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Focal Fatty Change of the Liver

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Focal fatty change of the liver is a nodular lesion which is a rarely described and poorly characterized entity. The hepatic nodule measured 1.4cm at its maximum diameter, was subcapsular in location and occurred adjacent to the falciform ligament. Microscopically it was composed of hepatic tissue with a preserved lobular architecture. The central venous structures and portal tracts with their triads were regularly placed. The cytoplasm of almost all of the hepatocytes within the nodule was replaced by macrovesicular fat vacuoles with the nuclei displaced. Several large abnormal vessels were found at the margin of the nodule. The nodule was discovered incidentally on postmortem examination of a female infant who proved, at autopsy, to have multiple cardiac anomalies and bronchopneumonia. The possible inadequate local tissue perfusion due to abnormal intrahepatic vessels at this particular location could be augmented by multiple cardiac anomalies culminating in focal ischemia and focal fatty change. When encountered in surgery or on gross examination, it could be confused with other space occupying lesions such as liver cell adenoma, abscess and metastatic lesions.

Key Words: Liver, fatty change, focal fatty change, lipoma

Fatty change of the liver is generally a diffuse process associated with many conditions, including chronic hypoxia, obesity, malnutrition and administration of steatogenic drugs. Frequently it has a zonal pattern, either central or periportal, depending upon its etiology.

In a recent autopsy case a focal fatty change of the liver was found, which on gross examination was thought to be a liver cell adenoma with a fatty change or a lipoma. There has been only one report which reviewed ten such lesions (Brawer *et al.* 1980), although two of the thirteen isolated liver nodules reported by Shojania and Hogg (1975) might represent similar lesions.

The objective of this report is to draw attention to this entity since it may mimic space occupying lesions of the liver and to discuss the pathogenic mechanisms.

CASE REPORT

The patient, a 6 month old female, was admitted due to dyspnea and cough for one day.

She was born normal spontaneous vaginal delivery at full term with a birth weight of 3.3 kg, and was known to have congenital heart disease. Her brother has died of unknown causes on the 2nd postnatal day. There were no particular parternal or maternal diseases.

On physical examination, the patient was cyanotic. The chest was asymmetrically expanded with a mild substernal retraction and a slightly bulged precordium. The breathing sound was coarse, and there were rales on the right lower lung field with bronchi on the whole lung field. An auscultation disclosed one plus thrill and heaving. There was a grade III/IV systolic murmur on the left midsternal region and an increase in the S2 sound. The patient died on the third hospital day. An autopsy was performed and the final anatomical diagnosis was multiple congenital heart disease (endocardial cushion defect, patent foramen ovale and patent ductus arteriosus), bilateral bronchopneumonia and a focal fatty change of the liver.

PATHOLOGY OF THE LIVER (TABLE 1)

Grossly the liver was of normal size and appeared normal except for a small yellow nodule. The nodule was located subcapsularly at the antero-inferior aspect of the medial margin of the right lobe, near the falciform ligament. It was roughly oval shaped,

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Table 1. Pathology of the hepatic nodule

Gross findings	Location	subcapsular
	Circumscription	well
	Encapsulation	–
	Cut surface	yellow
Microscopic findings	Compression perinodular tissue	–
	Large abnormal vessels within and at the margin of the nodule	+
	Evidence of regeneration	–
	Fatty change	4+
	Inflammatory infiltration	not significant
	Multilobular involvement	+

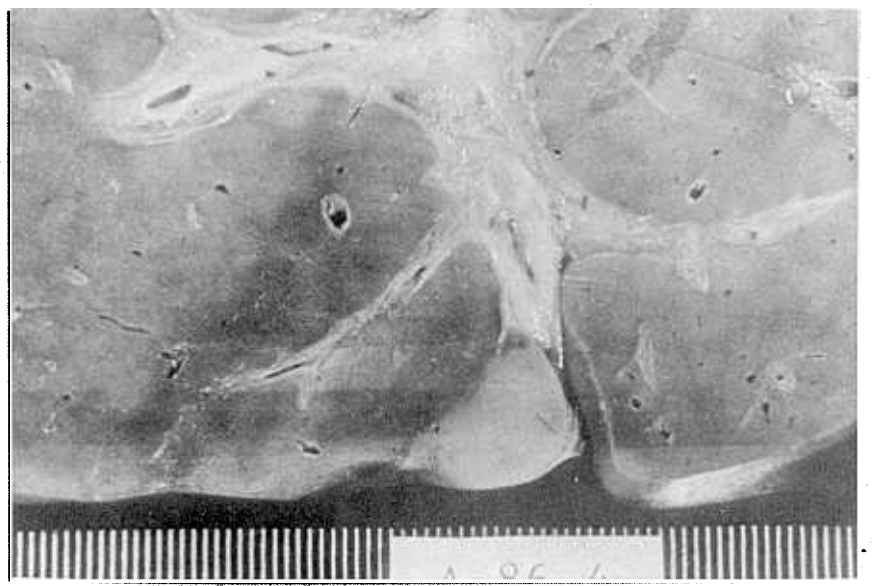

Fig. 1. *Gross finding of the fatty nodule; note the subcapsular location at the edge of the right lobe, closely approximated to the falciform ligament.*

measuring 1.4cm at its maximum diameter. It contrasted sharply with the surrounding liver tissue, but was not encapsulated (Fig. 1).

Microscopically the nodule was less well-defined than expected by the gross appearance. Within the nodule there were regularly spaced portal and central venous structures, reflecting that many hepatic lobules were involved (Figs 2 and 3a). One of the important features was the lack of compression of the perinodular liver tissue. The cytoplasm of the majority of the involved hepatocytes contained a single large fat vacuole displacing the nucleus. The remainder of the liver showed mild centrilobulr congestion which was present within the nodule as well, and there was a minimal fatty change in the hepatocytes adjacent to the nodule. There was no histological evidence of regeneration, inflammatory infiltration or fibrous septal formation. Of importance were the prominent blood vessels within and at the margins of the nodule. These were not associated with portal tracts or central veins, and has abnormally thickened walls (Fig. 3b). The vessels had no mural changes suggestive of a post-thrombotic state.

DISCUSSION

The occurrence of focal fatty change in the liver gives rise to interest, especially on its pathogenesis. The particular location and the associated congenital

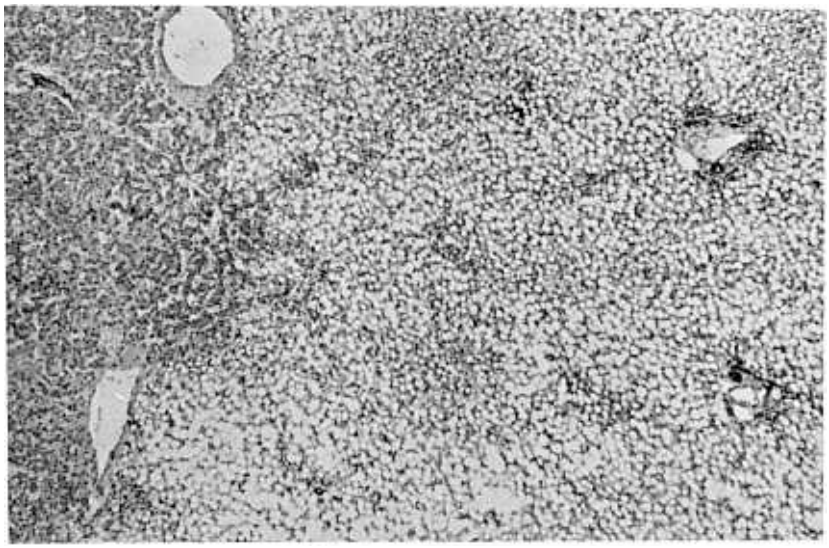

Fig. 2. *Low magnificaton of the liver; note the absence of capsule between the fatty nodule and the normal appearing hepatic tissue, several portal tracts within the fatty nodule, and two thick-walled veins adjacent to the lesion (Hematoxylin-eosin stain, 100×).*

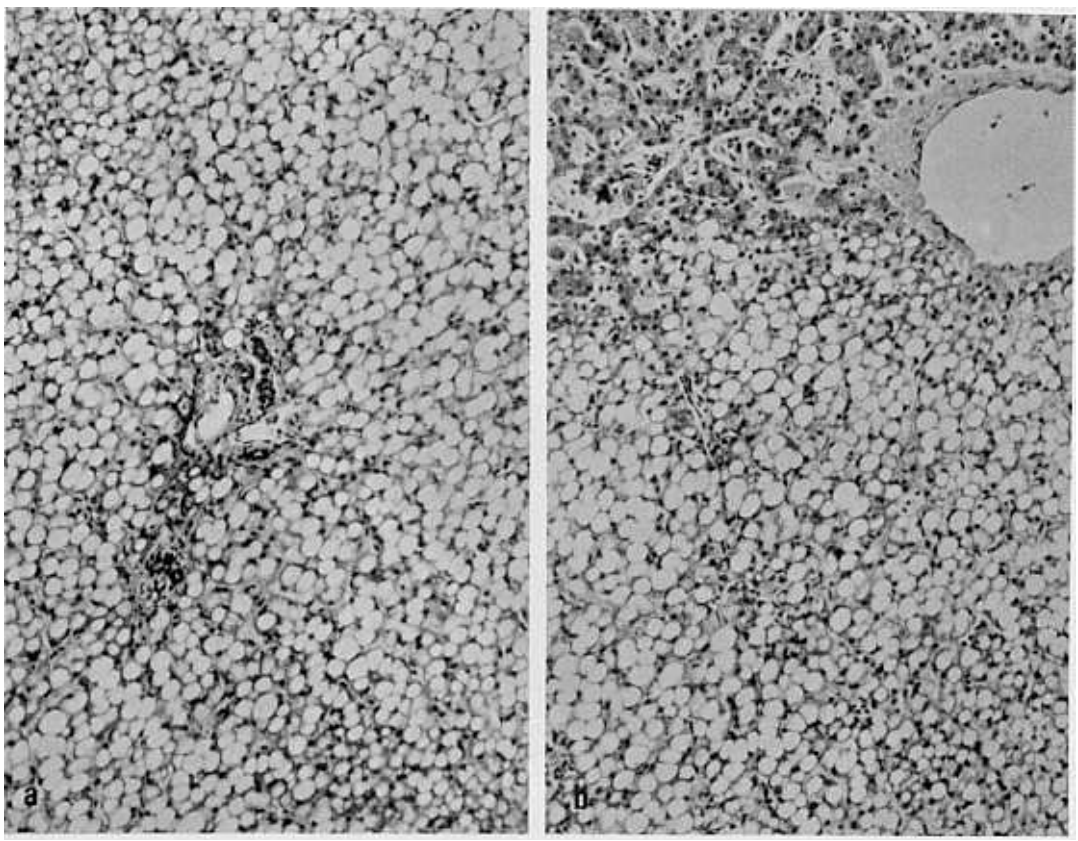

Fig. 3. *Higher magnification showing a normal appearing portal tract within the lesion(a), and fairly good demarcation of the lesion (b) (Hematoxyline-eoxin stain, 200×).*

heart disease of this case could explain the focality of this process. The subcapsular location, situated at the periphery of the portal venous and hepatic arterial circulation, suggests that focal tissue hypoxia may be present (Brawer *et al.* 1980). The focal fatty change was separated from Glisson's capsule by a narrow fringe of uninvolved parenchyma, that is similar to hepatic infarctions (Lund *et al.* 1934). In the present case as well as 1 of the 10 cases reported by Brawer *et al.* (1980), the fatty nodule occurred adjacent to the falciform ligament, at the watershed of the right and left hepatic arteries (Seeley *et al.* 1972). Alternately, inadequate local tissue perfusion by such abnormal anastomoses of the aberrant vessels as in the cases of Shojania and Hogg (1975) could cause tissue hypoxia leading to focal fatty change. The large abnormal vessels within and at the margin the nodule of the present case might be a finding in aggreement with this postulation. The occurrence of focal fatty change in an infant suggests that the local intrahepatic abnormal vasculature is of a congenital origin. Multiple cardiace anomalies with the consequent systemic hypoxia might have accentuated the local low perfusion.

It is apparently not a lipoma of the liver, in view of the fat vacuoles undoubtedly within the hepatocytes and the portal and central venous structures within the nodule. Brawer *et al.* (1980) believed that the lipomas of the liver reported by Young (1951) and Ramchand *et al.* (1970) were lesions of this type, since they were unencapsulated with less well defined borders and in Young (1951)'s case there was a mild fatty change in the surrounding parenchyma. It is possible that some other cases thought to be lipomas of the liver are of the same nature as already pointed out by Simon in 1934.

The fate of focal fatty change of the liver is unknown. The lack of compression of the perinodular hepatic tissue may be taken as evidence that the nodule is no longer growing. Also the focal subcapsular or septal fibrosis that is sometimes noted in surgical liver specimens may be a result of prolonged or severe local ischemia. Although none of the patient's symptoms appeared to be related to it and its discovery at autopsy was entirely incidental, there may be possible confusion during a laparotomy or on gross examination with other focal lesions of the liver such as liver cell adenoma, abscess or metastatic tumor.

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