Functional recovery after liver resection
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Liver adenomatosis: re-evaluation of etiology and management
Introduction

Hepatocellular adenoma (HCA) is a benign solitary liver lesion which mostly occurs in young women with a history of oral contraceptive use (OCC) and cessation of OCC may regress the nodule size (1,2,3). Flejou et al defined liver adenomatosis (LA) as the presence of multiple (arbitrary ≥10), adenomas scattered throughout normal liver parenchyma in a patient with no history of steroid therapy or glycogen storage disease (4,5). Contradictory to Flejou, recent reports show female predominance and relation with OCC (6,7).

Clinical presentation of LA relates to the size of the largest nodule causing abdominal symptoms (4). Larger nodules often present with intralesional haemorrhage that may rupture within the peritoneal cavity (6,8). In literature, the preferred management for symptomatic patients is resection of the larger tumor(s) (2,6,9). The potential risk of malignant transformation has also warranted orthotopic liver transplantation as the recommended treatment (2,4). Recent reports, however, favor a more conservative approach and surgery is recommended for more aggressively growing lesions (5,7). In addition, less invasive embolization methods has been proposed. However, due to the rarity of the disease, the optimal management and prognosis remain unclear and there are no established guidelines for the management of patients with LA.

Disease etiology is also uncertain which hampers effective therapy of this disease. A recent study reports the presence of nonalcoholic steatohepatitis in a patient with LA (10) and there is also growing evidence of a connection between altered glucose metabolism and LA through germline mutations in hepatocyte nuclear factor (HNF) -1α (11,12). As altered glucose metabolism and nonalcoholic fatty liver disease (NAFLD) are closely connected, a relationship between LA and NAFLD is also possible. The main focus of this study is to identify novel features associated with LA that may have impact on surgical management and the short and long-term prognosis of these patients. We also discuss innovative treatment strategies potentially applicable to patients with LA.

Materials and methods

The patient database of Academic Medical Center (AMC) in Amsterdam between 1997 and 2006 was analyzed. For the definition of LA, the presence of multiple hepatic (>10) nodules and the exclusion of patients with glycogen storage disease or previous steroid therapy was used. The definitive diagnosis was based on imaging studies and/or histopathology by an experienced pathologist. Furthermore, clinical status, laboratory and radiological evaluation, histopathology, surgical management and outcome were reviewed. All published case reports and series were reviewed and analyzed in detail (1-12, 14-44). A search in the Medline (1 January 1963 to January 2006) was performed using MESH terms liver adenoma and liver adenomatosis. Patients from our own series and from the literature were combined and analyzed by commercial computer package (SPSS 10.1.).
Results

Demographics

Six patients were referred to the AMC in the period 1997-2006 (Table 1). All referred patients were female and the mean age at the time of the diagnosis of LA was 39.6 years (range 32-50). All patients had a history of OCC use for periods ranging from 4-20 years; in three patients, the precise length of OCC use was uncertain. Medical history was negative for glycogen storage disease or any previous steroid therapy. Two patients had diabetes (type II) diagnosed either shortly before or at the time of LA diagnosis.

<table>
<thead>
<tr>
<th>Sex/Age</th>
<th>Patient 1</th>
<th>Patient 2</th>
<th>Patient 3</th>
<th>Patient 4</th>
<th>Patient 5</th>
<th>Patient 6</th>
</tr>
</thead>
<tbody>
<tr>
<td>OCC use</td>
<td>abdominal discomfort</td>
<td>chronic abdominal pain</td>
<td>acute abdominal pain</td>
<td>acute abdominal pain</td>
<td>pain in left flank</td>
<td>incidental</td>
</tr>
<tr>
<td>Clinical manifestations</td>
<td>abdominal discomfort</td>
<td>chronic abdominal pain for 5 y</td>
<td>acute abdominal pain</td>
<td>acute abdominal pain</td>
<td>pain in left flank</td>
<td>incidental</td>
</tr>
<tr>
<td>Serum</td>
<td>ALT 1.5N, t-Bil 2N</td>
<td>AST 21N, ALT 29N, t-Bil 1.5N</td>
<td>AST 2N, ALT N</td>
<td>AST 20N, AST 16N, t-Bil 1.5N</td>
<td>AST N, ALT N</td>
<td>AST N, ALT N</td>
</tr>
<tr>
<td>CEA, α-FP</td>
<td>negative</td>
<td>negative</td>
<td>negative</td>
<td>negative</td>
<td>negative</td>
<td>negative</td>
</tr>
<tr>
<td>Ultrasound</td>
<td>multiple hypodense lesions</td>
<td>multiple hypodense lesions</td>
<td>unspecific</td>
<td>unspecific, subcapsular hematoma</td>
<td>multiple hyperdense lesions</td>
<td>multiple pain hyperdense lesions</td>
</tr>
<tr>
<td>CT</td>
<td>hyperdense in AP</td>
<td>hyperdense in AP</td>
<td>isodense in AP</td>
<td>hyperdense in AP</td>
<td>hyperdense in AP</td>
<td>hyperdense in AP</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>laparoscopy, biopsy</td>
<td>CT</td>
<td>laparoscopy, biopsy</td>
<td>US-guided biopsy</td>
<td>laparoscopy, biopsy</td>
<td>US-guided biopsy</td>
</tr>
<tr>
<td>Complications</td>
<td>intratumoral bleeding</td>
<td>intratumoral bleeding</td>
<td>intraperitoneal bleeding</td>
<td>subcapsular hematoma</td>
<td>no</td>
<td>intratumoral necrosis</td>
</tr>
<tr>
<td>Tumor size</td>
<td>11.5 cm</td>
<td>7.4 cm</td>
<td>10 cm</td>
<td>4 cm</td>
<td>4.4 cm</td>
<td>3.5 cm</td>
</tr>
<tr>
<td>Additional histology</td>
<td>severe microvesicular steatosis</td>
<td>diffuse hepatic steatosis</td>
<td>macrovesicular steatosis in nodules, FNH</td>
<td>No</td>
<td>steatohepatitis, FNH</td>
<td>severe steatosis</td>
</tr>
<tr>
<td>Treatment</td>
<td>conservative, after 12 months HAE</td>
<td>conservative, after 12 months HAE</td>
<td>1. resection of extrahepatic tumor 2. segment V resection</td>
<td>conservative</td>
<td>segment VI resection</td>
<td>conservative</td>
</tr>
<tr>
<td>Outcome</td>
<td>Well after 6 months, no tumor growth in CT</td>
<td>Well after 12 months, no tumor growth in CT</td>
<td>Well after 8 years, tumor growth in CT</td>
<td>Well after 24 months, no tumor growth in CT</td>
<td>Well after 25 months, no tumor growth in CT</td>
<td>Well after 30 months, tumor regression in CT</td>
</tr>
</tbody>
</table>

t-Bil = plasma total bilirubin, AP = arterial phase of contrast CT, FNH = focal nodular hyperplasia, HAE = hepatic artery embolization
Diagnostic modalities
Elevation of liver enzymes and bilirubin was seen in four patients of our own series (Table 1). In all patients, blood coagulation values were normal and tumor markers (CEA, α-fetoprotein) and hepatitis markers (B, C) negative. Five patients were symptomatic at the time of the LA diagnosis. In patient 6, LA was incidentally diagnosed during US-examination performed in relation with severe obesity. One patient presented with an unspecified abdominal swelling and others with acute pain in the left flank, right epigastri area or unspecifically in the abdomen. The size of the largest tumors ranged from 3.5 cm to 11.5 cm without a direct relation with the severity of the symptoms as the patient with the largest single tumor had only abdominal discomfort and no actual pain. Abdominal US was the initial imaging modality applied in all patients, followed by contrast-enhanced CT. The presentation of nodules in US and CT is summarized in Table 2. In patient 1, US-guided liver biopsy was suspicious of hepatocellular carcinoma but additional diagnostic laparoscopy with a new biopsy was negative for malignancy. In patients 5 and 6, US-guided biopsy was performed and histopathology was typical of HCA, without signs of malignancy. Additionally, $^{99m}$Tc-RBC scintigraphy was performed in patient 1, because unclear diagnosis after US, CT and US-guided biopsy. Scintigraphy showed atypical findings for hemangioma; a malignancy could not be excluded.

Table 2. Review of patient characteristics

<table>
<thead>
<tr>
<th>Variables</th>
<th>Total</th>
<th>Tumor size ≤5 cm</th>
<th>Tumor size &gt;5 cm</th>
<th>Unknown</th>
<th>Asymptomatic Symptomatic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>34.0 (12.75)</td>
<td>37.7 (14.75)</td>
<td>33.9 (12.54)</td>
<td>32.3 (13.53)</td>
<td>32.5 (13-53)</td>
</tr>
<tr>
<td>Male</td>
<td>12% (11)</td>
<td>5% (1)</td>
<td>12% (6)</td>
<td>17% (4)</td>
<td>17% (4)</td>
</tr>
<tr>
<td>Female</td>
<td>88% (83)</td>
<td>95% (19)</td>
<td>88% (45)</td>
<td>83% (19)</td>
<td>83% (20)</td>
</tr>
<tr>
<td>OCC</td>
<td>Yes</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age/sex</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tumor characteristics</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Primary symptoms</td>
<td>No / incidental</td>
<td>26% (24)</td>
<td>25% (5)</td>
<td>22% (11)</td>
<td>35% (8)</td>
</tr>
<tr>
<td>Acute pain</td>
<td>43% (40)</td>
<td>30% (6)</td>
<td>59% (30)</td>
<td>17% (4)</td>
<td>57% (41)</td>
</tr>
<tr>
<td>Chronic pain</td>
<td>16% (15)</td>
<td>25% (5)</td>
<td>6% (3)</td>
<td>35% (8)</td>
<td>23% (16)</td>
</tr>
<tr>
<td>Pain #</td>
<td>9% (8)</td>
<td>15% (3)</td>
<td>8% (4)</td>
<td>4% (1)</td>
<td>11% (8)</td>
</tr>
<tr>
<td>Hepatomegaly</td>
<td>31% (29)</td>
<td>40% (8)</td>
<td>20% (10)</td>
<td>48% (11)</td>
<td>13% (3)</td>
</tr>
<tr>
<td>Non-specific</td>
<td>7% (7)</td>
<td>15% (3)</td>
<td>4% (2)</td>
<td>8% (2)</td>
<td>10% (7)</td>
</tr>
<tr>
<td>GI symptoms</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tumor size</td>
<td>Mean (range)</td>
<td>3.6 (2-4.5)</td>
<td>7.8 (5-15)</td>
<td>unknown</td>
<td>5.4 (3-8)</td>
</tr>
<tr>
<td>(n of patients)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bleeding</td>
<td>No</td>
<td>47% (44)</td>
<td>65% (13)</td>
<td>47% (24)</td>
<td>30% (7)</td>
</tr>
<tr>
<td>ITN</td>
<td>2% (2)</td>
<td>5% (1)</td>
<td>2% (1)</td>
<td>4% (1)</td>
<td>3% (2)</td>
</tr>
<tr>
<td>ITB*</td>
<td>26% (24)</td>
<td>20% (4)</td>
<td>26% (13)</td>
<td>28% (7)</td>
<td>13% (3)</td>
</tr>
<tr>
<td>IPB**</td>
<td>20% (19)</td>
<td>10% (2)</td>
<td>26% (13)</td>
<td>16% (4)</td>
<td>8% (2)</td>
</tr>
</tbody>
</table>
### Table 1

<table>
<thead>
<tr>
<th>Variables</th>
<th>Total</th>
<th>Tumor size &lt;5 cm</th>
<th>Tumor size ≥5 cm</th>
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<th>Asymptomatic Symptomatic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>94</td>
<td>21% (20)</td>
<td>53% (51)</td>
<td>26% (23)</td>
<td>25% (24)</td>
</tr>
<tr>
<td>Male</td>
<td>34.0 (12-75)</td>
<td>37.7 (14-75)</td>
<td>33.9 (12-54)</td>
<td>32.3 (13-53)</td>
<td>32.5 (13-53)</td>
</tr>
<tr>
<td>Female</td>
<td>88% (83)</td>
<td>95% (19)</td>
<td>88% (45)</td>
<td>83% (19)</td>
<td>83% (20)</td>
</tr>
<tr>
<td>OCC Yes</td>
<td>52% (43/83)</td>
<td>50% (10)</td>
<td>57% (29)</td>
<td>22% (5)</td>
<td>54% (13)</td>
</tr>
<tr>
<td>OCC No/ incidental</td>
<td>26% (24)</td>
<td>25% (5)</td>
<td>22% (11)</td>
<td>35% (8)</td>
<td>100% (24)</td>
</tr>
</tbody>
</table>

### Management and Outcome

#### Surgical treatment

Initial management consisted of surgical resection of the largest lesion in three patients (patients 1, 3 and 5). In patient 1, preoperative right portal vein embolization (PVE) was performed to increase remnant liver volume in the presence of severe steatosis. Subsequently, right hemihepatectomy extended with resection of half of segment 4a was performed without complications. In patient 3, a LA lesion hanging from the liver was resected laparoscopically and was complicated by bleeding. At subsequent laparotomy, the largest nodule located in the segment 5 was resected because of intended pregnancy. The patient has been asymptomatic during a follow-up time of 8 years; however, the size and number of nodules have slightly increased on CT. Also in patient 5, the largest nodule located in segment 6 was resected without complications. Follow-up of 25 months has been uneventful and patient is pain-free.

#### Conservative treatment

Initially, three patients (patients 2, 4 and 6) were observed including cessation of OCC medication. In patient 2, the diameter of the largest nodule had increased after 12 months on follow-up CT. Because the patient also had abdominal discomfort, percutaneous selective embolization of the nodule via the hepatic artery was undertaken. Follow-up of 12 months was uneventful and no change in nodule size or number were detected on CT. Patient 4 who presented with an intrahepatic bleeding was treated conservatively and after 2 months, resolution of a subcapsular hematoma was seen on CT with signs of recent bleeding. In addition, multiple lesions (< 2-4 cm) were detected in segments 2-4. Follow-up was uneventful for 24 months and the patient is currently symptom-free. Patient 6 was observed and after withdrawal of OCC, reduction of lesion size was observed. At 30 months after diagnosis of LA, no nodules were detected on CT.

#### Histopathology

In all our patients the histopathology of the nodules met with the previously reported criteria for benign adenoma (13). All nodules showed cell plate 1-3 layers thick in the reticulin stain with intact reticulin pattern. The lesional hepatocytes showed cellular proliferation, variable sized and
double-form nuclei most prominent in the largest nodules. In the nodules, the arterialization, small arteries, derived (CD-34 positive cells) from sinusoidal beds. The non-lesional hepatic parenchyma showed intact architecture with regular central veins and portal fields. In the portal fields and around central veins inflammatory cells with segment patterned nuclei were present. In three patients, a resection specimen and in two patients, a representative needle biopsy was available for histopathological evaluation. In patient 4, a biopsy obtained under US-guidance was considered inadequate for reliable evaluation. In patients 1 and 3, presenting with the largest tumors, i.e. 11.5 cm and 10 cm, respectively, the tumors were macroscopically encapsulated with intratumor necrosis. The smaller tumors were mostly encapsulated and well demarcated from the surrounding hepatic parenchyma. In 4 patients, steatosis was present in the non-lesional hepatic parenchyma surrounding the nodules. In all patients steatosis was of the severe type, involving more than 60% of all hepatocytes. Patient 1 presented with severe, centrilobularly located, microvesicular steatosis, patient 3 with severe macrovesicular steatosis and patient 6 with severe micro- and macrovesicular steatosis. In patient 5, steatosis had developed into steatohepatitis with fibrosis. In addition, patients 3 and 5 had also a single FNH lesion among multiple LA nodules. The overall non-lesional parenchyma showed intact cellular architecture with regular central veins and portal fields in all patients.

Discussion

Demographics

We report the largest review so far of 94 patients with LA collected to date, consisting of all published case reports and all small series including our own series (Table 2). Only descriptive data analysis was performed because of the heterogeneous source of data. Strong female predilection in incidence of LA suggested in literature was also seen in our review (6,7). Of all patients, 52% of females had a history of OCC use. Eight patients were younger than 18 years and 12% were male (Table 2). As estrogen/progesterone-positive and negative receptors can be unevenly found in LA nodules, it seems that the role of exogenous estrogen is not important in induction of LA but might play a role in disease progression (5). In our analysis a slightly higher incidence of OCC use was seen in females with larger tumor sizes, i.e. > 5cm.

Etiology

The etiology of LA remains unclear and a variety of predisposing conditions have been suggested. Congenital or acquired hepatic vascular abnormalities tend to occur more often in patients with FNH and HCA. Of all patients, 8 had a concomitant single lesion of FNH and some presented with co-existing vascular malformations suggesting some role for vascular abnormalities (6,7,23,29). The first connection between LA and diabetes was reported in 1978 (19). Interestingly, 2 of our own patients had diabetes (type II) diagnosed at the time of LA diagnosis. Unfortunately, diabetes was assessed only in few reviewed studies (10,11,12,19). Recent studies report a familial autosomal transmission of LA in one family and a familial type of LA linked with germline mutations of HNF-1α in two families (6,12). These mutations are associated with a rare form of diabetes, a non-ketotic type present in patients younger than 25 years (11-12). HNF-1α mutation could present a genetic predisposition and LA would develop in presence of another factor because the expression of
The mutation phenotype was variable within the families (12). This can be a vascular anomaly or somatic mutation in another allele. Unfortunately, HNF-1α detection by immunohistochemistry is not yet possible in paraffin-preserved tissue samples and therefore, this analysis was not possible in our own patient series.

The etiology of NAFLD has close connection with western lifestyle and is therefore considered a rising clinical challenge (45). An association between LA and NAFLD was recently suggested (10). Interestingly 18% of all LA patients had steatosis, varying from mild to steatohepatitis. In our own series, 5 of 6 patients had steatosis along with LA. Three patients had histologically confirmed steatosis; one patient had severe macrovesicular, one severe microvesicular and one had combined micro- and macrovesicular steatosis. In addition, one patient had diffuse steatosis diagnosed by US. Furthermore, one patient had steatohepatitis, which is second ever published in the literature (10).

LA nodules typically show steatosis but it seems that the co-existence of non-tumoral steatosis has increased during the last years. This might in part be explained by increased risk factors for steatosis and recognition of steatosis as a potential risk factor in liver surgery. However, LA is diagnosed also in young, lean adults and children. Furthermore, the microvesicular type of steatosis is not commonly connected with obesity-related steatosis as opposed to the macrovesicular type. The microvesicular steatosis is often seen in patients with more acutely induced steatosis, such as by toxins or drugs. Glycogen storage disease induces rapid and massive LA, presenting often in children younger than 5 years (2). Therefore, considering all these aspects, etiology of LA has a potential connection to deranged hepatic glucose metabolism with a yet unknown mechanism. Further research is needed to unravel this aspect.

The diagnosis

In 43% of all analyzed patients with LA (including the literature and our own series), clinical presentation was with acute pain. However, in contrast to previous reports, the diagnosis was fortuitous in 25% of patients. Early diagnosis is vital, as hemorrhage is a common complication in patients with LA. In literature, an overall bleeding rate from 46% to 63% is reported (4,5). In our analysis, the overall bleeding rate was 46% and had in two cases led to death (6, 23). In 54% of symptomatic patients, intraperitoneal or intratumoral bleeding was present at the time of diagnosis. The actual prevalence of hemorrhage is difficult to estimate as symptomatic patients are more likely to seek medical help thus creating a selection bias. However, of all asymptomatic patients with incidentally discovered LA, only 8% had intraperitoneal and 13% had intratumoral bleeding.

In patients with intra-abdominal bleeding, LA is diagnosed on CT or during emergency laparotomy. However, in less acute patients the diagnosis is more complicated. As seen in our series (Table 1) and in literature, plasma liver enzymes are unreliable with poor correlation with LA. Most symptomatic patients presented with epigastric or abdominal pain or discomfort of acute or chronic onset (Table 2). Additional symptoms include gastrointestinal complaints and hepatomegaly (4,6,32). During diagnostic work-up, several differential diagnoses might emerge, because of diversity of the LA tissue components such as necrosis, fat, calcification and hemorrhage, present as a variety of hypo-, iso- and/or hyperdense radiological features. Grazioli et al concluded that the most useful method for initial diagnosis is multiphase-helical CT or MRI (7). Most adenomas were uniformly or heterogeneously hyperattenuating lesions during the arterial phase like in our
series. However, noncontrast images are needed for identification of local fat and hemorrhage and portal phase images for evaluation of vascular anomalies (7). A potential modality to identify co-existing steatosis is nuclear magnetic resonance spectroscopy (1H-MRS) providing a sensitive and quantitative technique to measure intrahepatic fat content (46). The obvious advantage of 1H-MRS is that it samples noninvasively a considerably larger liver volume than standard liver biopsy. With 1H-MRS the analysis of various liver lobes and/or regions is also possible, diminishing the bias introduced by heterogeneity of distribution of hepatic fat (46,47).

Even though imaging is useful in lesion identification, the total number of lesions detected during surgery is usually higher because of the extensive number of small adenomas usually scattered throughout the liver (43). Together with multiphase-helical CT or MRI, histopathology of several nodules should be the gold standard for definitive diagnosis of LA. Only single core biopsy of one nodule might be misleading or even non-diagnostic due to co-existing parenchymal diseases and/or different lesion components. For an accurate diagnosis and a reliable exclusion of malignancy, several biopsies should be taken preferably during laparoscopy or laparotomy to ensure the control of any bleeding. It should be taken into account that the bleeding risk after single biopsy is 0.7% (49) and is likely higher in LA patients because of the hypervascular structure of the nodules. Intratumoral bleeding was already present in 26% of all LA patients at the time of diagnosis.

Management of LA

In figure 1 is summarized the suggested management of LA patients. In all management strategies, a close follow-up is necessary using CT or MRI even if clinical presentation does not require invasive management. Also, because of the unsolved issue of potential malignant transformation, control of α-fetoprotein levels should be added to the follow-up (2,5,7). In our patients no signs for malignant transformation could have been detected during follow-up. Surgical management remains complex, not only because of the large number of adenomas requiring major liver

![Figure 1. The proposed management strategy for asymptomatic and symptomatic patients with LA.](image-url)
Liver resection but also because of the risk of hemorrhagic complications during surgery. Large adenomas, particularly superficial, have a higher risk of bleeding with a mortality rate of 7%. In emergency, i.e. in case of rupture of the bleed into the peritoneal cavity, selective angiography of the hepatic artery with embolization is recommended (50). In case of extensive bleeding, either excision of the bleeding tumor or packing has been recommended (12,44). In our review, an equal number of symptomatic and asymptomatic patients were primarily resected (57% vs. 54%) and in both groups, an equal number of secondary interventions were performed (21% vs 17%). In patients with tumors >5 cm, 59% was resected compared to 35% of smaller tumors. In larger tumors, secondary interventions were needed in 25% of patients vs. 8% if the size was less than 5 cm. However, 40% of smaller tumors continued to grow during follow-up. Interestingly, after surgery in patients with tumors > 5 cm, the remaining tumors remained stable. Of all analyzed patients (94), tumor(s) showed regression during follow-up only in three patients. Liver resection should be considered when a nodule is larger than 5 cm, if malignancy cannot be ruled out or when a larger nodule presents with severe pain. For the most aggressively growing tumors, OLT has been considered too (6,7). Still, a selection of patients can benefit from extensive resection without any negative effect on outcome as we noticed no difference in the long-term outcome of conservatively or surgically treated patients. Resection can be preceded by PVE to increase the future remnant liver volume consequently decreasing the risk of postoperative liver failure and complications. However, as PVE increases hepatic arterial blood supply, an increase in the volume of hyperarterialized LA is possible, as found in our patient 1. Therefore, the addition of sequential arterial embolization (TAE) or the combination therapy, i.e. TAE followed by percutaneous ethanol injections, might be preferred (51). In patient 4, an effective hepatic artery embolization was sufficient to induce tumor regression.

In any patient with LA considered for liver resection, potentially co-existing steatosis should be acknowledged. This is because of the impact of liver steatosis on the outcome of liver surgery. Evidence is accumulating that all grades of steatosis, even the mildest, potentially affect the recovery of patients after transplantation and liver resection (52,53,54). It is noteworthy in this connection that in our own series of 6 patients, 5 featured severe type of steatosis.

Conclusion

LA is a progressive, benign parenchymal disease mainly occurring in females. There is a potential link with hepatic steatosis with implications for the management of patients with LA. Noninvasive diagnosis is difficult because of the variety of tissue components. Management should primarily be conservative with careful follow-up.

Reference list


44. Skarupa DJ, Ellison C, Vitellas KM, Frankel WL. Hepatocellular adenomatosis is a rare entity that may mimic other hepatocellular lesions. Ann Diag Pathol 2004; 8:43-49.


