Journal of Hepatology and Gastrointestinal Disorders

OPEN ACCESS Freely available online

Commentary

Hormonal Factors and its Mechanism of Action of Hepatocellular Adenoma

Hans Vlierberghe*

Department of Hepatology, Ghent University Hospital, Ghent, Belgium

ABOUT THE STUDY

Hepatocytes are the primary functioning cells of the liver, and they give rise to Hepatocellular Adenoma (HCA), a benign liver tumor. These tumors can range in size from a few millimeters to several centimeters, and they are usually solitary and well-circumscribed. HCAs are usually benign, although they can develop, rupture, or change into malignant tissue, especially in some subtypes linked to particular risk factors. Hepatocellular adenomas are thought to affect 3-4 people per million people annually, making them a very uncommon condition. Women of reproductive age are more likely to be diagnosed with them, especially if they use oral contraceptives or Hormone Replacement Therapy (HRT). Although cases have been observed in both men and women who use oral contraceptives, the occurrence has been linked to their widespread use.

Mechanism of action

Hepatocytes, the main functioning cells of the liver, experience complicated interactions between hormonal changes and genetic alterations that lead to the development of Hepatocellular Adenomas (HCAs). Usually, these adenomas are monoclonal, originating from a single hepatocyte that has experienced particular genetic modifications. Hepatocyte Nuclear Factor 1 Alpha (*HNF1A*) inactivating mutations and activating mutations in the β-catenin-encoding *CTNNB1* gene are two important genetic mutations linked to the pathophysiology of HCA.

The lack of *HNF1A* function in *HNF1A*-mutant HCAs leads to unchecked cell proliferation by upsetting normal hepatocyte differentiation and metabolic homeostasis. Conversely, mutations in *CTNNB1* cause the Wnt/β-catenin signalling pathway to be constitutively activated. This pathway normally controls cell division and growth, but mutations lead to an accumulation of β-catenin in the nucleus, which promotes abnormal gene expression and increases cell division.

Hormonal factors are also important, especially the oestrogen and androgen receptors that are present on hepatocytes. For example, oestrogen is known to promote hepatocyte proliferation, which could account for the higher prevalence of HCAs in women who use hormone replacement therapy or oral contraceptives and are of reproductive age.

The development and course of hepatocellular adenomas are influenced by these genetic and hormonal variables, which also affect the tumors growth patterns, clinical features, and propensity for problems including bleeding or malignant transformation.

HNF1A-mutated hepatocellular adenomas

Hepatocyte Nuclear Factor 1 Alpha (*HNF1A*) mutations that inactivate are the hallmark of *HNF1A*-mutated Hepatocellular Adenomas (HCAs). *HNF1A* is an essential gene that controls liver development and metabolism. Young women are primarily affected by these adenomas, and many of them use hormone replacement treatment or oral contraceptives. Histologically, *HNF1A*-mutant HCAs can show variable degrees of inflammation and cytological atypia in addition to prominent steatosis, or the deposition of fat within hepatocytes. Clinically speaking, adenomas with *HNF1A* mutations are typically thought to have a lower probability of malignant transformation than adenomas activated by β-catenin.

β-catenin-activated HCAs

Hepatocellular Adenomas (HCAs) that are activated by β-catenin are identified by mutations in the *CTNNB1* gene, which results in the disruption of the Wnt/β-catenin signalling system. In the regulation of gene transcription and cell adhesion, β-catenin is an essential component. β-catenin builds up in the nucleus of HCAs harbouring *CTNNB1* mutations, causing abnormal gene expression and unchecked cell division. Histologically, β-catenin-activated HCAs frequently show nuclear atypia, a trabecular growth pattern, and enhanced cellularity. Compared to other HCA subtypes, these adenomas have a higher risk of malignant development, which calls for cautious therapy and monitoring. Because surgical excision can lead to complications like bleeding and the development of Hepatocellular Carcinoma (HCC), it is frequently advised.

Correspondence to: Hans Vlierberghe, Department of Hepatology, Ghent University Hospital, Ghent, Belgium, E-mail: vlierhans83@yahoo.com

Received: 22-Apr-2024, Manuscript No. JHGD-24-32927; **Editor assigned:** 25-Apr-2024, PreQC No. JHGD-24-32927 (PQ); **Reviewed:** 09-May-2024, QC No. JHGD-24-32927; **Revised:** 16-May-2024, Manuscript No. JHGD-24-32927 (R); **Published:** 23-May-2024, DOI: 10.35248/2475-3181.24.10.309

Citation: Vlierberghe H (2024) Hormonal Factors and its Mechanism of Action of Hepatocellular Adenoma. J Hepatol Gastroint Dis. 10:309.

Inflammatory adenomas

As a subtype of Hepatic Adenomas (HCAs), inflammatory adenomas are distinguished by their correlation with obesity and the metabolic syndrome, as well as by their frequent manifestation of sinusoidal dilatation and inflammation. Histologically, these adenomas usually exhibit inflammatory infiltrates made up of lymphocytes and neutrophils as well as varied degrees of steatosis, or fat build up. In addition, they might exhibit sinusoidal dilatation, which on imaging can resemble hepatic vascular malformations.

Clinically, inflammatory adenomas are important. They carry a higher risk of complications such as hemorrhage and malignant transformation compared to other HCA subtypes. Management involves careful monitoring with imaging studies to assess growth and risk of complications. Surgical resection may be considered for symptomatic or enlarging adenomas, especially in cases where there is concern for potential malignant transformation. Management also includes addressing underlying metabolic conditions, such as obesity and insulin resistance, to mitigate the risk of adenoma recurrence and progression.