Tumor-like Necroses of the Liver: Liver Infarct and Hepatic Pseudo-infarct (Zahn's Infarct)

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Abstract

Liver infarcts are macroscopic parenchymal alterations that may mimic, through their mass effect, neoplastic processes. An anemic infarct or infarction of the liver is defined as an ischemic necrosis that is usually caused by hepatic artery occlusion, thrombosis of the celiac artery, portal vein occlusion, or mixed hepatic vascular occlusions. Although hepatic artery circulation is crucial for the biliary tract, acute occlusion of this artery can also cause hepatic ischemic necrosis. Anemic liver infarcts are well-demarcated pale areas of sometimes more than 10 cm diameter, often wedge shaped and located both peripherally and deep in the liver substance. These lesions can undergo secondary changes, including abscess formation following infection, hemorrhage, liquefaction, calcification, bile accumulation, and fibrosis/scarring. Large infarcts may be separated from preserved liver parenchyma, a process termed sequestration. In contrast to true infarct, hepatic pseudo-infarct or Zahn's infarct is a well-demarcated zone of hepatic congestion leading to parenchymal atrophy.

Introduction

Infarct (anemic infarction) of the liver is defined as an ischemic necrosis that results in a macroscopically visible area or mass that may be confounded with a neoplastic process. In contrast,

A. Zimmermann, *Tumors and Tumor-Like Lesions of the Hepatobiliary Tract*,
DOI 10.1007/978-3-319-26587-2_137-1

pseudo-infarct of the liver (Zahn's infarct) represents a hepatic area of marked congestion associated with atrophy of the parenchyma, but without coagulation necrosis. True anemic liver infarct can, due to the altered features of tissue and tissue swelling, produce a mass that can closely mimic a hepatic tumor (Bohn et al. 2010).

Epidemiology

True anemic infarction of the liver is rare, but has already been described in the nineteenth century (Rattone 1888; Ogle 1895; Baldwin 1902; Rudczynski 1905; Kretz 1916; Wiessner 1917; Pass 1935; Woolling et al. 1953; Parker 1955; Kanter 1965; Seeley et al. 1972; Soni and Persaud 1972; Chen et al. 1976). The rarity of this lesion has been attributed to the protection of the liver from ischemic events and complications provided by the double arterial and portal venous blood supply. However, this proposed explanation does not hold true, because if the arterial blood supply alone is arrested, an intact portal venous blood supply cannot protect the liver from infarction in many cases (Nakata and Kanbe 1966; review: Carroll 1963). Winternitz (1911) did not encounter a single case among 3,500 autopsies performed at Johns Hopkins Hospital. In an investigation of 5,420 consecutive autopsy cases, 20 hepatic infarction cases could be selected in Japan (Saegusa et al. 1993). Anemic liver infarction is a disorder mainly occurring in adults, but it also exists in the pediatric age group (Kochin et al. 2011).

Etiology of True Hepatic Infarction

Numerous types of vascular lesions can cause anemic liver infarction, but most of the reported cases are due to occlusion of the hepatic artery related to various pathologies. The main vascular lesions causing, or associated with, liver infarction are listed in Table 1. It has once again to be emphasized that an intact portal vein blood flow cannot protect the liver substance from infarction when the hepatic arterial blood flow is arrested. In addition to distinct occlusive events of hepatic blood vessels, systemic circulation failure can significantly contribute to the development of hepatic infarction, even in the absence of complete vascular occlusion. In the autopsy study of Saegusa and coworkers (1993), 85 % of the infarct cases were clinically associated with systemic circulatory insufficiency. In fact, the pathogenesis of hepatic infarction often requires an association of several factors synchronously compromising compensatory blood flow mechanisms in the liver.

Table 1 Vascular lesions causing anemic infarction of the liver

Hepatic artery occlusion
Spontaneous thrombosis of the hepatic artery
Hepatic artery thrombosis in hepatic cancer
Hepatic artery thromboembolism
Embolized vegetations in bacterial endocarditis
Polyarteritis nodosa and other types of arteritis/vasculitis
Hepatic artery aneurysms and pseudoaneurysms
Dissecting aortic aneurysm
Coagulopathies
Accidental artery ligation
Thrombosis of the celiac artery
Portal vein (PV) occlusion
PV occlusion in liver cirrhosis
PV occlusion in hematologic diseases and coagulation disorders
PV occlusion in infectious disease/pylephlebitis
PV occlusion in malignant hepatic disease
Mixed hepatic vascular occlusions
Thrombosis of both the hepatic artery and portal vein
Thrombosis of the portal vein combined with hepatic arterial vasospasm
Mixed thrombosis of the hepatic artery and portal vein (e.g., in polycythemia vera)

Hepatic Artery Occlusion

Although hepatic parenchyma strongly depends on portal vein blood flow, the hepatic artery plays a critical role in blood supply of the liver and specifically for the biliary tract (review: Rappaport and Schneiderman 1976). There are documented events of spontaneous hepatic artery thrombosis followed by anemic liver infarction (Carroll 1963; OConnor et al. 1976).

Thrombosis of the hepatic artery causing infarction was found in patients with primary hepatic cancer or hepatic metastatic disease (Carroll 1963). A further cause of hepatic artery occlusion is an entire spectrum of abdominal interventional procedures, including angiography, therapeutic transcatheter arterial chemoembolization/TACE, radiofrequency ablation, and ethanol injection (Trojanowski et al. 1980; Holbert et al. 1996; Fujiwara et al. 2004; Akahane et al. 2005; Dai et al. 2007; Kim et al. 2007; Chiu et al. 2009; Guiu et al. 2012; Ladra Gonzalez et al. 2013; Dolak et al. 2014), and disorders leading to hemoconcentration, such as nephrotic syndrome (Vea et al. 1990). The hepatic artery can also be occluded by a thromboembolus originating from the left heart (e.g., subsequent to atrial fibrillation or mural ventricle thrombus after myocardial infarction) or from atherosclerotic plaques of the aorta or celiac trunk. Embolic occlusion of the hepatic artery can be followed by secondary thrombosis proximal to the embolus. A second source of embolism is detached vegetations in bacterial endocarditis (Henrich et al. 1975; Carroll 1963). Hepatic infarction caused by hepatic vascular accidents is a well-established complication of preeclampsia and eclampsia (Kronthal et al. 1990; Miyakoshi et al. 2004; Cholongitas and Burroughs 2008).

True liver infarction is a well-documented complication of polyarteritis nodosa involving the celiac plexus and the hepatic artery (Pass 1935; Price and Flanagan 1953; Haratake et al. 1988, Bohn et al. 2010). Other types of vasculitis causing hepatic artery stenosis or occlusion and hepatic infarction include Churg-Strauss syndrome (Otani et al. 2003), chronic periaortitis (Salvarani et al. 2011), vasculitis in systemic lupus erythematosus/SLE (Matsumoto et al. 1992a), vasculitis in the antiphospholipid syndrome (Millan-Mon et al. 1993), and radiation aortitis (Cox and Millar 1993). The hepatic artery can also undergo occlusion in the setting of artery aneurysms and pseudoaneurysm followed by liver infarction (Ross and Osler 1877–1878; Chuang et al. 2005) or dissecting aneurysm of the aorta (Myrtue et al. 1994). Anemic liver infarct was also observed following therapeutic or accidental hepatic artery ligation, e.g., in the setting of cholecystectomy (Kehr 1903; Kerr 1933; Graham and Cannell 1932–1933). Similar to thrombosis of the hepatic artery proper, thrombotic occlusion of the celiac trunk can also cause anemic hepatic infarction (MacDonald and Holt 1966).

Occlusion of the Portal Vein

In comparison with hepatic artery occlusion, thrombotic occlusion of the portal vein alone only rarely gives rise to hepatic anemic infarction, although most of the parenchyma is supplied by portal venous blood (Versé 1907; Yamashita et al. 1997). Hepatic infarction due to portal vein thrombosis was found in patients with liver cancer, cirrhosis, or chronic pancreatitis (Kim et al. 2002).

Mixed Hepatic Vascular Occlusions

Anemic liver infarction resulting from mixed hepatic and portal vein thrombosis was observed following blunt abdominal trauma (Francque et al. 2004), liver transplantation (Haque et al. 2009), transcatheter arterial embolization/TAE (Takakuwa et al. 1993), or polycythemia vera rubra (Ghandur-Mnaymneh 1976). Hepatic infarct can also develop in patients with HELLP syndrome and factor V Leiden having portal vein thrombosis plus arterial vasospasm (Seige et al. 1998).

Complex Mechanisms Affecting Several Vascular Systems

Very rarely, hepatic infarction was caused by torsion of an accessory liver lobe, inducing complex patterns of vascular occlusion and also primarily involving the microvascular system (Lotte and Madier 1960). Hepatic infarction was also observed in the setting of S-A hemoglobin (Mengel et al. 1963).

Hepatic Infarction in the Absence of Vascular Occlusion

Exceptionally, hepatic infarction was observed in the absence of radiologically detectable vascular occlusions (so-called nonocclusive hepatic infarction; Sundaram et al. 1978).

Clinical and Imaging Features

The clinical signs and symptoms of anemic hepatic infarction are largely nonspecific and include upper abdominal pain or discomfort, vomiting, and signs of acute abdomen or upper abdominal peritonitis. At imaging, hepatic infarcts appear as wedge-shaped, rounded or oval, or irregularly shaped low-attenuation lesions usually paralleling bile ducts, whereby wedge-shaped infarcts are peripherally located (Adler et al. 1984; Lev-Toaff et al. 1987; Holbert et al. 1996; Cook and Crofton 1997; Smith et al. 1998).

Macroscopic Pathology

Macroscopically, anemic liver infarcts are well-demarcated pale areas of sometimes more than 10 cm diameter that are wedge shaped and located either deep in the liver substance or to peripheral parts of the organ (Figs. 1 and 2).

Fig. 1 Peripheral anemic infarction in a cholestatic liver

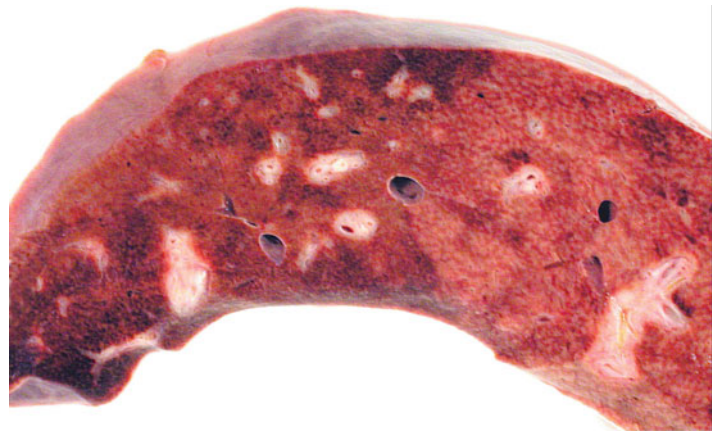

Fig. 2 Anemic infarction of the liver in hepatic arteritis

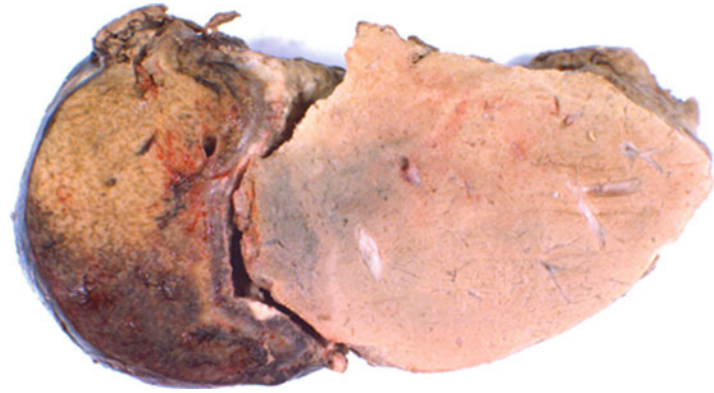

Fig. 3 Large sequestered infarct of the liver. The necrotic tissue has detached from the intact adjacent liver substance

Peripheral infarcts are typically separated from the liver capsule by a thin rim of preserved liver parenchyma, as this tissue is supplied by a different vascular system. Subcapsular infarcts were also observed following liver transplantation (Abecassis et al. 1991). In rare cases, an infarct can occupy almost an entire liver lobe (Carroll 1963). The pale area of ischemic necrosis can show a red rim at the edge caused by hyperemia (congestion) and hemorrhage from damaged microvessels. In infarcts caused by hepatic artery thrombosis, the arterial thrombus often or always extends up to the infarct in the liver substance (Carroll 1963).

Liver infarcts can undergo several types of secondary changes, including liver abscess due to superinfection, hemorrhage, liquefaction of necrotic tissue, calcification, and fibrosis/scarring. In case the infarction involves larger intrahepatic bile ducts, bile leakage ensues, resulting in bile accumulation in the infarcted tissue (bile lakes; Peterson and Neumann 1984). Large necrotic areas may become separated from preserved (vital) hepatic parenchyma, resulting in infarct sequestration (Fig. 3).

Pass (1935) reported that hepatic infarcts caused by polyarteritis nodosa of the hepatic artery frequently convert into large abscesses that may be difficult to distinguish from genuine pyogenic abscesses. Large postinfarction abscesses of the liver may be associated with collapse of the right lower lobe of the lung (-Davson et al. 1948).

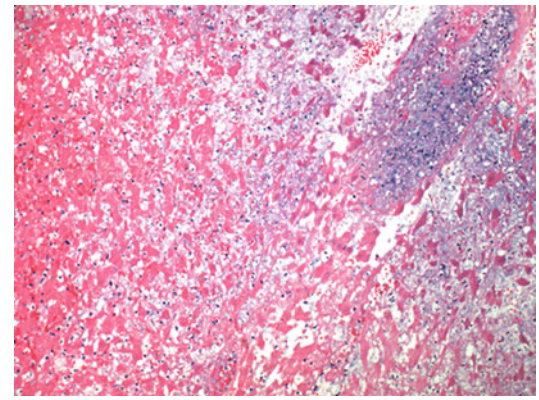

Fig. 4 Anemic infarction of the liver. The traces of the previous hepatic parenchyma are visible in the form of "shadow liver plates/trabecules." Note the numerous nuclear debris (hematoxylin and eosin stain)

Histopathology

Macroinfarcts typically show distinct zones, similar to those described in renal ischemia (Figs. 4, 5, and 6; Sheehan and Davis 1958) and animal experiments (Beresnegowski 1908). A central area of variable diameter shows eosinophilic cells with a granular cytoplasm, a vague circumference, and fading or faded nuclei (karyolysis). Nuclear debris are found (karyorrhexis). Remnants of sinusoids are noted, devoid of red cells and Kupffer cells, and usually without endothelial nuclei or then only nuclei with signs of karyopyknosis. Remnants of blood vessels can contain an affine web of fibrin (Carroll 1963), but no fresh thrombi and no fibrinoid thrombi. In

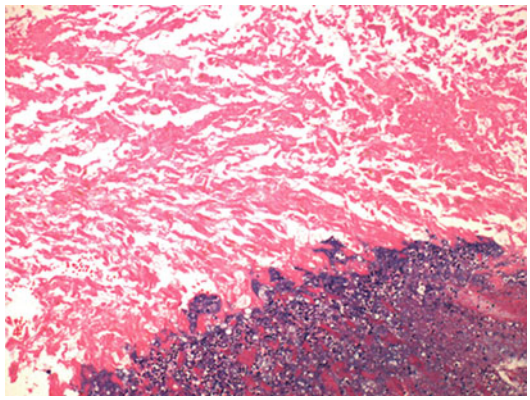

Fig. 5 In advanced hepatic infarction, compacted areas of coagulative necrosis, nuclear debris, chromatin fragments, and strongly basophilic "DNA powder" may ensue (hematoxylin and eosin stain)

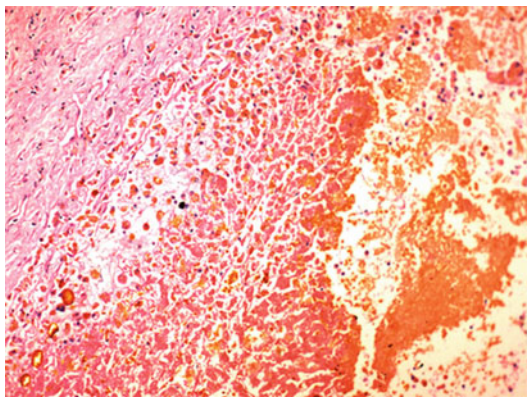

Fig. 6 Anemic infarction of the liver with bile duct destruction and leakage of bile (hematoxylin and eosin stain)

central parts, neutrophils are lacking, as they cannot be transported to this area excluded from circulation. In hepatic infarction caused by portal vein thrombosis, the borders between infarcted parenchymal areas and preserved parenchyma are usually located around central veins, corresponding to the periphery of the portal venous blood flow system (Saegusa et al. 1993). After few days, the centrally located area of coagulation necrosis is surrounded with a zone showing an infiltrate of neutrophils and few macrophages. Here, the hepatocytes appear shrunken, with a strongly eosinophilic cytoplasm and karyopyknotic nuclei. In this zone, sinusoids contain erythrocytes and Kupffer cells. After about 10 days, ductular proliferations containing progenitor cells start to appear. These proliferations originate from portal tracts and later invade the damaged lobular parenchyma. The tissue surrounding the infarct shows signs of hepatocyte regeneration, with an increase of hepatocyte mitotic figures. The necrotic process can also involve portal tracts, where interlobular bile ducts display severe ischemic damage or are absent, mainly in cases with hepatic artery occlusion (ischemic cholangiopathy). Involvement of larger bile ducts can result in bile leakage (Fig. 6) and the formation of bile duct cysts (Doppman et al. 1979). Infarcted liver parenchyma elicits an inflammatory reaction in the surrounding hepatic tissue, followed by granulation tissue, scarring, atrophy, and tissue contraction.

Differential Diagnosis

Anemic liver infarct clinically and radiologically resembles pyogenic liver abscess, in particular when infarcts undergo secondary suppurative changes, amebic liver abscess, and cancer. A difficult differential diagnostic situation may occur when ascending infection through the portal vein causes pylephlebitis followed by liver abscess (Brown et al. 2003). In patients with preeclampsia or eclampsia, hepatic hepatoma occurs (Sherbahn 1996) that may mimic infarction.

Hepatic Pseudo-infarct (Zahn's Infarct)

A rare differential diagnosis of liver infarct is the pseudo-infarct of the liver or Zahn's infarct (also termed infarct-like cyanotic atrophy). In contrast to true ischemic infarction, Zahn's infarct is a more or less, usually well-demarcated zone of hepatic congestion leading to parenchymal atrophy. Zahn's infarct is named after Friedrich Wilhelm Zahn (1845–1904), a German pathologist who studied under von Recklinghausen and received his M.D. in 1870 based on a work he performed in Bern, Switzerland, under Edwin

Klebs (review: Benaroyo 1991). After having served as a military physician during the war of 1870, he became professor of pathologic anatomy in Geneva, where he organized the new institute. Zahn is well known for contributions to the understanding and definition of thrombus. He was the first to put emphasis on the distinction of red and white thrombi and offered detailed descriptions of the process of thrombus corrugation (Zahn 1872, 1875, 1891: reviews: Bräunig and Doerr 1991, 1994). Zahn described the hepatic lesion named after him in 1897 under the term "atrophische rothe Leberinfarcte" (atrophic red hepatic infarcts), although he noted that there was no evidence of hepatocyte necrosis, but rather liver cell atrophy, "infarct" therefore being a misnomer. This has later led to the term "pseudo-infarction." As a pathogenic pathway, Zahn considered the combination of occlusion of a branch of the hepatic artery and an elevated pressure in the inferior vena cava and the hepatic veins caused by right-sided heart failure. However, part of these cases had portal vein thrombosis. Zahn's infarct has been observed in the setting of various vascular processes, including occlusive phlebitis in portal vein radicles (Matsumoto et al. 1992b), portal vein thrombosis (Funatsu et al. 1994), portal vein thromboembolism (Symmers 1951), and tumor-induced stenosis of intrahepatic vessels (Tsuuchi et al. 1990). Macroscopically, Zahn's pseudo-infarcts appear as more or less circumscribed dark red areas. Due to congestion and decrease of oxygenation, the lesions may turn bluish red, leading to the term "cyanotic atrophy." In contrast to true anemic infarction, Zahn's infarct mostly shows severe centrilobular congestion (passive hyperemia) associated with hepatocyte plate atrophy, but no coagulation necrosis, or then only a minor centrilobular hepatocyte necrosis. Sinusoids are usually dilated and packed with red cells, this change increasing in severity toward the center of lobules. In pericentral areas with incipient or established hepatocyte necrosis, neutrophils accumulate, reflecting that sinusoidal circulation is not interrupted, in contrast to true anemic infarction (Horrocks and Tapp 1966). The pathogenesis of centrilobular necrosis following congestion, including Zahn's pseudo-infarct, is complex and not yet fully understood (review: Shibayama et al. 1993).

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