic alterations[5,8,17,22]. Tumor size remains an important prognostic factor, with lesions larger than 5 cm demonstrating a higher likelihood of hemorrhage and a greater probability of harboring areas of dysplasia or occult malignancy (table 3)[2,14,19,22,24,25].

Risk factor	**Mechanism / association**
β-catenin–activated HCA	Activation of Wnt/β-catenin signaling pathway leading to increased cellular proliferation and genomic instability
Male sex	Higher prevalence of β-catenin–activated subtypes and more aggressive clinical phenotype
Anabolic-androgenic steroid (AAS) use	Hormonal stimulation of hepatocyte proliferation and promotion of secondary genetic alterations
Tumor size >5 cm	Increased likelihood of hemorrhage and presence of dysplastic or malignant foci

Risk factor	Mechanism / association
Hepatic adenomatosis	Multiple lesions contributing to cumulative tumor burden and increased complication risk
Oral contraceptive exposure	Estrogen-mediated hepatocyte proliferation (dose-dependent, partially reversible after withdrawal)

Table 3. Risk Factors and Molecular Mechanisms of Malignant Transformation in HCA.

10. Conclusions

Hepatocellular adenoma and hepatic adenomatosis represent classic models of hormone-dependent liver disease. The strong association between exogenous hormone exposure and hepatocyte proliferation highlights the importance of detailed clinical history-taking in diagnosis. Oral contraceptive-associated adenomas remain a well-characterized and largely reversible condition, for which hormone withdrawal constitutes the first-line therapeutic intervention. The increasing prevalence of AAS use has shifted the clinical profile toward more complex and potentially higher-risk disease in male patients, with an increased risk of hemorrhage and malignant transformation. Contrast-enhanced MRI with hepatobiliary-specific contrast agents is the diagnostic modality of choice, enabling non-invasive lesion characterization and risk stratification, particularly for β-catenin–activated subtypes, while avoiding biopsy-related hemorrhagic complications. Management of hepatic adenomatosis requires individualized, risk-adapted strategies integrating clinical, hormonal, metabolic, and molecular factors. Optimal care involves cessation of exogenous hormones, selective use of surgical or interventional procedures, and long-term imaging surveillance within a multidisciplinary framework.

Authors' Contributions:

Conceptualization: **D.S.**

Formal analysis: **D.S Ł.W. A.G.S. N.G. M.F. A.N. A.B. R.B. A.S. L.K.**

Investigation: **D.S Ł.W. A.G.S. N.G**

Writing – original draft: **D.S Ł.W. A.G.S. N.G. M.F**

Writing – review and editing: **D.S Ł.W. A.G.S. N.G. M.F.**

Visualization: **D.S Ł.W. A.G.S. N.G. M.F. A.N. A.B. R.B. A.S. L.K.**

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