Session 2. Mimickers of Hepatic Malignancy: What to Do?

Outcomes and management strategy for hepatic adenoma

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Curriculum Vitae

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Main Activities

Liver and pancreas surgery

Laparoscopic liver and pancreas surgery

Liver, small bowel and multiorgan transplantation

Outcomes and management strategy for hepatic adenoma

Hepatocellular adenoma (HCA) is a rare benign hepatocytic liver tumor observed mainly in young women and risk factors including not only oral contraception (OC) but also more frequently obesity including those with metabolic syndrome and steatohepatitis. HCA is rare in men (10-15%) and in this case, underlying liver disease or risk factors should be searched (anabolic steroid, glycogenosis, vascular anomaly, etc). HCA is usually unique but multiple forms are more frequently observed (50%). HCA is at risk of bleeding and malignancy. HCA is no more one radiological and histological entity of surgical indication whatever the size but many advances have been made in all fields since 2003. On imaging 3 subtypes are described (telangiectatic, steatotic and classic) and 4 subtypes on histology (inflammatory expressing inflammation markers, HNF 1 α mutated, β catenin mutated and unclassified). These subtypes are at different risk of complications and subtyping can be determined easily on MRI but in some cases, liver biopsy can be needed for accurate histological subtyping and management. Occasionally more than one subtype can be encountered in the same patient. Risk factors for complications are now well established according to clinical, radiological and histological data. Bleeding (20-25%) is the most serious complication and observed mainly in large (>5 cm) and telangiectatic HCA. Malignant transformation is low in female (4%), high in male (50%) and risk factors are male gender, size > 8 cm, classic subtype and β catenin mutated. In all cases and once the diagnosis is made, management includes withdrawal of OC for some months (long term withdrawal is not obligatory) and weight loss is more and more advocated. In men HCA should be resected whatever the size related to the risk of malignancy. In women HCA > 5 cm should be treated and those <5 cm can be observed with a very low risk of complication, 15-20% risk of slight progression and 10-15% risk of regression and therefore no more justification for liver transplantation for multiple HCA. Liver transplantation can be discussed in patients with HCA and glycogenesis, in men with multiple HCA and in patients with underlying vascular anomalies (like porto-caval shunt) and risk liver resection. Although resection was the standard of treatment, ablation and embolization were described especially for HCA sized around 5 cm. Patients with bleeding HCA are stable or rapidly stabilized and therefore they can be treated by embolization and delayed resection and urgent surgery should be avoided related to its morbidity. Patients with bleeding HCA and complete necrosis on imaging can be simply observed. Steatotic HCA (HNF1 α mutated) are at very low risk of complications and resection can be avoided in those >5 cm if major liver resection is needed. The management of HCA <5 cm and β catenin mutated is unknown but in our experience these patients are still observed especially that recently 3 genes of β catenin were described with only one at high risk of malignancy. Although bleeding and malignancy were reported in small HCA <5 cm, this should not modify the global strategy because these consist of small cases report or atypical presentation.